COUNTWAY LIBRARY
HC 4ZSC M

6.G.432

THE

PRACTICE

OF

MEDICINE

A TEXT-BOOK FOR PRACTITIONERS AND STUDENTS
WITH SPECIAL REFERENCE TO DIAGNOSIS AND TREATMENT

BY

JAMES TYSON, M.D.

PROFESSOR OF CLINICAL MEDICINE IN THE UNIVERSITY OF PENNSYLVANIA AND PHYSICIAN TO THE HOSPITAL OF THE UNIVERSITY; PHYSICIAN TO THE PHILADELPHIA HOSPITAL; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA; MEMBER OF THE ASSOCIATION OF AMERICAN PHYSICIANS, ETC.

Illustrated

PHILADELPHIA
P. BLAKISTON, SON & CO.
1012 WALNUT STREET
1896

BOSTON MEDICAL
SEP 25 1903
LIBRARY.

COPYRIGHT, 1896, BY P. BLAKISTON, SON & CO.

WM. F. FELL & CO.,
ELECTROTYPERS AND PRINTERS,
1220-24 SANSOM STREET,
PHILADELPHIA.

TO

PROFESSOR J. M. DA COSTA, M.D., LL.D.,

PRESIDENT OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA
AND
EMERITUS PROFESSOR OF PRACTICE OF MEDICINE AND OF CLINICAL MEDICINE IN THE
JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA,

IN EVIDENCE OF MY APPRECIATION OF HIS POSITION AS AN
ACCOMPLISHED PHYSICIAN, EMINENT TEACHER,
AND TRUSTED CONSULTANT.

Digitized by the Internet Archive
in 2025

https://archive.org/details/practiceofmedici0000jame_t1r4

PREFACE.

I have no apology to make for preparing this book. I have long contemplated it, and have finished it after several years' labor. It has taken some time, because it represents almost purely personal work, which has been frequently interrupted. It does not pretend to be based on my personal practice only. In these days of specialized work this would be impossible, though with most of even the rare forms of disease in every section I have had some experience. To fill in the gaps of my own knowledge I have used that of others, but have always sought to make suitable acknowledgment to the proper source, and if this has not been done in any case, it has been a matter of oversight.

I had not, at the outset, expected to illustrate the work, but, as it progressed, a certain number of illustrations seemed necessary, not only to explain the text, but also, in a few instances, to render clearer the treatment described. Thus the number of charts and other drawings has grown to nearly a hundred, all of which, it is hoped, will be found useful. In expectation of the ultimate adoption of the metric system for the measuring of doses, these have been indicated throughout the book in the metric and English measures.

Acknowledgment is due to Dr. Joseph P. Walsh and Mr. M. A. Morin for suggestions after reading the text, to Dr. William Schleif for material assistance in Section XV, and to my son, Dr. T. Mellor Tyson, for assistance throughout the work and especially in preparing the index.

1506, Spruce St., *October 1, 1896.*

CONTENTS.

PRACTICE OF MEDICINE.

SECTION I.

INFECTIOUS DISEASES.

	PAGE
Typhoid Fever,	17
Mountain Fever,	46
Typhus Fever,	47
Relapsing Fever,	51
The Malarial Fevers,	55
Clinical Varieties,	63
Intermittent Fever,	63
Remittent Fever—Æstivo-Autumnal Fever,	67
Pernicious Malarial Fever—The Congestive Chill,	68
Malarial Cachexia, or Chronic Malaria,	70
Irregular Forms of Malarial Fever,	71
Malarial Hæmaturia and Hæmoglobinuria — Intermittent Hæmaturia and Hæmoglobinuria,	71
Cholera,	72
The Examination for Cholera Bacillus,	84
Yellow Fever,	86
Measles,	90
Rubella,	94
Scarlet Fever,	95
Diphtheria,	104
Small-Pox,	116
Vaccine Disease,	121
Chicken-Pox,	125
Whooping-Cough,	126
Mumps,	129
Influenza,	130
Cerebro-Spinal Fever,	134
Dengue,	141
Erysipelas,	142
Dysentery,	147
Catarrhal Dysentery,	147
Amœbic or Tropical Dysentery,	149
Diphtheritic Dysentery,	151
Chronic Dysentery,	153
Septicæmia and Pyæmia,	154
Septicæmia,	154
Pyæmia,	155
Hydrophobia,	158
Tetanus,	162
Anthrax,	166
Glanders and Farcy,	169
Foot and Mouth Disease,	171
The Bubonic Plague,	172
Syphilis,	174
Tuberculosis,	182
Acute General Tuberculosis,	189
Tuberculosis of Lymphatic Glands,	191
Leprosy,	193
Actinomycosis,	196
Infectious Diseases of Doubtful Nature,	198
Ephemeral Fever—Febricula,	198
Protracted Simple Continued Fever,	199
Weil's Disease,	200
Milk Sickness,	201
Malta Fever,	202
Miliary Fever,	203

SECTION II.

DISEASES OF THE DIGESTIVE SYSTEM.

I. Diseases of the Mouth,	205
The Coated Tongue,	205
Derangement Due to Dentition,	206
Stomatitis,	207
Simple Acute Catarrhal Stomatitis,	207
Aphthous Stomatitis,	208
I. Diseases of the Mouth:—	
Ulcerative Stomatitis,	208
Mycotic Stomatitis,	210
Treatment of Different Forms of Stomatitis,	210
Cancrum Oris,	211

PAGE

I. Diseases of the Mouth:—
Glossitis, ... 212
Glossitis Dessicans, ... 213
Lingual Psoriasis, ... 213
Leucoplacia, ... 213
Mucous Patches, ... 214
II. Diseases of the Salivary Glands, . 214
Functional Derangements, ... 214
Inflammation of the Salivary Glands, 214
Angina Ludovici, ... 215
III. Diseases of the Tonsils and Pharynx, 216
Quinsy, ... 216
Follicular Tonsillitis, ... 217
Aphthous Tonsillitis, ... 218
Chronic Tonsillitis and Hypertrophy of the Adenoid Tissue of the Pharynx, ... 219
Simple Circulatory Derangements of the Pharynx, ... 221
Acute Catarrhal Pharyngitis, . . . 222
Chronic Catarrhal Pharyngitis, . . 223
Ulceration of the Pharynx, ... 223
Phlegmonous Pharyngitis, ... 224
Post-Pharyngeal Abscess, ... 224
Diseases of the Œsophagus, ... 225
Exploration of the Œsophagus with the Bougie, ... 225
Œsophagitis, ... 225
Spasm of the Œsophagus, ... 226
Cancer of the Œsophagus, ... 227
Stricture of the Œsophagus, . . . 227
Dilatation of the Œsophagus, . . . 228
Diseases of the Stomach, ... 230
Diagnostic Technique, ... 230
External Examination, ... 230
Internal Examination or Chemical Examination of Gastric Contents, 234
Acute Catarrhal Gastritis, ... 243
Chronic Catarrhal Gastritis, ... 245
Phlegmonous or Suppurative Gastritis, ... 250
Traumatic and Toxic Gastritis, . . 251
Nervous Dyspepsia, ... 252
Gastralgia, ... 254
Hyperchlorhydria, ... 256
Anorexia Nervosa, ... 259
Nervous Vomiting, ... 259
Gastric and Duodenal Ulcer, . . . 261
Cancer of the Stomach, ... 267
Dilatation of the Stomach, ... 273
Diseases of the Intestines, ... 276
Simple Acute Catarrhal Enteritis, . 276
Chronic Catarrhal Enteritis, . . . 280
Diarrhœas of Children, ... 283
Acute Dyspeptic Diarrhœa, . . . 283
Cholera Infantum, ... 285
Acute Entero-colitis, ... 287
The Cœliac Affection in Children, . 288
Cholera Morbus, ... 288
Pseudo-Membranous Enteritis, . . 290
Phlegmonous Enteritis, ... 290
Hemorrhagic Infarct of the Bowel, 291
Ulceration of the Bowel, ... 291
Tubercular Ulcer, ... 291
Syphilitic Ulcer, ... 292
Embolic Ulcer, ... 292

PAGE

Diseases of the Intestines:—
Appendicitis, ... 293
Recurring and Relapsing Appendicitis—Chronic Appendicitis, . 304
Intestinal Obstruction, ... 308
I. Internal Strangulation. . 308
II. Intussusception. Invagination, ... 309
III. Twists and Knots. Volvulus, ... 310
IV. Obstruction by Abnormal Contents or Foreign Bodies, ... 310
V. Strictures and Morbid Growths, ... 311
VI. Fecal Obstruction, . . . 311
Constipation, ... 318
Dilatation of the Colon, ... 322
Nervous Affections of the Bowel, . 322
I. Derangements of Motion, 322
II. Derangements of Sensibility, ... 323
III. Secretion Neuroses, . . 325
Carcinoma of the Bowel, ... 326
Diseases of the Liver, ... 330
Abnormalities in the Shape and Position of the Liver, ... 330
Diseases of the Bile Passages and Gall Bladder, ... 331
Jaundice, or Icterus, ... 331
Icterus Neonatorum, ... 334
Simple Catarrhal Jaundice, ... 335
Impacted Gall-stone, ... 336
Chronic Impacted Gall-stone, . . 339
Other Affections of the Bile Ducts, 342
Carcinoma, ... 342
Stenosis, ... 342
Parasites, ... 342
Cicatricial Contraction, . . . 342
Cancer of the Gall Bladder, . . . 343
Hyperæmia of the Liver, ... 343
Passive Hyperæmia — Red Atrophy, ... 343
Active Hyperæmia, ... 345
Diseases of the Blood-vessels of the Liver, 345
Pylethrombosis, ... 345
Pylephlebitis, ... 346
Changes in the Hepatic Artery and Vein, ... 346
Fatty Liver, ... 347
Fatty Infiltration, ... 347
Fatty Metamorphosis, ... 348
The Amyloid Liver, ... 348
Cirrhosis of the Liver, ... 350
Suppurative Hepatitis—Abscess of the Liver, ... 356
Acute Yellow Atrophy of the Liver, ... 359
Morbid Growths of the Liver, . . 361
Carcinoma of the Liver, ... 362
Sarcoma, ... 363
Syphilis of the Liver, ... 365
Parasites of the Liver, ... 367
Echinococcus Disease, or Hydatid Cyst of the Liver, ... 367
Other Parasites of the Liver, . . 370

PAGE

Diseases of the Blood-vessels of the Liver:—
Perihepatitis, ... 370
Glissonian Cirrhosis, ... 371
Diseases of the Pancreas, ... 372
Acute Pancreatitis, ... 372
Hemorrhagic Pancreatitis, ... 372
Gangrenous Pancreatitis, ... 373
Suppurative Pancreatitis, ... 373
Chronic Pancreatitis, ... 374
Cysts of the Pancreas, ... 374
Cancer of the Pancreas, ... 374
Sarcoma of the Pancreas, ... 375
Diseases of the Spleen, ... 375
Splenitis, ... 375
Perisplenitis, ... 375

PAGE

Diseases of the Spleen—
Abscess of the Spleen, ... 375
Rupture of the Spleen, ... 375
The Amyloid Spleen, ... 375
Atrophy of the Spleen, ... 376
Hemorrhagic Infarct of the Spleen, 376
Neoplasms of the Spleen, ... 376
Echinococcus of the Spleen, . . . 376
Wandering Spleen, ... 376
Diseases of the Peritoneum, ... 377
Acute Peritonitis, ... 377
Chronic Peritonitis, ... 382
Tuberculosis of the Peritoneum, . . 383
Cancer of the Peritoneum, ... 384
Ascites, ... 385

SECTION III.

DISEASES OF THE RESPIRATORY SYSTEM.

Diseases of the Nose, ... 389
Rhinitis, ... 390
Chronic Nasal Catarrh, ... 391
Hay Fever, ... 393
Diseases of the Larynx, ... 396
Examination of the Larynx, . . . 396
Acute Catarrhal Laryngitis, . . . 397
Spasmodic or Catarrhal or False Croup, ... 398
Simple Chronic Catarrhal Laryngitis, ... 400
Tubercular Laryngitis, ... 403
Syphilitic Laryngitis, ... 405
Œdema of the Glottis, ... 405
Paralysis of the Laryngeal Muscles, 406
Diseases of the Trachea and Bronchial Tubes, ... 410
Acute Tracheo-bronchitis, ... 410
Chronic Bronchitis, ... 412
Bronchiectasis, or Bronchial Dilatation, ... 417
Bronchial Asthma, ... 419
Plastic or Fibrinous Bronchitis, . . 423
Diseases of the Lungs, ... 425
Emphysema, ... 425

Diseases of the Lungs:—
Vesicular Emphysema—Pseudo-Hypertrophic Emphysema, ... 425
Croupous Pneumonia, ... 431
Chronic Interstitial Pneumonia, . . 446
Broncho-Pneumonia, ... 448
Embolic Pneumonia, ... 453
Embolic Non-septic Pneumonia, . . 453
Embolic Septic Pneumonia, ... 455
Pulmonary Tuberculosis, ... 456
Chronic Ulcerative Phthisis, . 458
Acute or Pneumonic Phthisis, . 470
Fibroid Phthisis, ... 471
Treatment of Tubercular Phthisis, 472
Tumors of the Lung, ... 486
Diseases of the Pleura, ... 488
Acute Pleurisy, ... 488
Varieties of Acute Pleurisy, ... 492
Chronic Pleurisy, ... 496
Hydrothorax and Hæmatothorax, . 497
Pneumothorax, ... 497
Morbid Growths of the Pleura, . . 499
Mediastinal Disease, ... 500
Mediastinal Tumors, ... 502
Mediastinal Abscess, ... 505

SECTION IV.

DISEASES OF THE HEART AND BLOOD-VESSELS.

Cardiac Asthma, ... 507
Diseases of the Pericardium, ... 508
Pericarditis, ... 508
Other Pericardial Affections, . . . 513
Hydropericardium, ... 513
Hæmopericardium, ... 513
Pneumopericardium, ... 514
Diseases of the Endocardium, ... 514
Endocarditis, ... 514
The Mild or Simple Form of Acute Endocarditis, ... 515
The Severe or Malignant Form of Acute Endocarditis, ... 517

Diseases of the Endocardium:—
Chronic Valvular Defects, ... 521
Mitral Insufficiency, ... 523
Mitral Stenosis, ... 525
Mitral Insufficiency and Stenosis, . 528
Aortic Insufficiency or Incompetency, 528
Aortic Stenosis, ... 532
Aortic Stenosis and Incompetency, . 533
Tricuspid Incompetency, ... 534
Tricuspid Stenosis, ... 535
Pulmonary Stenosis, ... 535
Pulmonary Incompetency, ... 536
Congenital Defects, ... 536

PAGE

Diseases of the Endocardium:—
Relative Frequency of Valvular Defects, ... 537
Associated or Combined Valvular Lesions, ... 538
Diseases of the Myocardium, ... 545
Hypertrophy and Dilatation—Atrophy, ... 545
Hypertrophy of the Heart, ... 546
Dilatation of the Heart, ... 549
Atrophy of the Heart, ... 552
Degeneration of the Cardiac Muscle, 553
Parenchymatous or Albuminoid Degeneration, ... 553
Fatty Degeneration, or Fatty Metamorphosis, ... 553
Fatty Infiltration, or Fatty Overgrowth, ... 554
Amyloid Degeneration, . . . 554

PAGE

Diseases of the Myocardium:—
Calcareous Degeneration, . . 555
Brown Atrophy, ... 555
Yellow Atrophy, ... 555
Myocarditis, ... 555
Fibro-Myocarditis, ... 555
Acute Suppurative Myocarditis, . . 557
Rupture of the Heart, ... 558
Aneurism of the Heart, ... 558
Neuroses of the Heart, ... 559
Nervous Palpitation, ... 559
Tachycardia and Brachycardia, . . 560
Irregular Pulse, ... 561
Angina Pectoris, or Stenocardia, . 565
Diseases of the Blood-vessels, ... 567
Arterio-Sclerosis, ... 567
Aneurism, ... 570
Aneurism of the Thoracic Aorta, . 572

SECTION V.

DISEASES OF THE BLOOD AND BLOOD-MAKING ORGANS.

The Anæmias, ... 583
The Primary or Essential Anæmias, 584
I. Chlorosis, ... 584
II. Progressive Pernicious Anæmia, ... 588
III. Leucæmia, ... 594
IV. Lymphatic Anæmia — Pseudoleucæmia — Hodgkin's Disease, ... 604
V. Splenic Anæmia, or Splenic Pseudoleucæmia, ... 607
Secondary or Symptomatic Anæmia, 609

SECTION VI.

DISEASES OF THE THYROID GLAND.

Goitre, ... 612
Simple Goitre, or Struma, ... 612
Exophthalmic Goitre, ... 614
Myxœdema, ... 619
Cretinism, ... 622
Congenital Cretinism, ... 622
Sporadic and Endemic Cretinism, . 623
Tumors of the Thyroid, ... 624

SECTION VII.

DISEASES OF THE URINARY ORGANS.

General Remarks on Albuminuria, . . . 625
Extra Renal Albuminuria, ... 625
Physiological or Functional Albuminuria, 627
Diseases of the Kidney, ... 629
Derangements of Circulation, . . . 629
Active Congestion, ... 629
Passive Congestion or Cyanotic Induration, ... 629
Acute Parenchymatous Nephritis, . 631
Chronic Parenchymatous Nephritis, 644
Interstitial Nephritis, ... 654
Amyloid or Lardaceous Kidney, 664
Suppurative Interstitial Nephritis and Pyelo-Nephritis, ... 669
Paranephritis or Perinephric Abscess, ... 675
Diseases of the Kidney:—
Tuberculosis of the Kidney, . . . 676
Nephro-lithiasis — Stone in the Kidney, ... 677
Tumors of the Kidney, ... 682
Cysts of the Kidney, ... 684
Anomalies of Form and Position of the Kidney, ... 687
The Movable or Floating Kidney, 688
Idiopathic Hæmaturia, ... 690
Hæmoglobinuria, ... 691
Toxic Hæmoglobinuria, . . . 691
Paroxysmal Hæmoglobinuria, . 692
Chyluria, ... 693
The Relation of Heart Disease to Kidney Disease, ... 694

PAGE

Diseases of the Bladder, ... 700
Cystitis, ... 700
Stone in the Bladder, ... 706
Neuroses of the Bladder, ... 707
Diseases of the Bladder:—
Muscular Spasm of the Bladder, . . 708
Hemorrhoidal Veins of the Bladder, 710
Morbid Growths of the Bladder, . . 711

SECTION VIII.

DISEASES OF THE SUPRARENAL CAPSULE.

Addison's Disease, ... 712

SECTION IX.

CONSTITUTIONAL DISEASES.

Rheumatism, ... 715
Rheumatic Fever, ... 715
Subacute Rheumatism, ... 720
Muscular Rheumatism, ... 723
Chronic Rheumatism, ... 725
Joint-affections Simulating Rheumatism, 727
Gonorrhœal Arthritis, ... 727
Arthritis Deformans, ... 729
I. Multiple Arthritis Deformans, . . 731
II. Partial or Monarthritic form, . . 732
Gout, ... 734
Lithæmia, ... 748
Diabetes Mellitus, ... 750
Diabetes Insipidus, ... 771
Obesity, ... 776
Rickets, ... 779
Osteomalacia, ... 783
Purpura, ... 785
Symptomatic Purpura, ... 785
Scorbutic Purpura, ... 786
Infantile Scurvy, ... 788
Arthritic Purpura, ... 789
Purpura Hemorrhagica, ... 789
Hemorrhagic Diseases of the Newborn, ... 790
Hæmophilia, ... 792

SECTION X.

DISEASES OF THE NERVOUS SYSTEM.

General Introduction, ... 794
Histology of the Nervous System, . 794
General Symptomatology (Investigation of a Case of Nervous Disease), ... 797
I. Phenomena of Motion, . 797
II. Sensory Phenomena, . . 808
III. Vaso-motor and Trophic Phenomena, ... 812
IV. Mental Phenomena, . . 813
V. Alterations in Hearing and Vision, ... 814
VI. Alterations in Breathing and Pulse, ... 814
Affections of the Peripheral Nerves, . . 815
Neuritis, ... 815
Localized Neuritis. 815
Special Varieties of Neuritis, . . . 818
Brachial Neuritis, ... 818
Sciatica, ... 819
Multiple Neuritis, ... 821
Endemic Neuritis, ... 828
Neuromata, ... 830
Affections of the Spinal Cord, ... 831
Localization of the Functions of the Segments of the Spinal Cord, . . 835
Affections of the Membranes of the Cord, ... 842
Affections of the Membranes of the Cord:—
Spinal Pachymeningitis, ... 842
Spinal Leptomeningitis, ... 843
Acute Leptomeningitis, . . . 843
Chronic Leptomeningitis, . . 844
Hemorrhage into the Spinal Membranes, ... 845
Affections of the Substance of the Cord, ... 846
Unilateral Lesions of the Spinal Cord, 847
Secondary Systemic Degenerations of the Spinal Cord, ... 848
Acute Affections of the Spinal Cord, 851
Disturbances of the Circulation of the Spinal Cord, ... 851
Hemorrhage into the Substance of the Cord, ... 851
Caisson Disease, ... 853
Myelitis (Acute and Chronic), . . 854
Myelitis of the Anterior Horns, . . 861
Acute Poliomyelitis in Adults, . . 864
Subacute and Chronic Poliomyelitis, 864
Acute Ascending Spinal Paralysis, . 864
Chronic Affections of the Spinal Cord, 866
Spastic Spinal Paralysis, ... 866
Locomotor Ataxia, ... 869
Hereditary Ataxia, ... 879
Syringo-Myelia, ... 881

PAGE

Affections of the Membranes of the Cord:—
Morvan's Disease, ... 883
Compression of the Spinal Cord, . 883
Lesions of the Cauda Equina and Conus Medullaris, ... 886
Spina Bifida, ... 887
Tumors of the Spinal Cord and Membranes, ... 888
Progressive Bulbar Palsy, ... 890
Acute Bulbar Palsy, ... 893
Amyotrophic Lateral Sclerosis, . . 894
Progressive Muscular Atrophy, . . 896
Diseases of the Brain, ... 902
Localization of Cerebral Disease, . 902
I. The Motor Areas of the Cortex, ... 903
II. Sensory Areas of the Cortex and Sensory Paths, 907
Cortical Areas Covering Speech, . 909
The Various Forms of Aphasia and their Anatomical Lesions, . . . 909
Apraxia—The Physical Basis of Thought, ... 910
Aphasia, or Loss of the Faculty of Speech, ... 913
Derangements of Speech of Irritative Origin, ... 918
Cortical Areas Whose Function is Unknown and Uncertain, . . . 919
Tracts Within the Brain—Centrum Ovale, Internal Capsule, Central Ganglia, Corpora Quadrigemina, 920
Diseases of the Cranial Nerves, ... 924
Olfactory Nerve, ... 924
Optic Nerve and Tract, ... 925
(1) Affections of the Retina, . 925
(2) Affections of the Optic Nerve, ... 927
(3) Lesions of the Chiasm and Tract, ... 930
(4) Lesions of the Tract and Centres, ... 932
Symptoms of Lesions of the Optic Nerve, Chiasm, Tract, and Optic Cortex, ... 933
Lesions of the Motor Nerves of the Eyeball, ... 936
Third Nerve, ... 936
Fourth Nerve, ... 939
Sixth Nerve, ... 939
Phenomena in general of Paralysis of Motor Nerves of the Eye, ... 940
Ophthalmoplegia, ... 940
Treatment of Ocular Palsies, . 942
Lesions of the 5th Nerve (Trigeminus), ... 942
Lesions of the Facial Nerve, or Seventh Pair, ... 944
Paralysis of the Facial, . . . 944
Facial Spasm, ... 950
Lesions of the Auditory or 8th Nerve, ... 951
(1) Loss of Function; Nervous Deafness, ... 952
(2) Auditory Hyperæsthesia, . 954

PAGE

Diseases of the Cranial Nerves:—
(3) Irritation of the Auditory Nerve—Tinnitus Aurium, . 954
(4) Disturbance of Equilibrium Associated with Defect of Hearing, Labyrinthine Vertigo, Ménière's Disease, . . 955
Lesions of the 9th or Glosso-Pharyngeal Nerve, ... 957
Lesions of the Pneumogastric or Vagus Nerve, the Tenth Pair, . . 958
Lesions Involving the Nucleus and Trunk of the Pneumogastric and Branches, . . . 958
Lesions of the Pharyngeal Branches, ... 958
Lesions of the Laryngeal Branches, ... 959
Spasm of the Larynx, ... 962
Lesions of the Cardiac Branches, 963
Lesions of Gastric and Œsophageal Branches, ... 963
Lesions of Pulmonary Branches, 964
Lesions of the Eleventh Pair or Spinal Accessory Nerve, ... 965
Symptoms of Paralysis of the external branch of the Spinal Accessory, ... 965
Symptoms of Accessory Spasm, 966
(1) Congenital Torticollis, or Fixed Wry Neck, ... 966
(2) Spasmodic Wry Neck, . . 967
Lesions of the Twelfth Pair or Hypoglossal Nerve, ... 969
Diseases of the Spinal Nerves, ... 971
Cervical Plexus, ... 971
Affections of the Phrenic Nerve, . 971
Lesions of the Brachial Plexus, . . 972
Of the Combined Plexus, . . 972
Lesions of Individual Nerves, . . 972
Of the long Thoracic or Posterior Thoracic—Serratus Palsy, . 972
Nerves of the Arm, ... 972
Lumbar and Sacral Plexuses, . . . 975
Diseases of the Membranes of the Brain, 977
Pachymeningitis, ... 977
External Pachymeningitis, . . 977
Internal Pachymeningitis, . . 977
Purulent and Pseudomembranous Pachymeningitis, . . 977
Hemorrhagic Pachymeningitis, 977
Leptomeningitis, ... 979
Acute Leptomeningitis, . . . 979
Chronic Leptomeningitis, . . 983
Affections of the Blood-vessels of the Brain, ... 984
Hyperæmia, ... 984
Anæmia of the Brain, ... 985
Œdema of the Brain, ... 986
Apoplexy, ... 987
I. Cerebral Hemorrhage, . . 987
II. Embolism and Thrombosis of the Cerebral Vessels, 996
Morbid changes due to Thrombosis and Embolism, ... 997
Intracranial Aneurisms, ... 1001

PAGE

Affections of the Blood-vessels of the Brain:—
Thrombosis of the Cerebral Sinuses and Veins, ... 1002
The Cerebral Palsies of Children, . 1003
Infantile Hemiplegia, ... 1003
Bilateral Spastic Hemiplegia, . 1006
Spastic Paraplegia, ... 1008
Sclerosis of the Brain, ... 1009
Multiple Sclerosis of the Brain and Spinal Cord, ... 1009
Dementia Paralytica, ... 1011
Tumors of the Brain, ... 1015
Suppurative Encephalitis, ... 1022
Encephalitis without Abscess, . 1025
Chronic Hydrocephalus, ... 1025
External Hydrocephalus, . . 1025
Internal Hydrocephalus, . . . 1025
Congenital Hydrocephalus, . . 1025
Acquired Hydrocephalus, . . . 1026
General and Functional Diseases, . . . 1028
Acute Delirium, ... 1028
Paralysis Agitans, ... 1029
Other Forms of Tremor, ... 1032
Acute Chorea, ... 1032
Choreiform Affections, ... 1038
I. Simple Tic, ... 1038
II. Tic with Explosive Utterances, Caprolalia, Echolalia, etc., ... 1039
III. Complex Coördinated Tic, . 1040
IV. Spasms of the Muscles of Respiration and Deglutition, ... 1040
V. Chronic Progressive Chorea, 1041

PAGE

General and Functional Diseases:—
VI. Chorea Major, ... 1042
VII. Pre- and Post-Hemiplegic Chorea, ... 1043
Epilepsy, ... 1044
Reflex Convulsions of Children, . . 1052
Migraine, ... 1054
Neuralgia, ... 1057
Varieties Depending Upon Nerves Involved, ... 1058
Occupation Neuroses, ... 1063
Writer's Cramp, ... 1063
Athetosis, ... 1067
Tetany, ... 1068
Hysteria, ... 1070
Neurasthenia, ... 1078
Traumatic Neuroses, ... 1082
Other Forms of Functional Paralysis, 1083
Abasia-Astasia, ... 1083
Periodical Paralysis, ... 1084
Vaso-Motor and Trophic Derangements, ... 1085
Acute Angeioneurotic Œdema, 1086
Raynaud's Disease, ... 1086
Progressive Facial Hemiatrophy, 1089
Acromegaly, ... 1089
Scleroderma, ... 1091
Morphœa, ... 1092
Ainhum, ... 1093
Relation of Infectious Processes to Disease of the Nervous System, . 1093
Summary of Facts and Conclusions, ... 1094
Syphilis of the Nervous System, . . 1097

SECTION XI.

DISEASES OF THE MUSCULAR SYSTEM.

Myositis, ... 1103
Rheumatic Myositis (Acute and Chronic), ... 1103
Infectious Myositis, ... 1103
Progressive Ossifying Myositis, . . 1103
Idiopathic Muscular Atrophies. Primary Myopathic Forms of Muscular Atrophy, ... 1104
I. Pseudo-Hypertrophy of Muscles, ... 1104
II. Erb's Form of Juvenile Hereditary Atrophy, ... 1105
III. The Facio-Scapulo-Humeral Type of Juvenile Palsy, . . 1106
IV. The Peroneal Type of Progressive Atrophy, ... 1106
Myotonia Congenita (Thomsen's Disease), ... 1107

SECTION XII.

THE INTOXICATIONS.

Alcoholism, ... 1109
Acute Alcoholism, ... 1109
Chronic Alcoholism, ... 1110
Delirium Tremens, or Mania a Potu, 1112
The Morphine Habit—Morphinism, . . 1115
Chloralism, ... 1117
Cocainism, ... 1118
Lead Poisoning, ... 1119
Arsenical Poisoning, ... 1124
Ptomaïne Poisoning, ... 1125
Poisoning by Milk and its Products, . . 1126
Grain Poisoning, ... 1128
1. Ergotism, ... 1128
2. Pellagra, ... 1129
3. Lathyrism, or Lupinosis, . . . 1129

SECTION XIII.

EFFECTS OF EXPOSURE TO HIGH THOUGH BEARABLE TEMPERATURE.

PAGE

Heat Exhaustion, ... 1130

Thermic Fever—Sunstroke, Coup de Soleil, ... 1131

SECTION XIV.

ANIMAL PARASITES AND THE CONDITIONS CAUSED BY THEM.

I. Protozoa, ... 1135
II. Platyhelminthes, or Flat-Worms, . 1136
(A) Trematodes, or Flukes, . 1136
(B) Cestodes, or Tapeworms, . 1138
III. Nematodes, or Thread Worms, . . 1146
(A) The Ascarides, ... 1146
(B) Trichiniasis, ... 1148
(C) Anchylostomiasis, ... 1150
(D) Filariasis, ... 1151
(E) Other Nematode Worms, 1154
IV. Acanthocephali—Thorn-head Worms, ... 1155
V. Arthropoda, ... 1155
(A) Arachnoidea, ... 1155
(B) Insecta, ... 1157

SECTION XV.

Summary of Symptoms Following Over-doses of Poisons, and the Treatment of their Effects. To which is Added a Table of Minimum Dose which has Caused Death and Maximum Dose Followed by Recovery, ... 1160

APPENDIX.

Tables for the Conversion of the English Into Metric System, and the Reverse, ... 1172

INDEX, ... 1175

CHARTS AND ILLUSTRATIONS.

FIG.		PAGE
1.	Temperature Chart of a Typical Case of Typhoid Fever Uninfluenced by Treatment,	25
2.	Chart Showing Drop in Temperature Incident to Intestinal Hemorrhage,	27
3.	Chart Showing Anæmia of Typhoid Fever,	30
4.	Chart Contrasting the Drop in Temperature after the Bath Early and Later in the Disease,	38
5.	Burr's Portable Bath-Tub,	40
6.	Temperature Chart of Relapsing Fever, Showing Relapses,	54
7.	Plate Illustrating Different Forms of the Malarial Organism with their Stages of Development,	57
8.	Temperature Chart in Intermittent Fever, Showing the Paroxysms and Intermission,	64
9.	Temperature Chart in Intermittent Fever, Showing the Paroxysms and Intermission,	65
10.	Fountain Syringe for Hypodermoclysis,	82
11.	Temperature Chart of Measles,	91
12.	Temperature Chart of Scarlet Fever,	98
13.	Temperature Chart of Small-Pox,	117
14.	Syphilitic Teeth,	179
15.	Diagram Showing Eruption of Milk Teeth,	206
16.	Arrangement for Auto-Lavage,	250
17.	Temperature Chart of Appendicitis Showing Temperature Maintained by Abscess after Partial Decline,	302
18.	Chart of Appendicitis Showing Misleading Fall to Normal, Incident to Perforation,	303
19.	Giant Congenital Dilatation of Human Colon,	321
20.	Comparative Enlargements of the Liver, Corresponding to the Different Morbid Growths,	363
21.	Tænia Echinococcus,	367
22.	So-called "Ovarian Cells,"	387
23.	Hard-Rubber Nasal Speculum,	389
24.	Spring Nasal Speculum,	389
25.	Technique of Rhinoscopic Examination,	390
26.	Coarse Spray Apparatus,	393
27.	Technique of Laryngoscopic Examination,	396
28.	Natural Size of Image of the Vocal Apparatus,	397
29.	Cadaveric Position of the Left Vocal Cord,	407
30.	Complete Both-sided Abductor Paralysis of the Posterior Crico-Arytenoid Muscles,	407
31.	Paralysis of the Internal Thyro-Arytenoid Muscles,	408
32.	Paralysis of the Transverse and Oblique Inter-Arytenoid Muscles,	408
33.	Bilateral Paralysis of the Thyro-Arytenoids Combined with Paresis of the Arytenoid,	408
34.	Showing Crisis in Pneumonia,	436
35.	Showing Drop in Temperature in a Case of Pneumonia Succeeding Application of the Cold Wet-Jacket,	445
36.	Lobule of Lung Showing Acini and Alveolar Passages,	456
37.	Temperature Chart of a Case of Tubercular Consumption Without Fever,	466
38.	Temperature Chart Showing Extreme Range of Temperature in Tubercular Phthisis,	467
39.	Pasteboard Spit-Cup,	483
40.	Section through Frozen Thorax at 2d Interspace in Front, looking from above downward,	500
41.	Section through Frozen Thorax at 2d Interspace in Front, looking from below upward,	501
42.	Temperature Chart, Mycotic Endocarditis,	519
43.	Tracing of Pulse of Mitral Insufficiency,	525
44.	Tracing of Pulse of Mitral Stenosis,	527
45.	Tracing of Pulse of Aortic Regurgitation,	529
46.	Pulse Tracing of Aortic Stenosis,	533

FIG. PAGE

47. Normal Pulse Tracing, . . . 563
48. Tracing of Pulse of High Arterial Tension, . . . 563
49. Sphygmogram of an Atheromatous Vessel, . . . 570
50. Aneurism of the Aorta Showing Sites of Election, . . . 572
51. Chart Showing Blood in Chlorosis, . . . 586
52. Chart Showing Blood in Pernicious Anæmia, . . . 590
53. Colored Plate Showing the Different Forms of Colorless Corpuscles in the Blood of Leucæmia, . . . 597
54. The Relative Sizes of the Various Cells Compared with the Red Corpuscle, . . . 600
55. Chart Showing the Blood in Simple Anæmia, . . . 609
56. "Seal-Fin" Deflexion of the Hand in a Case of Protracted Arthritis Deformans, . . . 731
57. Rickety Chest, . . . 780
58. Diagram of an Element of the Motor Path, . . . 795
59. Illustrating Crossed Paralysis, . . . 798
60. Diagram Showing Probable Plan of the Centre for Micturition, . . . 801
61. Diagram Showing Relation of Vertebral Spines to Their Bodies and to the Nerve-Roots, . . . 832
62. Section of Spinal Cord in the Cervical Region, . . . 833
63. Diagram Showing Relative Size and Shape of the Cord and Grey Matter at Different Levels, . . . 834
64. Diagram of Sensory Skin Areas, Corresponding to the Different Spinal Segments—Anterior Surface, . . . 837
65. Diagram of Sensory Skin Areas, Corresponding to the Different Spinal Segments—Posterior Surface, . . . 839
66. Diagram of Lesion, Showing Brown-Séquard's Paralysis, . . . 847
67. Schema Showing Chief Symptoms in Left Unilateral Lesion of the Dorsal Cord, . . . 847
68. Secondary Descending Degeneration of the Pyramidal Tracts in a Primary Lesion of the Left Half of the Cerebrum, . . . 849
69. Secondary Ascending and Descending Degeneration in a Transverse Section of the Upper Dorsal Region, . . . 850
70. Diagram of Descending Degeneration of a Pyramid, with a Lesion in the Left Internal Capsule, . . . 850
71. Transverse Section through the Lumbar Region in Locomotor Ataxia, . . . 871
72. Transverse Section through the Cervical Region in Locomotor Ataxia, . . . 871
73. Transverse Section through the Posterior Columns of the Cord in Beginning Locomotor Ataxia, . . . 872
74. Clawed Hand, . . . 898
75. Lateral Aspect of the Brain, . . . 903
76. Aspect of the Median Surface of the Cerebrum, as it appears when the Two Hemispheres are Separated, . . . 904
77. Lateral Aspect of the Brain, . . . 904
78. The Motor Tract, . . . 906
79. Primitive Speech Apparatus of the Child in Mechanical Repetition of Words, . . . 910
80. Wernicke's Schema Showing the Association of the Various Partial Conceptions to Form the Whole Conception or Word-image of an Object, . . . 911
81. Simplification of the Schema of Voluntary Speech, . . . 912
82. Diagram of Seats of the Lesions of Word-deafness, Word-blindness, Motor Aphasia, and Agraphia, . . . 917
83. Simplification of Wernicke's Schema of Voluntary Speech, . . . 917
84. Transverse Section through the Crura Cerebri in Secondary Degeneration of the Right Pyramidal Tract, . . . 921
85. Commencing Optic Neuritis from a Case of Caries of the Sphenoid Bone with Secondary Meningitis, . . . 928
86. Diagram Showing Course of Optic-Nerve Fibres, . . . 931
87. Schema for Central Innervation of the Facial Nerve, . . . 945
88. Schematic Representation of the Trunk of the Facial Nerve from the Base of the Skull to the Pes Anserinus, . . . 948
89. Focal Symptoms of Brain Tumor, . . . 1018
90. Temperature Chart from a Case of Sunstroke Treated by Ice-Water Baths and Frictions. Recovery, . . . 1132

BOSTON MEDICAL
SEP 25 1903
LIBRARY.

PRACTICE OF MEDICINE.

SECTION I.

INFECTIOUS DISEASES.

TYPHOID FEVER.

SYNONYMS.—*Typhus abdominalis; Enteric Fever; Pythogenic Fever; Gastro-enteric Fever; Nervous Fever; Autumnal Fever; Slow Nervous Fever.*

Definition.—Typhoid fever is an acute infectious fever due to the implantation and proliferation of the typhoid bacillus or bacillus of Eberth. It is especially characterized anatomically by hyperplastic and ulcerative lesions of the lymph follicles of the intestine, of the mesenteric glands, and by enlargement of the spleen.

Historical.—The disease is probably coeval with civilization, and is easily recognizable in the descriptions of Hippocrates (B. C. 460–357) and Galen (A. D. 130–200) and in more modern times of Adrianus Spigelius (1624), Thomas Willis (1659), F. Hoffmann (1699), Thomas Sydenham (1685), and others in the seventeenth century, and in the next, especially of E. Gilchrist (1734), John Huxham (1739), J. C. Riedel (1748), and R. Manningham (1746). Doubtless Huxham's "slow nervous fever" described in his "Essay on Fevers" was the typhoid of the present day, and his "putrid malignant" the rarer typhus of to-day. It was, however, regarded by him rather as a variety of continued fever than as a distinct and separate fever, and it was not until 1813 that Pierre Bretonneau of Tours described it under the name *dothiénentérite*, and Petit and Serres as *fièvre entéro-mésentérique*. It was, however, the writings and teachings of the great French physician, Louis, which did most to disseminate a knowledge of the true nature of typhoid fever—to which he gave the name it bears. His great work was published in 1829,* while his pupils came from every quarter. Included among them was a coterie of brilliant young Americans, among whom were William W. Gerhard and C. W. Pennock of Philadelphia and James Jackson, Jr., of Boston. The first

* Louis, P. C. A., *Recherch. anatom., patholog. et thérapeutiques sur la maladie connue sous les noms gastro-entérite, fièvre putride*, etc. Paris, 1829.

after his return to America had the opportunity, in conjunction with Pennock, of studying the disease in the wards of the Philadelphia Hospital in the spring and summer of 1836, and of contrasting it with typhus fever, of which there was an epidemic then prevalent in Philadelphia. These two observers were the first to point out the distinctive features of the two diseases in the American Journal of the Medical Sciences in 1837. Their publications were followed in 1838 by a paper by James Jackson, Sr., entitled "Report on Typhoid Fever," and another by Enoch Hale "On the Typhoid Fever of New England," which probably had their impulse in the knowledge furnished by the younger Jackson to his father on his return from Paris. Thus it came to pass that Elisha Bartlett's work on the "Diagnosis and Treatment of Typhus and Typhoid Fever," an American text-book published in 1842, contained the first separate description of the diseases. For up to 1838 only typhoid fever was known in Paris. At this time Alfred Stillé, who had been the house physician of Gerhard and Pennock in the Philadelphia Hospital and had learned there the distinctive features of typhus and typhoid, went to Paris and in 1838 read a paper before the *Société medicale d'Observation*, pointing out the differences between the two diseases. George C. Shattuck had been similarly trained in Boston and contributed a paper to the same Society. Shattuck also went to London at Louis' request, and at the Fever Hospital there saw the two distinct affections, on which he reported to the Society on his return to Paris.* He insisted on the existence of two fevers in England.

In Germany, J. V. Hildenbrand had pointed out differences between typhoid and typhus as early as 1810, but also regarded them as varieties of the same disease and not distinct diseases. These views were maintained for many years in Germany, but since 1859, at least, correct notions have prevailed. In Great Britain, in 1835, Peebles of Glasgow, who had observed the rubeoloid eruption in the contagious typhus of Italy, pointed it out to Drs. R. Perry and A. P. Stewart. The former, according to Stewart, was the first to contend for the difference between the eruptions of typhus and typhoid. His writings as quoted by Murchison do not show this. Stewart, however, separated the two affections in a paper published in 1840.† In England it was not until 1849–51 that Sir William Jenner‡ clearly demonstrated their difference, and about the same time definite ideas were arrived at in France. Since 1850 the two diseases have been everywhere recognized and described as distinct and separate except in Germany, where the recognition came a few years later.§

Etiology.—The bacillus to which prevailing views ascribe typhoid fever was discovered by Eberth in 1880 in the intestine of a case of the disease. This observation was promptly confirmed by Klebs, Eppinger, Koch, von Meyer, Friedlander, Gaffky, and others, who found it in the intestines, lymphatic system, including the mesenteric glands and spleen,

* Amer. Med. Examiner, February 1, March, 1840.

† Edinburgh Med. and Surg. Jour., April, 1840.

‡ Jenner, Med. Chir. Trans., Vol. XXXIII; Edinburgh Mo. Jour. of Med. Sci., Vols. IX and X, 1849–1851; Med. Times, Vols. XX–XXIII, November, 1849, to March, 1851.

§ For an interesting and very much fuller historical sketch of the development of our knowledge of typhoid fever, see Murchison's treatise on the "Continued Fevers of Great Britain," 3d ed., London, 1884.

in the liver and the kidneys, the blood and bone marrow, and even bile and urine, as well as the rose-colored spots. It was secured in pure culture from the spleen and affected lymphatic glands by Gaffky in 1884. The bacillus is described as a short, rod-like bacterium whose length (three micromillimeters) is about one-third the diameter of a red blood disc and whose breadth is about one-third its length, though its size and shape vary somewhat with the medium and age of the culture. Its ends are rounded and sometimes there can be seen near them dark-colored, glistening, round bodies at one time believed to be spores, but recently this germ has been classed among those which do not form spores.*

Early observations have been rendered somewhat unreliable by the very close resemblance of the bacterium to that of the colon. The following method suggested by Elsner† for differentiating the two bacilli seems to be sustained by the experience of others:—To 500 gm. of chopped potato add a liter of water, and boil for one and a half hours. After straining through a cloth add ten per cent. gelatine. This potato gelatine should have a degree of acidity such that ten c. c. require 2½ c. c. of a decinormal soda solution to neutralize it. When using, one per cent. of iodide of potassium is added and plates made. In twenty-four hours the typhoid bacillus is scarcely visible, while the colon bacillus is well grown out. In forty-eight hours the typhoid bacilli present clear, shining, drop-like, finely granular colonies, the colon bacilli large colonies, more coarsely granular, brownish in color.

The bacillus stains readily in a saturated watery solution of methyl blue. Cultures may be made from the fecal discharges on the tenth day or later, but with difficulty, and many times are negative, probably because the bacilli are not numerous. E. Fränkel and M. Simmonds‡ early injected pure cultures of the typhoid fever bacillus into the blood of mice, rabbits, and guinea pigs with fatal results, which are now ascribed to toxins thus introduced. By introducing the cultures into the duodenum, Klemperer, Levy, and others caused lesions similar to those of typhoid fever, though more recently similar intestinal lesions have been produced by other bacteria, including the bacterium coli commune. The resisting powers of the typhoid bacillus are very great. It thrives at room temperature. The thermal death-point is given by Sternberg at 156° F. (69° C.). According to Klemperer and Levy, the bacilli remain vital for three months in distilled water, though in ordinary water the commoner and more vigorous saprophytes consume them. When buried in the upper layers of the soil they retain their vitality for nearly six months. Cold has no effect upon them, for freezing and thawing several times fails to kill them. They have lived upon linen for from sixty to seventy-two days, and on buckskin from eighty to eighty-five days. Sternberg has succeeded in keeping hermetically sealed bouillon cultures alive for more than one year. John S. Billings and Adelaide Ward Peckham in some experiments in the Laboratory of Hygiene, University

* Sternberg, Jour. of Amer. Med. Asso., August 22, 1891, p. 390.

† *Zeitschrift für Hygiene und Infectionskrankheiten*, January, 1896.

‡ v. Jaksch, *Klinische Diagnostik*, 1892, S. 213.

of Pennsylvania, dried bouillon cultures on threads and found that typhoid bacilli lived in a vacuum two hundred and seven days, in a desiccator over sulphuric acid two hundred and three days, in a closet two hundred and twenty-eight days, and proved more resistant than the bacillus coli communis or staphylococcus aureus. One-tenth to two-tenths per cent. carbolic acid added to a culture medium is without effect upon its growth. Of all agents except high heat, sunlight seems to be among the most powerful to destroy it. The experiments of Billings and Peckham,* just alluded to, go to show that insolation for two hours destroys 98 per cent. of the germs and in three to six hours kills all. This very important observation, made first by Janowski in 1890,† has been confirmed by Dieudonné.‡

L. Brieger announced in 1885 that the pathogenic action of the typhoid bacillus was due to a specific product of the bacillus, a typhotoxin. More recently R. Pfeiffer concluded that the specific poison of the bacillus is not present in the filtered cultures, but is closely connected with the protoplasmic bodies of the bacteria themselves. Whether typhoid fever is caused by the direct action of its specific bacilli or by a typhotoxin produced from them, the bacillus itself most frequently enters the blood through the stomach in drinking water or milk, in both of which it has been found during epidemics. It has been found in water filters by Harold C. Ernest and T. M. Prudden. It may also be inhaled. It is also quite well settled that the bacilli find their way into the food and drink through the careless disposition of the alvine discharges from typhoid fever patients. An oyster bed may be infected by sewage, green vegetables by polluted water sprinkled upon them. Whether the bacilli multiply outside the body after they have obtained access to the water of wells or rivers is not well settled, but judging from the large number of persons sometimes infected from those sources it is not unreasonable to conclude that such multiplication can take place. A most noteworthy instance was the epidemic of 1885 at Plymouth, Penna., U. S. A., where 1200 persons were attacked and 130 died, all the cases starting from a single subject, whose discharges contaminated the water supply. The bacilli develop rapidly in milk and in the soil. The relatively infrequent communication of typhoid fever to physicians, nurses, and others in close communication with the disease is explained by the fact that the contagion escapes from the patient in the stools alone, and as these are commonly promptly disposed of, the chances for the dissemination of the poison are correspondingly few. Carelessness in the disposition of these discharges, as the result of which they are allowed to dry on linen, whence the bacilli pass into the air of the room, does sometimes occasion the infection of nurses and physicians and others attending on typhoid cases.

Predisposing Causes.—Experience fails to establish definite individual predisposing causes of typhoid fever, but new comers are more

* "Influences of Certain Agents in Destroying the Vitality of the Typhoid and Colon Bacillus." Science, February 15, 1895.

† "*Zur Biologie des Typhus-Bacillus.*" *Centralbl. f. Bakteriol.*, VIII, 1890.

‡ *Beiträge zur Beurtheilung der Einwirkung des Lichtes auf Bacterien. Arbeiten aus dem kaiserlichen Gesundheitsamte.* Band IX, s. 405, 1894.

likely to be attacked than old residents, as early shown by the French physicians in Paris. It certainly often attacks the strong and healthy as fiercely as the feeble and delicate,—indeed, there is reason to believe more readily,—while allowance must be made for the more frequent exposure of the healthy. Thus caused, typhoid fever is unlimited in its distribution by climate or civilization, but it may be complicated by disease peculiar to certain localities, preëminently malaria. Typhoid fever is a disease of adolescents and adults under thirty, although it may occur at any age. Less common in children, perforation has been found in a child five days old, while not a few cases have been reported in sucklings. Infection *in utero* is claimed as possible because of successful cultures of bacilli from the fœtus. In the young the duration of the disease is short and the prognosis singularly favorable. On the other hand, it has occurred at the age of seventy-five, eighty-six, and even ninety. More men than women have typhoid fever, fully four to one, probably because of their more frequent exposure rather than greater susceptibility. The pregnant state seems to protect against typhoid fever. At least it is rare in the pregnant woman.

Typhoid fever is undoubtedly more common in the late summer and autumn months than at any other time of the year, whence one of the names, "autumnal fever." Heat has probably to do with the ripening of the cause, but the relation of moisture to such maturing is not so well settled. It has, however, been observed that hot and dry summers are followed by more cases than hot and moist summers. This is, on the whole, best explained by the observations of Buhl and Pettenkofer at Munich. They have shown that more cases succeed seasons when the ground water is low,—that is, when the springs are low and the upper layers of the soil comparatively dry,—than when the ground water is high and the soil is saturated with moisture to a point nearer to the surface. Under the latter condition of high ground water the germs are retained *in situ*. When the ground water is low, on the other hand, the constant circulation between the air in the loose soil and that above it conveys the germs upward, and they pervade the air accordingly. This, of course, has nothing to do with the causes of special epidemics, which may occur at any season, and may quite eclipse the operation of the ordinary causes. Thus the recent epidemic of typhoid fever at Plymouth, Penna., alluded to, began April 10th and raged with greatest fury during May and June. Other epidemics illustrate the same truth. Liebermeister prefers to explain the relation of typhoid to the hot and dry season by the fact that at this season the quantity of solid matter in springs is relatively larger, that the poison, in other words, is more concentrated, and therefore more virulent.

Morbid Anatomy.—The essential morbid anatomy of typhoid fever includes the changes in the lymphatic structures so constantly associated with the disease. These are more striking in the solitary glands of the ileum and their agminations known as Peyer's patches. The glands are enlarged by the accumulation of outwandering and proliferated leucocytes which develop to the stage of epithelioid cells, when they become necrotic and disintegrate. The acme of this process prior to disintegration is known as medullary infiltration and is reached from the eighth to the tenth

day of the disease. The disintegration is molecular or massive. The former is followed by a corresponding absorption, the latter by a massive discharge of the dead cells into the bowel, resulting in the well-known typhoid ulcer. This, when it represents a single follicle, is small and circular, not more than ⅛ of an inch (three mm.) to ¼ of an inch (six mm.) in diameter, large and elliptical when an entire Peyer's patch is involved. Such a patch is usually opposite the mesentery, has its longest diameter parallel with the length of the bowel and its shorter transverse, thus reversing the relations of the tubercular ulcer. Much larger ulcers are sometimes formed, especially toward the lower end of the bowel, by the union of others. The borders are commonly raised. The floor of the ulcer is usually the submucosa, or the muscular coat of the bowel, but it may be the peritoneum, and even this is sometimes sphacelated, appearing as an opaque white membrane which sooner or later breaks and the bowel is perforated. The discharge of its contents into the peritoneal cavity is followed by fatal peritonitis. More commonly the ulcer heals, and the patient recovers, but the normal glandular structure of the gut is not restored at the seat of the ulcer. Autopsy discovers ulcers in different stages of healing, sometimes all healed except the single fatal spot which has become the seat of perforation. The large intestine is also invaded, in probably about one-third of the cases, and the process may terminate here also in perforation. It may extend to the appendix, where, too, perforation has taken place.

Similar infiltration of the lymph nodules and lymph cords of the mesenteric glands and of the spleen may occur, contributing to the enlargement of these organs. In the spleen it is associated with an active hyperæmia which contributes to further enlargement, generally recognizable during life. The organ may reach twice or three times its normal size, *i. e.*, 14 to 20 ounces (435 to 650 gm.). There has even been rupture of this organ. Hemorrhagic infarcts have been found in the spleen in four to seven per cent. of cases coming to autopsy.

As lesions not essential to the disease may be mentioned cloudy swelling, granular and fatty degeneration of liver cells, lymphatic nodular areas in the liver, liver abscess with pylophlebitis, and acute yellow atrophy. Also cloudy swelling and granular degeneration of the renal cells, more rarely acute nephritis, which may even be hemorrhagic; also miliary abscesses in which typhoid bacilli have been found. Diphtheritic and catarrhal inflammation of the pelvis of the kidney and catarrhal inflammation of the bladder are occasionally present.

Changes in the respiratory organs are often found. Among the rarer of these are ulceration of the larynx and œdema of the glottis. Hypostatic congestion of the lungs is quite common. Even gangrene of the lungs was found in 40 of the Munich cases, abscess in 14, and hemorrhagic infarction in 129. Pleurisy and empyema are rare events. Dr. Arthur V. Meigs * has recently described changes of a hemorrhagic character in the lungs, and others in the muscular and nervous system the essential relation of which to typhoid fever remains to be demonstrated. Thrombosis of veins, especially of the femoral, causing the not very rare symp-

* Proceedings of the Pathological Society of Philadelphia, 1890.

tom of milk leg, occurs; more rarely there is thrombosis in the femoral artery, it may be from embolism. Endocarditis and myocarditis may be present. The latter condition is attested by a yellow, soft, and flabby muscle.

Notwithstanding the intensity of the nervous symptoms at times, meningitis is a rare event, though both serous and purulent forms have been met, with typhoid bacilli *in loco* as the apparent cause; also thrombosis of cortical veins. Granular and hyaline transformation of voluntary muscle may occur, as in the other fever processes; also parenchymatous changes in nerve trunks even when there have been no symptoms of neuritis. Abscesses in the parotid gland are a familiar complication; more rarely abscesses in the intermuscular tissue. General invasion of all organs and of the blood by the typhoid bacillus has been found once in William Osler's wards at the Johns Hopkins Hospital by Simon Flexner.

Symptoms and Course.—A certain period of incubation is necessary after the successful implantation of the bacillus before typhoid fever arises. This varies from a few days to two weeks and even longer. The period of incubation may be without symptoms, but a sense of weariness and indisposition to exertion, the latter often overcome by force of will, are usually present. A want of appetite and a slight coating of the tongue are not infrequent. The disease itself usually sets in very gradually and is often quite advanced before suspected,—indeed, sometimes well advanced, constituting the "walking" or "ambulatory" typhoid. In children the onset is less gradual. There may be headache, anorexia, a furred tongue, nausea, chilliness, but seldom a decided rigor. The disease may be ushered in by pain in the back or legs or muscles and nosebleed. The last has always been considered characteristic, and yet I meet it less frequently than might be expected from the text-book statements. More common is looseness of the bowels. All this time there is slight fever and the patient feels wretched. The fever and the discomfort increase and finally he goes to bed. The tendency to looseness of the bowels and epistaxis more than any other symptoms of this group justify strong suspicion of the existence of typhoid fever. Yet one or both are quite often absent. The abdomen soon becomes slightly distended and tympanitic, and pressure in the right iliac fossa will usually elicit tenderness with gurgling. At times there is colicky pain of varying severity independent of pressure, at others the gastric symptoms are marked and there is stubborn vomiting.

Usually about the eighth day, rarely later and sometimes a little earlier, *rose-colored spots* make their appearance on the skin of the abdomen and chest, more rarely elsewhere on the body. These call for further description. They are usually bright red in color, and are well compared to a flea-bite. They are very slightly if at all raised above the surface and disappear on pressure, to return instantly after its removal. Their number varies greatly. Sometimes they are very numerous, oftener there are four or five to ten, again one or two, most rarely none. When numerous they occur in successive crops, each crop lasting for two to four days. Histologically they are circumscribed, actively hyperæmic areas, the hyperæmia being excited by some irritant, which may be the typhoid bacillus itself, since it has been found in the spots. Only in

most malignant cases is there any blood found outside of the vessels, and when this occurs the spots can be made to disappear but partially on pressure. The association of roseolar spots is so intimate with the disease that they are regarded as pathognomonic.

In addition to rose-colored spots, *sudamina* are often present in large numbers on the skin, especially when the disease is associated with much sweating, but their occurrence is by no means constant and their association with other diseases in which there is perspiration is well known. More rarely *petechiæ* and *vibices* are noted in adynamic forms of the disease. An *erythema* is quite often found on the skin of the chest and abdomen in typhoid fever. Peliomatous patches,—the *tache bleuâtre*,—sometimes are found on the skin of the thorax, abdomen, and thighs; also the *tache cérébrale*, a red line with white borders, may be produced on drawing the nail over the skin, but neither have any symptomatic significance. *Herpes* is so rare that it is often spoken of as *negatively pathognomonic*. *Jaundice* is a rare symptom.

The *fever* is at once the most important and characteristic symptom, and from the temperature alone a diagnosis can be made. During the *increment* of the disease it exhibits a peculiar, tide-like evening rise and morning fall, while the temperature of each morning and evening is from one and a half to three degrees higher than that of the previous morning and evening. This initial period is rarely met at its very beginning, but should it be, it will be found to last commonly a week. Frequently because of the later date of observation it will be succeeded at the end of four or five days by the acme or *fastigium*, in which is continued the evening rise and the morning fall, but the evening and morning difference is less marked, the tidal character is no longer present, and the temperature is high throughout. The average duration of the fastigium is five to eight, rarely ten, days, being longer in severe cases and shorter in milder ones. It is during it that we meet the maximum temperature, quite often 105° F. (40.5° C.) or a little above, more rarely 106° F. (41.1° C.). A temperature of 106° F. is not infrequently followed by recovery, and while 107° F. (41.6° C.) and 108° F. (42.2° C.) and even 109° F. (42.7° C.) are met, such cases have invariably, in my experience, terminated fatally.

The fastigium is succeeded by the third stage, or *period of decrement* or decline, in which the reverse of the initial stage is shown by an evening temperature lower than that of the previous evening, and the morning temperature lower than that of the previous morning, but with the evening temperature still higher than that of the morning of the same day. This decline continues until the normal is reached, and from one to two weeks are consumed before that is attained. The whole is much better shown and more easily understood from a chart than from a description in words. Such a chart of the temperature uninfluenced by treatment is seen in Fig. 1, although the rise and fall are not always as regular as indicated. In a typical case one might safely place the first stage at four days to a week, the second, or fastigium, at seven to ten, and the third about as long as the second, the short period corresponding to a mild case and the longer to a severe one. The fever does not always reach the higher temperature shown in the chart and sometimes the maximum never reaches

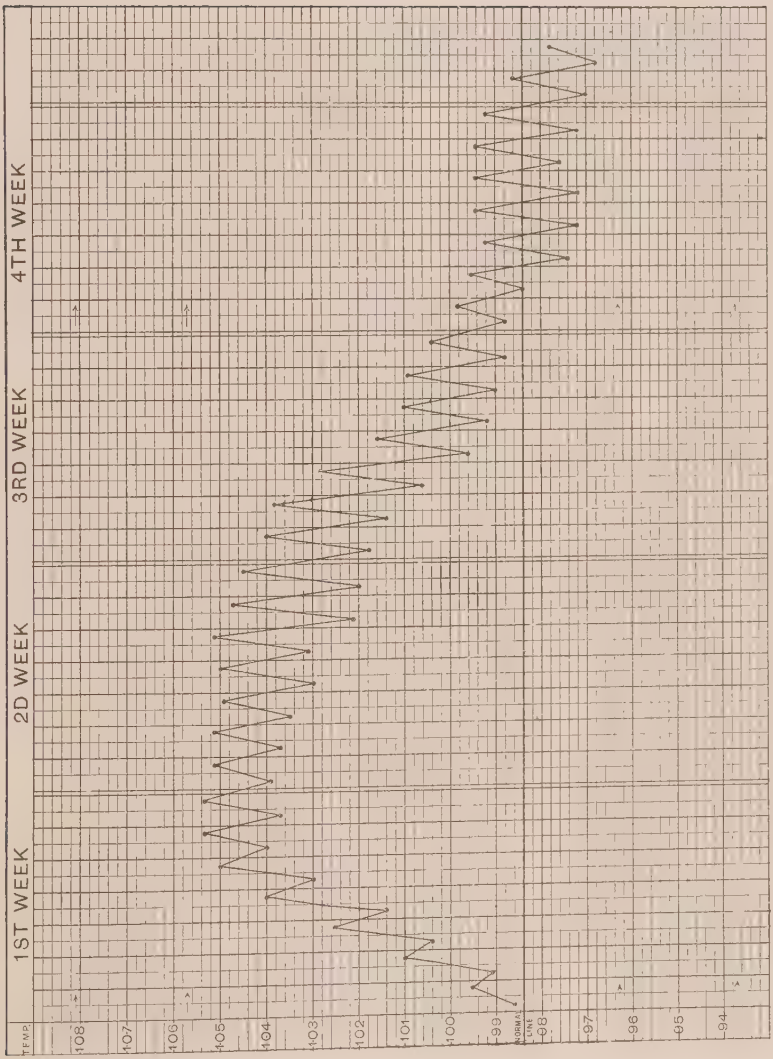


FIG. 1.—TEMPERATURE CHART OF A TYPICAL CASE OF TYPHOID FEVER UNINFLUENCED BY TREATMENT.

102° F. (38.9° C.). On the other hand, there is sometimes a difference of three or four degrees in the morning and evening temperature and the latter may drop to the normal. In ordinary cases the evening temperature falls to the normal in the course of the fourth week, but in severe cases the temperature keeps up during the fifth and even sixth week, these cases having almost invariably extensive ulceration with great tenderness of the abdomen and meteorism. Many of them terminate unfavorably by hemorrhage or perforation.

When the disease begins with a chill, a rare event, the temperature rises more rapidly in the beginning. Sudden falls of a decided character may occur in consequence of hemorrhage from the bowels or the nose or from collapse after perforation of the bowels. Sudden rises are produced by indiscretion in diet and over-exertion or the supervention of some acute inflammatory affection, as pneumonia.

Copious *sweating* characterizes some cases of typhoid fever, though the skin is more commonly dry. Sometimes during the reaction after a cold bath there is perspiration. The profuse sweats first alluded to are not attended by a reduction of temperature. On the other hand, they are sometimes present when the temperature is highest. Cases of recurring paroxysms of chill, fever, and sweat are reported, which simulate intermittent fever, and may reasonably be mistaken for it.

The *pulse* is not as a rule very frequent, 90 to 120 being the usual range, while the neighborhood of 100 is quite frequently maintained. In grave cases it becomes more frequent, 140 or more, when, if maintained, it is a rather unfavorable symptom, due to high temperature or complications. Temperature and pulse do not always increase *pari passu*. Dicrotism may occur with frequent pulse, but dicrotism also occurs in the early stage, when it is regarded by some as diagnostic. During convalescence it gradually resumes its normal character, and sometimes becomes quite frequent, falling to 30 or less. The *breathing rate* commonly advances with the rate of the pulse, but is sometimes increased in frequency by temporary causes and rarely is disproportionately slow.

The *heart sounds*, at first natural, grow less loud as adynamia progresses, and the first may even disappear in grave cases. Sometimes a soft systolic murmur is heard at the apex.

As the disease advances, the *tongue*, previously furred, tends to become dry and brown, clearing, however, at the edges and tip as the case improves. In severe cases, especially if the mouth is not kept clean, *stomatitis* with fissures and bleeding may occur and sordes collect on the teeth, while the lips become covered with black crusts, constituting the "fuliginous coating." These phenomena are almost unknown with the bath treatment. Mild grades of pharyngitis, producing difficulty in swallowing, sometimes usher in the attack, more particularly in certain epidemics.

The *diarrhœa* of typhoid fever has been alluded to. Usually corresponding in severity with the extent of the local lesion, it is seldom troublesome or difficult to control, and is sometimes absent throughout. The stools are apt to be greyish yellow, and about the consistence of pea soup. *Hemorrhage* from the bowels, also a consequence of intestinal ulceration, is a serious symptom, but by no means always fatal,

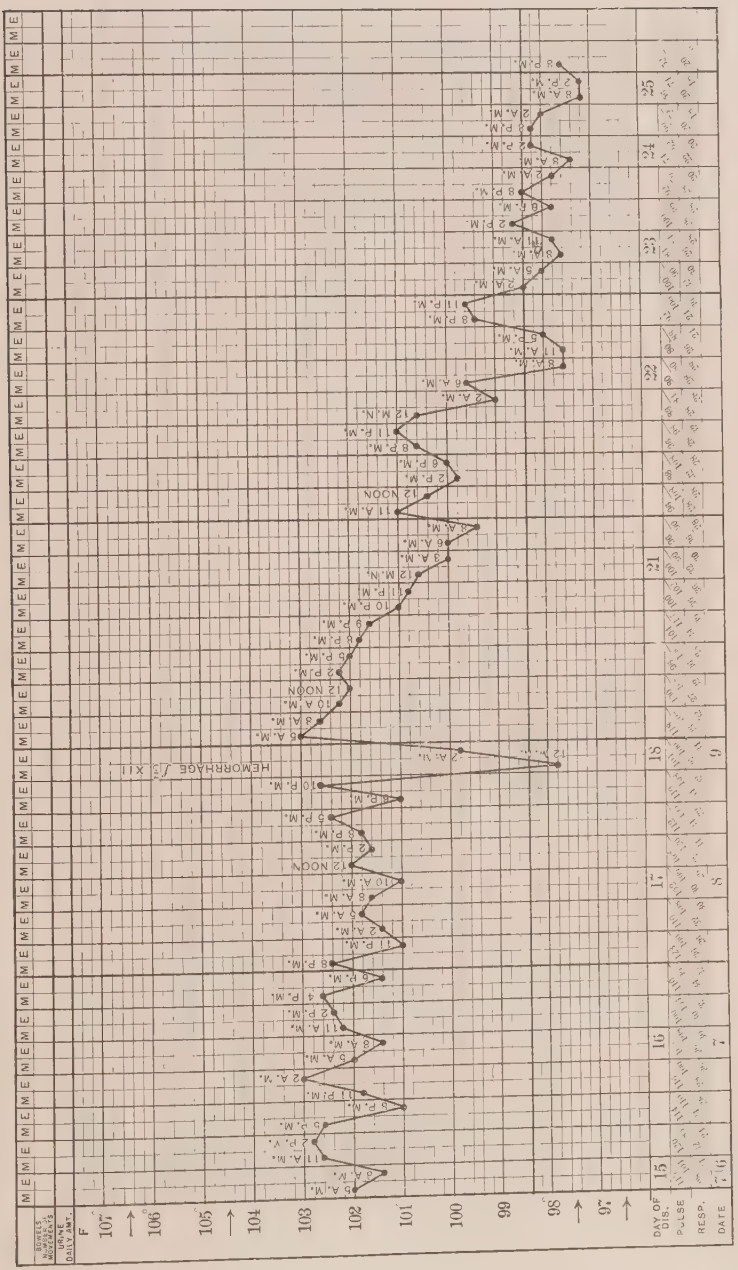


FIG. 2.—SHOWING DROP IN TEMPERATURE INCIDENT TO INTESTINAL HEMORRHAGE.

though large quantities of blood are sometimes discharged *per anum*. The occurrence of such hemorrhage is followed by a rapid reduction of the temperature, as shown in the appended chart, and a pallor and faintness such as is common to large hemorrhages elsewhere. As stated, very profuse hemorrhages may be followed by recovery, and it is barely possible that a favorable influence may sometimes be exerted by them. Very rarely a patient will bleed to death.

Meteorism in mild degrees is an almost constant symptom. The accumulation of gas is commonly ascribed to atony of the bowels. Its presence in high degrees adds to the seriousness of the case, since it corresponds usually with the extent of bowel lesion.

Delirium is less constantly present in typhoid fever than in typhus, and may be absent throughout. It may, however, be very active, requiring the patient to be carefully watched to prevent him from leaving his bed and seriously endangering his life. More than one victim has leaped from a window with fatal results under such circumstances. In certain cases, especially where the initial headache is very intense, this symptom continues, and to it are added fever and delirium so extreme that meningitis is simulated, though true meningitis very rarely occurs. Such cases are called "nervous cases." A tendency to drowsiness, and even to stupor, suggested the common name of the disease, but it is less characteristic than in typhus. *Muscular tremor* is a symptom in severe cases where it would seem to indicate a muscular weakness or exhaustion, which may be an effect of high temperature or of the specific poison of the disease. Carphologia, or "picking at the bedclothes," is a symptom of which the unfavorable import has been somewhat exaggerated, probably because of the popular familiarity with Dame Quickly's interpretation in Falstaff's illness. Concurrently with these "typhoid" symptoms, the tongue reaches its maximum dryness, may be dark and leathery in appearance, while sordes may collect on the teeth.

Apart from an initial bronchial catarrh which sometimes ushers in the disease, the typhoid patient sooner or later acquires a *slight cough*, due to hypostatic congestion of the lungs, but is easily kept within bounds by frequent changes in the position of the patient. Occasionally the cough is quite severe, but seldom requires more active treatment than this. The initial bronchial catarrh occasionally assumes severity, while more rarely the symptoms and signs of pneumonia usher in the disease.

Enlargement of the spleen is an almost constant clinical feature of typhoid fever. If the vertical dulness exceeds the depth of two ribs and an interspace, enlargement is present. Not only may such enlargement be recognized by percussion, but by palpation as well. Clinicians generally lay great stress on palpation, and enlargement may be detected by it when the organ eludes percussion by reason of tympany. Hermann Eichhorst advises the following method of examination: Put the patient in the right diagonal position and lay the finger gently between the anterior ends of the 11th and 12th ribs, when the enlarged organ can almost always be felt with every deep inspiration, in spite of meteorism. At times the outline is indistinct, at others both the tip and anterior edge of the organ can be distinctly perceived. Strong pressure should not be exerted by the fingers, for in this way the spleen may be insensibly

pressed backward into the excavation of the left hypochondrium. Enlargement can generally be detected at the end of the first week or in the first half of the second week, when the organ may reach twice or three times its normal size. By the end of the third week it begins to diminish in size. The enlargement may be accompanied by tenderness.

The *urine* in typhoid fever is always dark hued and concentrated, with a correspondingly high specific gravity. Often when the fever is high the urine contains a small amount of albumin. When complicated with nephritis there is more albumin, and tube casts are also present. The toxic properties of urine are increased during typhoid fever, especially while the cold baths are being used.

The so-called *diazo*-reaction of urine, to which attention was first called by Ehrlich in 1882, is so constant in this disease as to be deservedly regarded as a symptom. It was found by John Hewetson in 136 out of 196 cases, and by A. R. Edwards in 128 out of 130 cases, and by Simon in 22 out of 26 cases. I have never found it absent where the test was made sufficiently early.

Three solutions are kept in separate bottles.

(1) A five per cent. solution of hydrochloric acid saturated with sulphanilic acid. This solution should be fresh.

(2) A half of one per cent. solution of sodium nitrite.

(3) Ammonium hydrate.

When it is desired to make the test, 40 c. c. of (1) and 1 c. c. of (2) are mixed. The hydrochloric acid, acting on the sodium nitrite, liberates nitrous acid, which in its nascent state combines with the sulphanilic acid, producing diazo-benzine-sulphonic acid. Equal parts of this mixed solution and urine are thoroughly shaken; enough of the ammonia is then allowed to flow carefully down the side of the tube to form a colorless zone above the urine mixture. At the junction of the two fluids a dark garnet or cherry red ring will form if the reaction takes place, and if the tube is well shaken a uniform red color is imparted to the entire fluid, which, when allowed to stand for some hours, shows a characteristic olive green precipitate, the upper layer of which as a rule has a still darker green color. The reaction occurs about the time of the appearance of the rash and usually continues until the twenty-second day, but it may disappear before the end of the second week. It is, as stated, symptomatic, but not diagnostic, certainly not pathognomonic, as it occurs in many diseases with high fever, among which measles and miliary tuberculosis are conspicuous. It may, however, be regarded as negatively pathognomonic, that is, its absence is strongly presumptive against the presence of typhoid fever.

The state of the *blood* in typhoid fever early claimed attention, and even the earliest observers, beginning with LeCanu in 1837, noted a *falling* off of *red* blood *corpuscles*. This observation has been essentially confirmed by the most recent studies with modern accurate methods, among which those by Ouskow* and of Khetagurow† and of W. S.

---

* "The Blood as a Tissue." St. Petersburg, 1890.

† "Pathological Changes in the Blood in Typhoid Fever." Inaug. diss. St. Petersburg, 1891.

Thayer* are conspicuous. At the beginning of the fever the number of red blood corpuscles is normal and even at the upper limit of normal, because the patients are apt to be young and strong, while in some instances the initial diarrhœa or pronounced sweating may cause slight concentration of the blood. During the first two weeks the number of red corpuscles gradually falls, though but slightly. With defervescence they fall off more rapidly, reaching a minimum usually about the

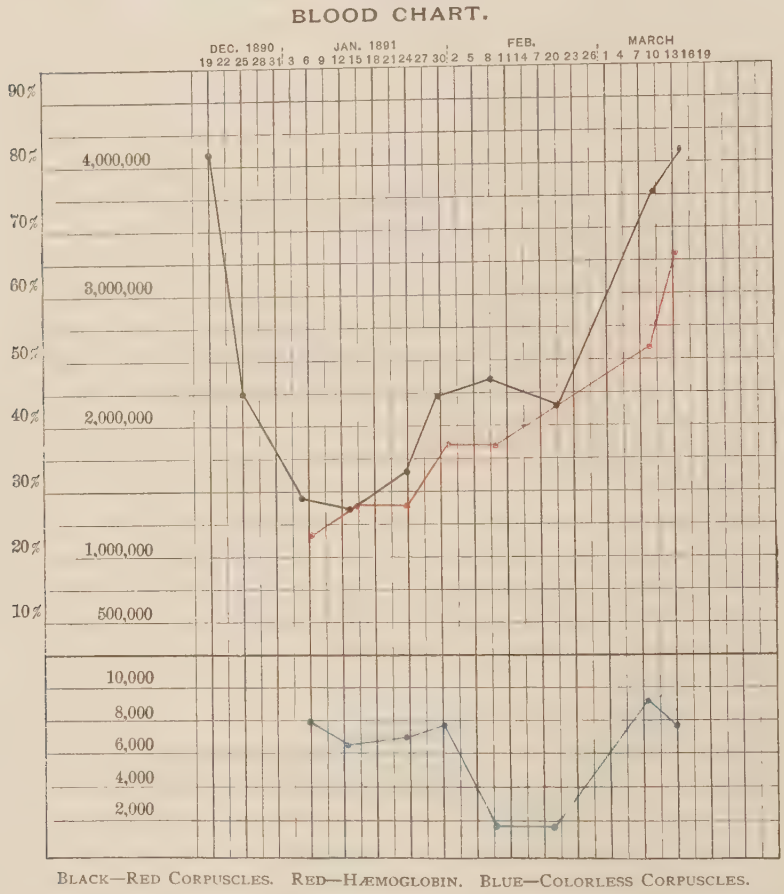


Fig. 3.—Chart Showing Anemia of Typhoid Fever.—(*From Thayer's Monograph.*)

first week of convalescence, after which there is a gradual rise to the normal during convalescence, followed again by a possible slight fall when the patient gets up. The fall in the number of red corpuscles while relatively slight bears usually a direct relation to the severity of the case. The *hæmoglobin* is always reduced and the reduction is relatively greater than the corpuscular loss, with an even slower return to

* "Two Cases of Post-Typhoid Anæmia, with Remarks on the Value of Examination of the Blood." Vol. IV., Johns Hopkins Hospital Reports, 1895.

the normal. Extreme anæmia with a blood count as low as 1,300,000 corpuscles per cubic millimeter and hæmoglobin as low as 20 per cent. has been met.

The number of *leucocytes* per cubic millimeter, normal at the beginning, tends also to *diminish* slightly through the course, reaching a minimum toward the end of defervescence, increasing again with the beginning of convalescence, and reaching the normal after several weeks. Thayer regards the absence of leucocytosis of real diagnostic value, being in marked contrast with the distinct increase in the number of colorless corpuscles and over-ripe elements (multinuclear cells) characteristic of miliary tuberculosis associated with any local inflammatory process, such as exists in the majority of cases; in contrast also with other acute inflammatory processes and septic fevers, in which there is always marked leucocytosis, typhus fever is unattended by blood changes, and while in pneumococcus infection there may be no leucocytosis, there is no *diminution* in the leucocytes, as in typhoid. It is important to remember that cold baths have the effect of producing a decided temporary increase in the number of leucocytes, probably rather, as Thayer thinks, in consequence of an accumulation of white cells in the vessels of the surface than as the result of a true leucocytosis.

Special and Unusual Symptoms and Unusual Modes of Onset. Deviations from the Typical Course.—It has been mentioned that while slight chilliness is often an initial symptom, *severe rigor* at this stage rarely occurs. It does, however, happen, as in 13 out of 79 of William Osler's cases. More frequently chills have been observed in the course of the disease from some one of the following causes:—

(1) At the onset of a relapse.

(2) As a result of treatment, especially by antipyretics internally, guaiacol externally, or of a hypodermic injection of a sterilized culture of typhoid bacilli.

(3) At the onset of complications, such as pneumonia or pleurisy or thrombosis.

(4) From sepsis during convalescence in severe and protracted cases. Chills may be frequent and severe under these circumstances and of grave import.

(5) From concurrent malaria.

In epileptics who acquire typhoid fever, the latter disease is very apt to be ushered in by an unusual number of epileptic *convulsions*, which continue frequent until the fever becomes established, then diminish, and finally cease, often not recurring until some time after recovery, deluding the victim and his friends to believe that the chronic malady has disappeared. It returns, however, sooner or later. The same is true of choreic attacks. In diabetes the sugar may also disappear during the fever.

Among the more unusual modes of onset should be mentioned cases beginning with severe bronchitis; those with the initial symptoms of pneumonia, including chill; or with intense nervous symptoms, suggesting cerebro-spinal meningitis. Among the latter are intense headache and photophobia, combinations rapidly passing over into active delirium, with

muscular twitching and retraction of the head, constituting the *nervous* or *meningeal* form.

Deviation from the typical course is exhibited in the so-called *abortive form.* This doubtful form is said to be more sudden in its onset, beginning with shivering and fever of 103°F. (39.4°C.) or higher. The rose-colored spots appear at from the second to the fifth day. The fever falls at the end of the first week or beginning of the second, commonly by crisis with a sweat, after which follows convalescence. The *hemorrhagic* is a grave variety characterized especially by cutaneous and mucous hemorrhages, and is also fortunately rare. The *mild* form is sometimes so mild as scarcely to be recognized as typhoid fever, and is often called gastric fever or simple febricula. There is, however, no more important lesson for the inexperienced practitioner to learn than that some cases which begin as a mere febricula may pass over into forms of great severity, which may even terminate fatally. A very rare form is the *tonsillar typhoid*, in which whitish elevations appear on the tonsils which subsequently ulcerate.

Complications.—In malarial districts particularly it is not unlikely that malarial fever and typhoid fever may be associated, though errors of diagnosis are often made, remittent fever being taken for such association, while uncomplicated typhoid is also sometimes similarly misinterpreted. To the association referred to the term *typho-malarial* may be applied, but it is to be distinctly understood that there is no separate disease which is to be thus indicated. Persons with tuberculosis, epilepsy, and other forms of chronic nervous disease are liable to typhoid fever, while measles, chicken pox, and especially erysipelas may befall a typhoid case. Typhoid fever itself predisposes to tuberculosis, and not a few patients recover from the former disease to fall into the latter.

Thrombosis of the femoral vein, more frequently the left, resulting in *phlegmasia alba dolens*, or milk leg, is a complication which often greatly delays convalescence. It occurs, according to Murchison, in one per cent. of all cases. It sometimes invades both legs in succession and may extend into the iliac veins and vena cava. However tedious the recovery, it takes place ultimately almost without exception. Very rarely there may be suppuration. Bacilli have been found in the thrombus. Arterial as well as venous thrombosis may occur and the former may start with embolism; femoral arterial obstruction is most common, resulting in gangrene of the leg and foot. Embolic abscess may occur in the kidney and lung.

Noma has occurred as a complication or sequel in children.

Albuminuria is present to some extent in all severe cases, commonly as the result of the fever, but sometimes is the direct result of an *acute nephritis*, already referred to, when it is accompanied by the usual tube casts with or without other symptoms of nephritis. Recent French statistics place albuminuria, regardless of its cause, at over 20 per cent. While such albuminurias are found in grave cases, they do not appear to add greatly to the gravity of the case, and recovery is by far the more usual termination. More rarely nephritis may develop in the mild form during convalescence. Most rarely it may be the initial symptom of the disease, as a nephro-typhoid analogous to the

pneumo-typhoid to be described, in which it may mask the true disease by the severity of its symptoms. It is well named by the French *fièvre typhoide à forme renale*. Only the appearance of the intestinal symptoms and the spots clear up the diagnosis. Such nephritis may rarely be hemorrhagic.

It has already been said that *pneumonia* may usher in the disease, and a few words may be said here of the relation of the two conditions, pneumonia and typhoid fever. The term *typhoid pneumonia* is one in common use by many who have no definite notion of its meaning, and, like the term typho-malarial, has occasioned confusion. In the first place, the case may begin as a lobar pneumonia, the intestinal symptoms appearing at the end of the first week or later, at which time also the spots may appear, establishing the diagnosis, while the usual crisis of pneumonia fails to make its appearance. Again, a pneumonia may supervene in the second or third week of a typhoid fever as a complication in which the true relation is less difficult to determine. Finally, there may be a true pneumonia, to which stupor, a dry tongue, and general adynamia may be added, without the distinctive lesions of typhoid fever. This is true typhoid pneumonia, which it may not always be easy to separate from the typhoid fever beginning with pneumonia. Both of the forms of pneumonia may be caused by the typhoid bacillus or the pneumococcus. Hypostatic congestion has been referred to. Many cases formerly thus named are really cases of catarrhal or lobular pneumonia belonging to the class of inhalation pneumonias. Such are prone to terminate in abscess and gangrene. *Pleurisy* may occur and have the same relations to the disease as pneumonia. It is, however, more rare, but may also be purulent.

Certain *suppurative processes* sometimes included as symptoms are to be regarded rather as complications than symptoms. Of these those about the ear are the most serious. They are, however, less frequent in typhoid than in typhus fever. They are most common in the parotid gland, where, however, the inflammatory process does not always terminate in suppuration, occasionally resolving itself with or without local treatment. The duct of Steno is probably the route of infection in these cases by the pus organisms which find conditions favorable to their work. The middle ear may be invaded, producing otitis media. Here the Eustachian tube becomes the route of infection. Sometimes abscesses are multiple.

The bladder may be a seat of suppuration and pyuria is not infrequently present. George Blumer found it in 10 out of 60 cases, or nearly 17 per cent., of a series admitted to the Johns Hopkins Hospital. I have met it only once in a pronounced form in a series of 41 cases. It is believed to be caused in some cases, at least, by the typhoid bacillus. The inflammation may extend to the pelvis of the kidney or start therefrom. Orchitis is also an occasional symptom during convalescence.

Cardiac complications, including pericarditis, endocarditis, and myocarditis are sometimes present. The latter may be a cause of sudden death.

Neuritis is an occasional complication or sequel in both the local and multiple form. Osler found it, however, in but four of 389 cases. The pain may be severe and associated with the usual tenderness of the

nerve trunks. I have met it rarely, but have at this time a patient under my care who has made a splendid recovery under the tub-bath treatment, but has the exquisitely tender toes first described by Handford. The tenderness is so great that the bed-clothing must be kept raised by a cradle. *Tetany* sometimes succeeds typhoid fever.

Sequelæ.—Two sequelæ of typhoid fever, neither of frequent occurrence, are conspicuous by their symptoms. They are *insanity* and *tubercular phthisis.* The former is often typical acute mania requiring the utmost vigilance to prevent the patient from injuring himself and others and from escaping from the house or jumping from a window. Although this form of insanity is often prolonged for many weeks, the prognosis is singularly favorable and recovery sooner or later takes place. Tubercular phthisis when it occurs has its predisposing cause in the lower tone of cell life, favoring the successful implantation of the specific bacillus, and is followed by its usual consequences.

Post-typhoid *bone lesions* are surprisingly common when investigated. Sir James Paget, Murchison, W. W. Keen, Haywood, Harold C. Parsons, and others have collected many cases. They include osteitis, necrosis, and periostitis. Ebermaier, in 1887, obtained from two cases of suppurative post-typhoid periostitis the bacillus of Eberth in pure culture, and since then quite a number of cases have been reported. Whence pyogenic properties of this bacillus may be inferred. Other bacilli, viz., the staphylococcus, streptococcus, and pneumonococcus, are, however, at times associated. Golgi also produced suppuration by injecting pure typhoid bacilli subcutaneously at a distance from the fractured ends of a long bone in a lower animal. The pus showed in culture only typhoid bacilli.

The *typhoid spine* is a sequel of undetermined and perhaps not always of identical nature, succeeding typhoid fever, to which Gibney of New York called attention in 1889. There is severe pain in the back, commonly aggravated by motion. The pain may be throughout the whole spinal region or limited to the cervical, dorsal, or lumbar. From the latter it may extend toward the hips. It may be a spondylitis, but is probably a pure neurosis. Allied to this condition is perhaps an obstinate periostitis of the sternum or the crest of the ilia or front of the spinal column after typhoid fever, alluded to by William Pepper in the "Text-Book by American Teachers." These conditions are rare and sometimes, at least, may be coincidences.

Relapses.—These occur readily, succeeding, it is commonly taught, upon premature relaxation of diet. The demand of the convalescent for change in food, and especially for solid food, is often well-nigh irresistible, but should be denied until the temperature has been normal for a week. With our present views as to the etiology of typhoid fever it seems scarcely explainable why relapses should be induced by this assigned cause. For while such indiscretion would reasonably be expected to renew internal lesions, it would not be expected to revive the life of the original cause, the bacillus. The necessary symptoms are those which are essential to the primary diagnosis, viz., the characteristic spots, a return of the tidal or step-like temperature, and scarcely less so the enlarged spleen, and all of these after complete defervescence. The attack is, however, usually less severe, the duration shorter, and recovery the rule. Relapses are to

be distinguished from recrudescence, which is a simple return of fever, often, also, induced by lapses in diet, and may be regarded as a warning. Relapses may be multiple. Transverse markings on the nails incident to multiple relapses are sometimes noted. The number of relapses varies greatly, in the experience of different observers from one to 18 per cent. Certainly it is smaller with the bath treatment.

Diagnosis.—Typhoid fever is usually easily recognized by the fairly well-trained medical man, while the experienced hospital physician may even know the disease by the dull, dusky facies—at least it was so before the bath treatment became general. At other times diagnosis may have to be delayed until the distinctive signs appear. The peculiar range of temperature is the most distinctive symptom, and from it alone the diagnosis may be made. The rose-colored spots, occurring about the eighth day, are conclusive if present, but they are occasionally absent. Diarrhœa is less constant and in my experience nosebleed still less so, but more distinctive. Both, however, require to be weighed in association with other symptoms. The resemblance of typhoid fever to certain cases of rapid consumption has long been recognized, but the modern temperature chart has greatly diminished the difficulty of distinguishing them. Certain cases of malarial fever, especially the autumnal type, also very closely resemble typhoid, but here, too, the temperature diagram is not identical, while the usually easy recognition of the malarial organism completes the solution. Where the two diseases are concurrent, as is sometimes the case, the difficulties are increased.

Mention has already been made of the close resemblance of the so-called nervous variety of typhoid fever to cerebro-spinal meningitis, and it is sometimes so misinterpreted. As the disease progresses, however, the distinctive signs develop and the correct diagnosis is gradually made. Further, unless an epidemic of cerebro-spinal meningitis prevails the probability that this combination represents the early stage of typhoid fever is far greater than that it is cerebro-spinal meningitis. The popular term, "brain fever," now passing into disuse, doubtless included many of the cases of nervous typhoid.

More misleading, even though less frequent, are the cases beginning with decided pulmonary symptoms suggesting pneumonia rather than typhoid fever, and unless the physician is awake to the possibilities of such a beginning and watches further developments the case may be regarded as one of typhoid pneumonia.

Certain cases of concealed suppuration resemble typhoid fever in the symptoms produced and may for a time mislead. But again the temperature chart after a few days' observation will solve the question. It is in such cases that a study of the blood is of value, the presence of leucocytesis pointing to suppuration, and its absence to typhoid.

Prognosis.—The mortality of typhoid varies so much in different epidemics and under different circumstances that statistics are of doubtful value in measuring fatality. Extremes of mortality are as low as one per cent. and even less by the Brand bath method as carried out on the Continent of Europe, and as high as 55, the latter in army practice during campaigns and among negroes. The average

of all may be put down approximately at from 10 to 30 per cent. before the Brand cold tub treatment was instituted. Prior to this, hospital treatment appeared less successful than that of private practice. Since its introduction, because of the greater ease with which that treatment can be applied in hospitals, this can hardly be said to be the case.

In private practice a decided majority get well, fully 80 per cent., with rest, liquid diet, and family nursing. With skilled nursing, judicious feeding, and cautious symptomatic treatment, a larger proportion of recoveries takes place, say 90 per cent. In hospitals where the Brand method is correctly carried out there is an easy reduction of mortality to seven per cent. and less. In this country the results have not been quite as satisfactory as on the Continent of Europe. The mortality of William Osler at the Johns Hopkins Hospital, Baltimore, has been 7.3. My own at the Hospital of the University of Pennsylvania and at the Philadelphia Hospital has been 7.3, that of James C. Wilson and others at the German Hospital up to January 1, 1896, 7.25 per cent., astonishingly uniform results. Brand's own mortality has been but 1 per cent. Of my own cases treated by the Brand method all who died perished through perforation or hemorrhage of the bowels.

Unfavorable symptoms are persistent high temperature, above 105° F. (40.5° C.), low muttering delirium, extreme tympany, hemorrhage from the bowels, and the signs of perforation. Walking typhoid has been almost always fatal in my experience, exhaustion being apparently caused by the continued muscular effort during fever. Sudden death by syncope occasionally occurs, sometimes when least expected, during convalescence, or it may occur during the acme of the fever. In either event the immediate cause is not evident. Sudden death is much more frequent in men than women, 114 to 26, according to Dewèvre's statistics, a surprising and almost incredible difference.

The *prognosis in children* under fifteen is especially favorable. Recovery takes place in them with few exceptions, while I have been struck with the number of fatal cases in young people from eighteen to twenty-two. Then follows a period favorable to recovery, but after forty the mortality again increases. The dangers at this older age appear to be from complications, especially pneumonia, as the symptoms peculiar to the diseases are not increased in severity. The prognosis in pregnant women is grave. In the first place, the pregnant woman usually aborts in the second week. According to L. Brieger, the mortality was 20 per cent. of cases treated by other than the bath method. The results of the bath treatment seem to be better. Under any circumstances more women die of typhoid than men,—this, too, though the disease is more frequent in men than in women. Fat persons bear the disease badly.

Treatment.—*Rest and Diet.*—The primary conditions of a successful treatment of typhoid fever are rest in bed and a liquid diet, of which milk is the type. No one questions the necessity of putting the typhoid fever patient absolutely at rest in bed and permitting him to rise for no purpose until convalescence is thoroughly established. That the diet should be liquid is as little disputed, while milk is generally conceded to be the safest form. It should be given at stated intervals, say once in two hours, in doses of four to six or even eight ounces, as circumstances determine. Very rich

milk is not indicated, hence such milk should be diluted with water or carbonic acid water, Vichy, or lime water. The stools should be closely watched for undigested fragments of casein, and when these are present the milk should be reduced in quantity or further diluted. If there is diarrhœa, the milk should be boiled while this lasts, and in obstinate cases peptonized. Animal broths of mutton or of chicken, also beef-peptonoids, may be associated with milk when change is demanded, but they are not as suitable, while beef tea and essences are harmful. Where the stomach is very irritable albumen water may be substituted, in the proportion of the whites of two eggs to a pint of water, to which may be added a little lemon or whiskey or brandy, if stimulants are indicated. Wine whey may also be associated or substituted where for any reason milk cannot be used. In extreme feebleness of digestion, peptonized milk may be administered by the rectum, but this is rarely necessary. Not more than four ounces of any nutriment should be administered at one time by the rectum.

The Brand Bath Treatment.—In addition to rest and liquid nourishment the treatment which my own experience and a careful study of the experience of others places easily at the head in every case where it can be carried out is the cold tub-bath treatment, commonly known as the Brand treatment. Our method in carrying out the treatment in the Hospital of the University of Pennsylvania is as follows:—

Before the bath the patient is first encouraged to empty the bladder, and if sweating, he is wiped dry. He is then covered loosely with a sheet and gently lifted into the bath sufficiently filled with water at 70° F. (21° C.), provision being made to rest the head upon an air cushion or platform. Unless very weak he may even step from the edge of the bed into the tub, which should be lower than the bed. During the bath he is vigorously rubbed by the nurse and encouraged also to rub himself. A compress wrung out of ice-water is kept upon his head, or water at the same temperature is poured at intervals upon it, say, three times in the course of the bath, or the head is sponged with cold water from time to time. This is important in severe cases with decided nervous symptoms. At the end of fifteen minutes he is lifted on to the bed, which has been previously protected with a mackintosh and blanket. The wet sheet is replaced by a dry blanket and the patient is rubbed dry. When this is accomplished the under blanket and mackintosh are withdrawn and he is comfortably covered.

As soon as the patient ceases to shiver after his removal from the bath, which is usually in twenty minutes, the temperature is taken with a view to determining the effect of the bath. If delayed longer the patient may be in a restful sleep, and to wake him for the purpose of taking his temperature is needlessly disturbing. After this the temperature is not again taken until three hours after the bath. If then it is 102.2° F. (39° C.) or above, the bath is repeated. If the temperature is 102° F. (39° C.) or below, but above 101° F. (38.2° C.), it is taken again in an hour; if below 101° F. (38.3° C.) and above 100° F. (37.8° C.), in two hours; if below 100° F. (37.8° C.), not until three hours; but whenever the temperature reaches 102.2° F. (39° C.) the bath is given, provided three hours have elapsed since the

last bath. This makes more than eight baths in the twenty-four hours impossible.

The effect of the bath upon the temperature varies with the stage of the disease, it being frequently the case throughout the first week that it is reduced less than one degree, while toward the end of the second week and in the third week a fall of two or more degrees is quite usual. In addition to the lowered temperature the immediate effect of the bath is

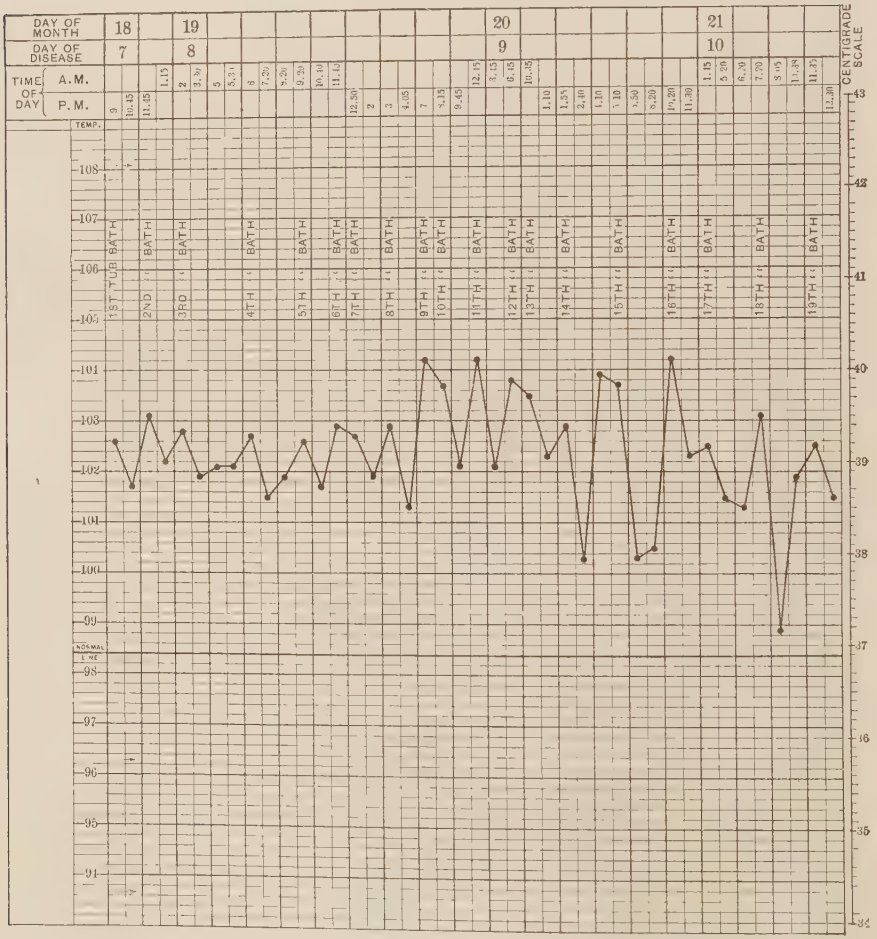


FIG. 4.—CHART CONTRASTING THE DROP IN TEMPERATURE AFTER THE BATH EARLY AND LATER IN THE DISEASE.

to add strength to the heart and volume to the pulse. The shivering, which begins five to ten minutes after the immersion, is not allowed to interfere with the continuance of the bath, and it very rarely happens—indeed, almost never—that anything occurs to interrupt the bath. It would be wrong, however, to say that there are no conditions under which it should be discontinued and the patient at once restored to bed. Such conditions would be an almost absolute pulselessness with a blue,

cyanosed appearance of the skin. Should this occur, hot-water cans should be applied to the feet and legs after the patient is put to bed.

The more remote effect of the bath may be said in a word to be mildening of the symptoms in every particular. Delirium and stupor are scarcely known. The dry tongue is very much more infrequent, and diarrhœa rarely demands other treatment. In the majority of cases I give no medicine, but do not hold myself bound to such course, meeting whatever symptoms seem to demand it by appropriate treatment. For a time I used to give the patient a little whiskey and water during the bath. Recently I have discontinued this, unless there seems some special reason for it. There is, however, no harm in it, and it serves to entertain and comfort the patient. I do not give a preliminary dose of calomel, as recommended by some, as there seems nothing gained by it.

None of the complications except hemorrhage from the bowels are allowed to interfere with the carrying out of this treatment, nor is menstruation or even pregnancy. The baths are discontinued during hemorrhage, lest the necessary movements of the body should excite hemorrhage; but with the portable bath-tub to be described there need be no interruption even during hemorrhage, should the baths be indicated by the temperature.

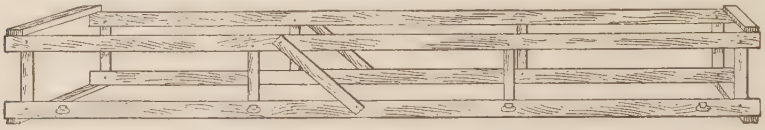
In private practice the difficulties of the treatment are greatly increased—unfortunately, sometimes are insuperable. They consist chiefly in the difficulty in arranging the bath and the tax on the attendants. By means of a portable tub devised by Dr. A. H. Burr, of Chicago, a very large part, if not the whole, of these difficulties is removed. Dr. Burr's tub consists, first, of a large rubber sheet, with rings attached near its margins by elastic tapes; second, of a light wooden crib, with fastenings along the lower rail to hold the sheet. This frame folds by two movements into a compact bundle. The accessories are a siphon-shaped piece of hose and a bath thermometer. In using, the sheet is first slipped under the patient, brought up over the pillow, and tucked up alongside of the body. The frame is unfolded and placed down over the patient, resting on the mattress, and surrounding patient and pillow. The edges of the sheet are then drawn up and over the top rail of the crib down to the lower rail, and fastened by its rings. This completes a light and perfect tub, capable of holding 20 gallons of water. It can be emptied by siphon in four minutes (see Fig. 5).* If the ordinary tub is used, and in hospital service this is usually more convenient, the same water, if it remains unsoiled by discharges, as it should, may serve for a dozen baths.

Other Methods of Reducing Temperature.—As contrasted with this, other methods of securing the good effects of hydrotherapy, if not of reducing temperature, seem trifling; yet, as it may be impossible to carry out the Brand treatment, these methods must be considered. Sponging is one of the most usual, and if rightly carried out may be quite efficient. It should be resorted to, as is the bath, when the temperature reaches 102.2° F. (39° C.), and continued fifteen minutes, or until the temperature falls. An important condition of successful

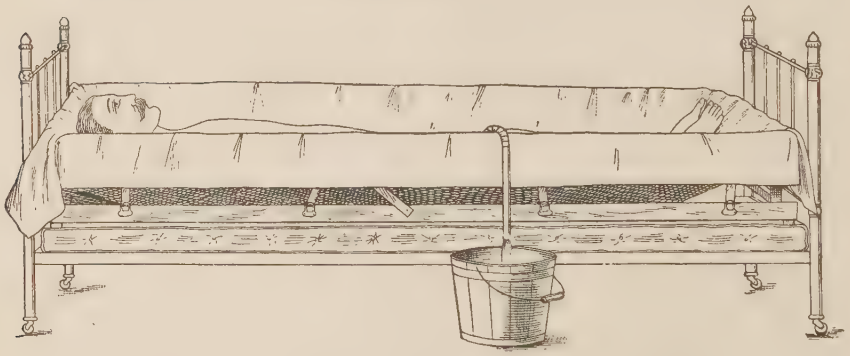
* While printing the above, Mr. S. Clifford Boston, a medical student, has shown me a bath quite as easily managed and more portable than the Burr bath, since the framework is dispensed with and strong iron supports substituted.

sponging is often overlooked. A thin film of water should be left on the surface sponged, as it is the evaporation of this, rather than the temperature of the water, which is effectual in cooling the body. Temperatures that cannot be thus controlled can often be brought down by a partial wet pack, which I have found very efficient: The patient's trunk is enveloped from the axilla to the thighs in a folded sheet, which is kept constantly wet, or as much so as is required to control the temperature, by the continual addition of cold water.

Antipyretics, including antipyrin, antifebrin, phenacetin, and others of the same class, which act by producing copious perspiration, are no substitutes for the baths, for, while they reduce temperature, their effect is but temporary, and their continuous employment too depressing to the patient. Moreover, they are purely antipyretic, and lack the tonic influ-



FRAME OPENED.



BATH-TUB COMPLETED, SHOWING SYPHON FOR DRAWING OFF THE WATER.

FIG. 5.—BURR'S PORTABLE BATH-TUB.

ence to the nervous and muscular system which characterizes the cold tub baths. Quinine, formerly used in massive doses for its antipyretic effect, has been replaced by the more modern methods.

Guaiacol locally applied is undoubtedly an efficient agent for reducing temperature and has a warm advocate in Horace G. McCormick in the treatment of typhoid fever. Some rather alarming symptoms are, however, reported from its use, and it has failed as yet to secure a permanent footing.

The Expectant Symptomatic Treatment.—Where the difficulties in the way of the Brand method are insuperable, I prefer to place the patient in bed on the diet described, combat the temperature by sponging or packs, and for the rest adopt what may be termed the symptomatic expectant method, meeting the symptoms as they arise in accordance with the following :—

(*a*) *Indications for Alcohol and Other Stimulants.*—I prefer to reserve alcohol until called for by signs of waning strength. That it is a remedy of the greatest value I fully admit, but it is also true that mild cases may be carried to a favorable termination without a drop. On the other hand, I favor its liberal use when needed, giving sometimes as much as an ounce (30 c.c.) of whiskey or brandy every hour, though such doses are rarely needed. More frequently a half ounce (15 c. c.) every four or two hours is quite sufficient even where there is considerable adynamia. A low, muttering delirium, feeble, dicrotic pulse, and dry tongue are among the indications which imperatively demand alcohol; a high temperature does not contraindicate it, as an antipyretic effect also follows the use of large doses, and delirium is sometimes calmed by it. Other diffusive stimulants which may be used in conjunction or alternation with alcohol are the aromatic spirit of ammonia and the carbonate of ammonium, while digitalis and strychnine may tide a feeble heart over a period of weakness. From five to ten minims (.333 to .666 gm.) of the tincture of the former, and $\frac{1}{30}$ to $\frac{1}{20}$ (.00216 to .00324 gm.) of the latter may be given as demanded, while their hypodermic use may be availed of.

(*b*) *Treatment of Special Symptoms.*—Methods more directly adapted to control *delirium* are a soothing touch and voice, the bromides, spirit of chloroform, chloral, Hoffman's anodyne. With the cold-bath treatment they are rarely necessary. Occasionally, meningeal symptoms are so violent that leeches may be used to the temples or behind the ears. I have seen an almost magically quieting effect thus produced. Blisters are useless.

Little difficulty is commonly experienced in controlling the *diarrhœa* of typhoid fever. As stated, with the cold-bath treatment very little special treatment is necessary. Simple preparations of opium, either alone or in combination with bismuth or nitrate of silver or acetate of lead, are usually sufficient. Specific action has been claimed for nitrate of silver. I have not been convinced of this, yet it is, in combination with the extract of opium, ¼ gr. (.0162 gm.) of each, my favorite remedy for the diarrhœa. Similar specific effect, more particularly in healing the ulcers, has been claimed for the oil of turpentine. The impression made by the teachings of the late George B. Wood on the profession of the United States as to this effect has not yet been effaced. He held that the dry, leathery tongue so often presented in this disease was the indication for its use. Whether such view was correct or not, few who have used the oil of turpentine have failed to see the coated tongue clear up under its use. Turpentine is also useful as a stimulant. It should be administered in doses of ten minims (.66 gm.) in mucilage of acacia every six or eight hours.

Constipation, especially during convalescence, is not infrequent and should not be too hastily interfered with. If it is necessary to interfere, it should be by simple enema only. Aperients by the mouth in this stage are dangerous, and I am confident I have seen at least one life sacrificed by purgatives thus administered, having been succeeded by perforation, peritonitis, and death.

Hemorrhage from the bowels should be treated by absolute quiet and cold to the abdomen. Food should be reduced to a minimum and

should be of the blandest character as represented by peptonized milk and liquid beef peptonoids. The administration of food may be suspended for some hours without risk. In severe cases the foot of the bed should be raised and a hypodermic injection of ⅛ to ¼ gr. (.008 to .016 gm.) given at once. In such cases where prompt and decisive action is necessary a syringeful of a filtered fluid extract of ergot may be injected hypodermically, and repeated later if necessary in half the dose. In mild cases astringents such as tannic acid or gallic acid and the acetate of lead may be given by the mouth, the former in doses of ten to 15 grains (.666 to one gm.) hourly until some hours have elapsed without a hemorrhage. The acetate of lead should be given in one to three grain (.066 to .194 gm.) doses every three hours combined with extract of opium ¼ grain (.016 gm.).

Tympanitic distention of the abdomen is often a distressing symptom. It is usual to treat it with turpentine in ten-minim (.666 gm.) doses every four to six hours. The rectal tube should be cautiously used if the meteorism is great, and large quantities of gas are sometimes thus disengaged. The quantity of food should also be reduced to a minimum.

The *pain* induced by meteorism or otherwise may be allayed by turpentine stupes or poultices over the abdomen, though sometimes it may be necessary to reinforce the stupes by small doses of opium, or a light, warm poultice may be substituted. Sudden, sharp pain, similar to that produced by tympanitic distention of the bowel, is also caused by *peritonitis*, of which tympany is also a symptom, and the two often occasion many anxious moments to the physician necessarily in doubt as to whether this serious complication may occasion them. If a peritonitis is the result of extension by continuity and not of perforation, a possible condition, recovery may take place. Such recovery is favored by absolute rest of the bowel, best secured by hypodermic injection of morphine ¼ grain (.016 gm.), repeated if necessary. Even such movement as is necessitated by the use of the bed-pan is of questionable propriety. It is much better to permit the discharges to pass into a sheet.

It is claimed that recovery has taken place where peritonitis has been caused by *perforation*. The extreme rarity of such recovery and the less infrequent though still rare recovery after laparotomy suggest that this operation should be considered. Up to the present time there have been 19 per cent. of recoveries after operation. A surgeon should be immediately called, and if collapse is not too great laparotomy should be done. Unfortunately, these are cases in which the administration of ether adds to the existing danger.

For *sleeplessness* the milder soporifics usually answer; ten to 15 grains (.666 to one gm.) of sulphonal generally furnishes the required rest. Chloralamide 30 grains (two gm.), trional 15 to 30 grains (one to two gm.), or chloral 10 to 15 grains (.666 to one gm.), may be used. If these remedies are insufficient, morphine must be used, ¼ of a grain (.016 gm.) being given by the mouth or half as much hypodermically, or more if necessary.

One of the dangers of protracted cases are *bed-sores*. These can generally be averted by scrupulous attention to cleanliness, the thorough drying of the patient after washing, removing thus all traces of urine

or other discharges, and by sponging the patient daily with alcohol or whiskey. Above all, his position in bed should be frequently changed and all inequalities in the bed clothing should be kept smoothed out, while the bed should be kept clear of crumbs and other particles of food. Should a sore appear it must be antiseptically dressed, while the part should be protected from pressure by pads and air-cushions.

The Treatment of Convalescence.—In no disease is watchfulness during convalescence more important. The effect of indiscretion in diet in producing relapse and recrudescence has already been referred to. But there are other dangers during convalescence. It is to be remembered that the complete healing of intestinal ulcers is often delayed after all other symptoms have disappeared except a slight elevation of temperature; that a deep-seated ulcer may thus remain with the thin peritoneum for its floor, rendered weaker by reason of imperfect nutrition. Such a membranous floor is known to have been torn by simply reaching over for a book and to be followed by a fatal peritonitis. These are reasons, too, for putting off the use of solid food until the temperature has maintained the normal for a considerable time, certainly a week. Then the diet should be changed most gradually, first permitting a soft-boiled egg or poached egg in the morning, and awaiting developments. If no fever follows it may be continued daily. The next step is to allow some thoroughly softened milk toast, then a small quantity of well-boiled rice, allowing a suitable interval after each first trial until sure that no harmful results follow. Finally tender meat is allowed, and then soft vegetables one after another.

Emotional disturbance is a well recognized cause of recrudescence, and should be carefully guarded against.

I have already referred to constipation and the importance of correcting it by enemata only.

During convalescence the *hair* is very apt to fall out, but usually returns in a natural way. It is, however, desirable to cut it close, though scarcely necessary to shave the head, as some recommend.

Prophylaxis.—Most important in the management of typhoid fever is the *disinfection of the excreta*, which are the contagium bearers, through the careless handling of which the disease is communicated to others. The same is perhaps true of the vomited matters. Probably the best disinfectant, on account of its cheapness, harmlessness, and effectiveness, is chlorinated lime or bleaching powder, also called chloride of lime, which contains from 25 to 40 per cent. of available chlorine. A solution made in the proportion of four to 100 of water, containing, therefore, at least one per cent. to 1.5 per cent. of chlorine, is sufficiently strong. Some of the solution is placed in the chamber vessel before it is used, and the remainder, in all say a pint, is added afterward. Thorough admixture should be made, and an hour allowed to elapse before the stool is thrown into the privy well or water closet, if disposed of thus. In the country the disinfected stool may be buried. Solution of chlorinated soda, or Labarraque's solution, is a more elegant but no more effective disinfectant. As it contains about two per cent. of chlorine it is nearly equivalent when undiluted to the above solution of chlorinated lime. Chlorinated lime rapidly loses its chlorine, and should be kept in tightly closed vessels.

Carbolic acid in the proportion five parts of the commercial acid to 100 of water is an efficient disinfectant. The same method must be employed in its use and an exposure of twenty minutes to half an hour maintained. Quite as good a disinfectant for intestinal evacuations is milk of lime or ordinary "white wash," composed of lime in solution and in suspension. This should be thoroughly mixed with the evacuations until the mass is distinctly alkaline, and should remain in contact for one or two hours, since it is slower in its action than chlorinated lime or carbolic acid, and much longer exposures are required to destroy the bacillus. It is particularly adapted to the disinfection of privy wells and latrines, into which it may be thrown, freshly prepared in the proportion of one part by weight of recently burned calcium hydrate to eight of water, or about 12 per cent. It is not harmful to water-closet pipes in such quantities as required to disinfect the stools of a single case of typhoid fever.

Sulphate of iron or copper is a good antiseptic and deodorant, but not a true disinfectant,—an antiseptic because it prevents the growth of bacteria without necessarily killing them, as do disinfectants which are antiseptic as well, while an antiseptic may not be a disinfectant.

Most important in the prophylaxis of typhoid fever is drainage. It seems to be now definitely settled that the fever originates in every instance from the ingestion in some way of the typhoid bacillus, commonly in drinking water or milk, or in food contaminated with it, more rarely by inhalation. Hence it is of the greatest importance that the sources of water used in domestic economy should be protected against contamination by discharges containing the specific bacilli, which sometimes find their way into wells and other sources of water supply.

The following directions may be followed with advantage by nurses :—

(1) Since the contagion of typhoid fever resides solely in the stools and vomited matters, it is important, not only that these should be thoroughly and properly disinfected, but also that linen which has been in the least degree soiled by them should be immediately removed and sterilized.

(2) Stools may be disinfected in the following manner : Place in the bed-pan, before using, a small quantity of solution of carbolic acid (one to 20) or chlorinated lime of the same strength. Cover the evacuation completely with another quantity of the disinfectant solution, mix thoroughly, and empty the whole into the water-closet hopper, rinsing out the emptied vessel thoroughly with the disinfectant solution and hot water. Linen may be submerged in carbolic solution one to 60, or in water (bichloride of mercury solution is objectionable because it stains) until it can be boiled. Thorough boiling of linen for half an hour effectually disinfects it.

(3) After the bowels have been moved, the buttocks and anus of the patient should be thoroughly washed with the carbolic solution (one to 60) or bichloride of mercury solution one to 2000, followed by hot water and soap.

(4) Door knobs or parts of a door touched with soiled hands should be washed off with the carbolic solution or bichloride solution, since they are a possible source of infection through the dried fecal matter disseminated from them.

(5) Finally, the nurse should wash her own hands with soap and water and then rinse them in fresh bichloride solution. As it is either from soiled hands or by floating bacteria from dried feces that nurses who acquire typhoid fever become infected, the importance of the last injunction cannot be overestimated.

(6) Where tub-bathing is employed it is possible that the water of the bath which has been used several times may be a source of infection. The nurse should therefore wash her hands after tubbing a patient, and watchful care should be taken not to carry them to the mouth during the bath.

(7) After death or discharge of the patient the mattress, which should be well protected during use by a rubber sheet, should be thoroughly aired. The rubber sheet itself should be wiped with carbolic solution, rinsed in cold water and dried in the open air.

The Specific Treatment of Typhoid Fever by Cultures or Serum.—This has been as yet tentative and the results admit of no definite conclusion. I can only give them as far as obtained. Three sets of observations have been made. E. Fraenkel * and Manchet treated 57 cases by injecting first .5 c. c. of the sterilized cultures of typhoid bacilli grown in thymus bouillon, deep into the muscular tissue in the lateral gluteal region; the next day one c. c. and then each second day one c. c. more. Usually there followed a noticeable rise of temperature and in some cases a more or less pronounced chill. On the third day of treatment there was usually a fall in temperature followed the next day by a still greater fall. The conclusion arrived at was that the course of the fever is materially influenced, being changed from a continued to a more or less remittent character, and that a complete pyrexia occurs in shorter time.

Th. Rumpf † used cultures of the *bacillus pyocyaneus* prepared in a similar way, but in other respects followed Fraenkel. He treated 30 patients, of whom two died, one from pneumonia and one from intestinal hemorrhage. The injections were commonly followed by a rise in the temperature and a subsequent fall with diminished pulse-rate and profuse perspiration. Sometimes a chill and rise of temperature followed the injection. As a rule general improvement in the feeling of the patient and a prompt disappearance of delirium were noted.

Fr. Kraus and H. C. Buswell ‡ treated 12 cases with sterilized pyocyaneus cultures, of which ten recovered and two died. Usually an immediate reduction of temperature occurred lasting for several hours. They noticed also an increase in the number of leucocytes in the blood following the injections, and the same result followed the injection of sterilized pyocyaneus cultures in rabbits. The conclusion reached by them was that this method had no specific curative value.

---

* E. Fraenkel, "*Ueber specifische Behandlung des Abdominal-Typhus.*" *Deut. Med. Wochenschrift*, 1893, s. 41.

† Rumpf, "*Die Behandlung des Typhus abdominalis mit abgetöteten Kulturen des Bacillus pyocyaneus.*" *Deut. Med. Wochenschrift*, 1893, s. 987.

‡ Kraus and H. C. Buswell, "*Ueber die Behandlung des Typhus abdominalis mit abgetöteten Kulturen des Pyocyaneus.*" *Wien. Klin. Wochenschrift*, 1894, s. 511, 595. See also Sternberg, "Immunity and Serum Therapy," New York, 1894, p. 296.

Most recently (1896) R. Pfeiffer* and W. Kolle showed (1) that the *serum* of the convalescent from typhoid contains substances which in very small amounts act as a bactericide and solvent against living typhoid bacilli in the peritoneum of guinea pigs, but that this action is not due to a preformed bactericidal substance but to a reaction induced in the animal by the serum.

(2) That antitoxic substances are not demonstrable in such serum, *i. e.*, it produces of itself no antitoxic effect, but incites in the animal a reaction which is destructive to bacteria.

(3) That normal human serum has a similar action upon the bacilli in the peritoneum of guinea pigs, but differs quantitatively and qualitatively. Quantitatively, 20 to 100 times the dose of ordinary serum is required; qualitatively, it lacks the specific action upon typhoid bacilli which typhoid serum has.

(4) That serum from convalescents can be used to differentiate typhoid from similar bacteria, and its action would make a post-disease diagnosis of typhoid possible.

(5) That its action shows that it is the typhoid bacillus alone that causes true typhoid.

(6) Immunized goat serum contains the bactericidal substance, but cannot be obtained in sufficient strength to be of use as yet.

The **antiseptic treatment** has not a truly rational basis, while the extravagant claims of its advocates discredit their results.

Mountain Fever.

Definition.—A form of fever met in the mountain regions of Western United States, characterized by its moderate temperature, 101 to 103° F. (38.2 to 39.3° C.), a duration of two to four weeks, and generally mild course.

Mountain fever has come to be pretty generally acknowledged as a variety of typhoid fever, modified by the combined factors which go to make up the influence of high altitudes. Certain it is that if a careful study of the cases reported by various observers is made, the clinical picture differs no more from the typical picture of typhoid fever than the abortive forms of typhoid occurring at low altitudes. It must be admitted, however, that this view is not unanimous, and it should be mentioned that surgeon Charles Smart,† of the United States Army, in a careful review of the subject, in 1878, in a paper entitled "On Mountain Fever and Malarious Waters," concluded the disease was typho-malarial fever. Such a view implies a separate specific disease, typho-malarial fever, which is not admitted at the present day by the best authorities. The crucial test is found, however, in the presence of the lesions of typhoid fever in the ileum in at least two of the very few fatal cases reported.‡ So far as I know, the test of bacteriology has not yet been brought to bear in settling the matter. An enlarged spleen is

* *Zeitschrift für Hygiene und Infections-Krankheiten*, Band XXI, zweites Heft, 1896.

† Amer. Jour. Med. Sci., January, 1878.

‡ One is reported by Surgeon Hoff, U. S. Army, Amer. Jour. Med. Sci., January, 1880; the specimen from another is in the United States Army Medical Museum, Washington, D. C.

also found. Epistaxis is not rare. So far as I am aware, however, no spots have been reported except a "doubtful *tache rouge*" by Roland G. Curtin * in one of four cases seen by him in 1868 in Wyoming Territory. Diarrhœa has been noted, but there is a tendency rather to constipation, not infrequently the case in typhoid fever. Tympanites also occurs. The other symptoms are those incident to all fevers, such as debility, headache, and frequent pulse. I do not know that the best of bacteriology has as yet been brought to bear to settle the matter.

Doubtless many imperfectly studied instances of other forms of disease are classed by indifferent observers as mountain fever, as in a case mentioned by Curtin.

Another condition incident to mountain residence, commonly called *mountain sickness*, should not be confounded with mountain fever. In it there is dyspnœa, frequent pulse, dizziness, and bleeding at the nose; also great prostration on exertion, and sometimes slight elevation of temperature.

Treatment.—The treatment of mountain fever, mainly symptomatic and roborant, would be, so far as any special measures are needed, that of typhoid fever.

TYPHUS FEVER.

SYNONYMS.—*Typhus exanthematicus; Petechial Fever; Pestilential or Putrid Fever; Ship Fever; Jail Fever; Camp Fever.*

Definition.—An acute, highly contagious fever, favored by closely crowding human beings, especially characterized by sudden onset of high fever, by a petechial eruption, typhoid symptoms, and short duration as compared with typhoid fever, with sudden termination at the end of the second week.

Historical.—It is commonly conceded that the *plague* of Athens, so graphically described by Thucydides (B. C. 470 to 400), was the same as the typhus fever of to-day, though it has also been held identical with the Oriental or bubonic plague, which, like typhus, is growing very rare. It is also possible that what is so often mentioned in the Scriptures as pestilence may have been typhus, although this may also have been the Oriental plague. The same is true of numerous epidemics which prevailed during the first fifteen hundred years of the Christian era, many of which were undoubtedly typhus, especially in Spain and Italy. Among the names were *La Pourpre* in French, *Tabardiglio* in Spanish, *Petecchie* in Italian, and *Fleckfieber* in German, all of which meant "spotted." It was called pestilential, putrid, malignant, petechial, and "jayl" fever in England, and also "the plague" until 1760, when the name typhus was given it by Sauvages. The history of its separation from typhoid was given under that disease.

Etiology.—Though of acknowledged infectious nature, no organism has as yet been isolated which can be held responsible for typhus

* "Rocky Mountain Fever." Reprint from the New York Med. Jour., January 8, 1887.

fever.* Fostered by close crowding, filth, and famine, it each year becomes more infrequent as the conditions favoring it are eliminated, and there is reason to believe it will ultimately be stamped out. Ireland has been its home for centuries, but filthy and crowded sections and the almshouses of large cities have at different times furnished seats for its lodgement. My own experience with the disease is limited to two mild epidemics in the Philadelphia Hospital in 1866 and 1883, and another quite serious in the Camden County (New Jersey) Almshouse in the winter of 1880–81. Quite a serious epidemic prevailed in New York city in 1881–82, and a milder one in 1892–93. Sporadic cases rarely occur, but its spontaneous origin is scarcely possible, though such possibility was admitted by Murchison, whose judgment on fevers was at one time regarded as almost infallible.

Typhus fever is eminently contagious, and cases should be promptly isolated. Nurses and others in constant attendance upon typhus patients are more liable to be attacked than those who, like the physician, merely visit them daily, although perhaps no disease in the past has included among its victims so many medical men. It is not known precisely what the contagium bearer is. It may be all the exhalations and discharges from the body, but it is not especially the bowel discharges.

Morbid Anatomy.—As to the morbid anatomy of this disease, there is really nothing distinctive. Rigor mortis is apt to be delayed. The petechial eruption remains after death, and gangrenous bedsores may be found on the body. The most constant lesion is moderate enlargement of the spleen, and in this enlargement the liver and kidneys may share, and their cells be the seat of cloudy swelling due to fever heat. Indeed, all the tissues, including the heart muscle, may be granular from this cause. The splenic enlargement is mainly due to vascular engorgement, but there may also be some hyperplasia of lymph cells. The lymph follicles of the intestine may be enlarged from the same cause, but there is no ulceration of these or of Peyer's patches. The blood is dark and liquid. Hypostatic congestion of the lungs is very frequently found; likewise bronchial catarrh. The permanence of the eruption after death is in strong contrast with that of typhoid fever, which disappears after death.

Symptoms.—The period of incubation is usually about twelve days. It may be less. There is seldom any prodrome, the invasion being sudden, ushered in by a chill or chills followed by headache and great muscular pain, especially in the back, and by high fever, the temperature rapidly rising to 103°, 104°, 105°, and 106° F. (39.4°, 40°, 40.5°, 41.1° C.) without any of the tidal-wave rise characteristic of typhoid fever. The pulse is at first full and strong but soon weakens and becomes frequent, 120 and more. There is extreme debility. Almost characteristic is the red, congested conjunctiva, the dusky face and dull expression, and low, muttering delirium, which contrasts strongly with the sometimes active delirium of typhoid. The tongue is early coated and becomes rapidly dry. The bowels are constipated.

*For a summary of the observations thus far made on the "Micro-Organisms in Typhus Fever," see a paper with this title by J. B. Byron and Egbert Le Fevre, in Vol. II, "Researches of the Loomis Laboratory," New York, 1892, p. 130.

On the third to the fifth day the eruption presents itself and is of two kinds. The petechial, or more characteristic, is at first not unlike that of typhoid fever, but it is darker in hue and it disappears less readily on pressure. A little later it is barely influenced and later does not respond at all to pressure. It has become hemorrhagic, petechial,—the blood is without the vessels. There may be spots exhibiting each one of these stages. This eruption is also more scattered than that of typhoid fever, appearing all over the body, while that of typhoid is limited to the chest and belly. In addition to the petechial eruption there is also a peculiar dark mottling of the skin, an alternation of purple blotches with others of a light hue, generally capable of being influenced by pressure, but these blotches, too, may become blood extravasations.

With the beginning of the second week all of the symptoms deepen, the tongue becomes dry, fissured, and leathery, sordes collects on the teeth, stupor deepens, there is subsultus and nystagmus, *coma vigil*,—the patient is unconscious but the eyes are wide open,—picking at the bedclothes. The breathing becomes more rapid, the pulse weaker, scarcely appreciable, and the patient may die of adynamia. Or at the end of the second week a crisis occurs, he falls asleep, the temperature declines as rapidly as it rose, and often after a long sleep the patient wakes up refreshed and with a clear head. Convalescence now progresses, and although it may be slow, relapses rarely occur.

A few symptoms require special allusion: First, the *fever*. The skin is burning hot and the thermometer rises to 106° F. (41.1° C.), and even 108° F. (42.2° C.) and 109° F. (42.7° C.) toward a fatal termination. It is the *calor mordax*. There is always *hypostatic congestion of the lungs*, and along with this a good deal of *bronchial catarrh* and *cough*. Such catarrh may pass into a broncho-pneumonia, which may terminate in gangrene of the lungs.

The *urine* is concentrated, as in all high fevers, and urea and uric acid are relatively increased. *Albuminuria* is also common, but there is not usually any organic change in the kidney beyond the cloudy swelling referred to. *Retention of urine* on account of the mental hebetude may occur and should be guarded against by frequent examination and catheterization. *Bed-sores* are common and there may even be gangrene of the extremities.

Instances of the ambulatory form of typhus fever are much more rare than of typhoid, but they are occasionally met with.

Diagnosis.—How does typhus fever differ from typhoid fever? I have already referred to the differences in the eruption in the two diseases. But the temperature of typhus fever is quite as characteristic as that of typhoid fever. In the latter disease we have the peculiar tidal-wave course described. In typhus fever in the first place the average maximum temperature is higher; for a while a temperature of 106° F. (41.1° C.) is not uncommon in typhus fever; 105° F. (40.5° C.) in typhoid is quite high. The temperature in typhus quickly reaches the maximum, usually from the third to the fifth day, continues with light remissions until the twelfth or fourteenth, then there is a sudden decline. The ascent is steady and continuous, and only marked

by slight morning remissions, while in typhoid fever the morning remissions are decided. The pulse, during the first three days, is usually about 100; after that it becomes more frequent and feeble, running up to 120 or higher, until the drop in temperature, when there is a corresponding fall in the rate of the pulse. It is seldom dicrotic, as in typhoid fever. Typhus fever more frequently begins with a chill than does typhoid; the important symptoms, including the eruption, appear earlier. In isolated cases, however, there may be difficulty in diagnosis.

Malignant measles and cerebro-spinal meningitis are the only remaining diseases for which typhus fever may be mistaken. Some similarity is found in the early stage of malignant small-pox. Bubonic plague has been confounded with typhus, but it seems to have been a very different disease, resembling typhus only in its fatality. The eruption of malignant measles is not unlike that of typhus fever, and it appears about the same time, but in measles it appears first in the face. The extreme adynamia and the typhoid symptoms are very similar. There is bronchitis in both, but the coryza and acute nasal catarrh are not found in typhus, while concurrent with the case of malignant measles are others of a milder and more typical nature. The latter fact also aids the diagnosis in variola, where, too, in the malignant form, the hemorrhagic tendency is more marked and occurs early in the disease.

Cerebro-spinal meningitis has often been mistaken for typhus, and in the early stage the suddenness of onset, the eruption, and the nervous symptoms are all calculated to mislead. One has to wait but a few days, however, before the course of the two diseases diverge. Bubonic plague as described to us, for our knowledge is historical, is characterized by the same suddenness of onset, the chill, high fever, and prostration, as is typhus fever; but the eruption appears earlier, and quickly becomes carbuncular, while the course of the disease is much shorter.

Prognosis.—The mortality of typhus is high, but different epidemics vary in this respect. During the epidemic at the Camden County Almshouse (1880–81) referred to, 103 of the officers and inmates were attacked. Of this number 23 died, giving a mortality a little over 32 per cent. I might add that of the officers of the institution, seven, including an attending physician, the steward, the matron, the assistant matron, and two nurses, together with the builder of a new hospital building, were attacked, and all died. In some epidemics the mortality is even greater, reaching 50 per cent., but it is commonly put down at from 12 to 20 per cent. The disease attacks either sex at any age. One of the modes of death is by acute fatty degeneration of the heart, and the peculiar dusky complexion sometimes seen may be due to the inability of a weak, fatty heart to propel the blood through the capillaries. Sudden death is not unusual. It is more than likely that with the improved nursing and hygiene of the present day the mortality of typhus would be less.

Treatment.—Whenever possible, typhus fever should be treated in the open air, in tents, as the safety of attendants as well as recovery of patients is favored thereby. There is no reason why hydrotherapy should not be as serviceable in typhus as in typhoid, but it is absolutely necessary that free stimulation should be associated with any treatment.

We know that the greatest danger lies in the asthenia, which can only be met by stimulants. If there is one disease in which the free use of alcohol is indicated more than in another, it is typhus fever. The quantity, of course, must be governed by the condition of the patient. In some cases it may be necessary to give an ounce (30 c. c.) every hour. Quinine is also strongly indicated, as are also digitalis and strychnine as heart strengtheners. When the temperature becomes high, if the cold bath is not used, sponging of the body in the way described under typhoid fever may be substituted. Other symptoms should be treated as they arise. Specific antiseptic treatment has proved to be without peculiar advantage.

The same objection exists to phenacetin and antifebrin as in typhoid; that is, they dare not be relied upon as an exclusive means of reducing temperature, but they may be used as adjuvants. After the crisis, which, as has been said, is strikingly well marked in this disease, it is simply necessary to treat symptoms as they arise. The accompanying bronchitis is treated, if it requires treatment, like any other bronchitis, but the ammonium salts are especially indicated on account of their stimulating qualities, while the aromatic spirit of ammonia is an especially convenient preparation for these purposes. Alcohol and ammonia may be given hypodermically to tide over emergencies.

The patient should be nourished as in typhoid fever, by nutritious liquids, including milk, milk punches, egg-nog, and nutritious broths.

RELAPSING FEVER.

SYNONYMS.—*Febris recurrens; Famine Fever; Seven-day Fever; Typhus icterodes.*

Definition.—Relapsing fever is an acute infectious disease characterized by two or more febrile relapses separated by periods of total remission, and caused by the inoculation and multiplication of the spirochæta of Obermeier.

Historical.—Like typhus and typhoid fevers, relapsing fever is not a new malady, for a disease corresponding very closely to it was described by Hippocrates as prevailing 2000 years ago in the island of Thasus, off the coast of Thrace. Typhus and relapsing fevers often prevailed together, and many of the older reports of typhus with relapses doubtless referred to relapsing fever. Strother, in 1729, speaks of frequent relapses; also Lind, in 1763. John Rutty, however, in 1770, gave the first clear description of relapsing fever as it prevailed in Dublin in 1739, 1741, 1745, 1748. After this many epidemics of what is evidently relapsing fever were recorded until 1817. It was still regarded as a modification of typhus, even in 1817–1819, and, according to Christison, "there was a very general impression that the relapsing fever could produce the common typhus." After 1819 the disease almost disappeared until 1826, when another epidemic of both typhus and relapsing fevers broke out. Now for the first time a distinction was drawn between the two fevers, especially by Dr. O'Brien, who published an account of the epidemic as it appeared in Dublin. Numerous epi-

demics appeared from time to time since that date in Great Britain and Ireland and on the Continent of Europe, but it has been growing less as cleanliness and hygiene improved. It prevails, also, as might be expected on account of the defective sanitary conditions, in India and Eastern Europe. It made its first appearance in America in 1844 in an emigrant ship from Liverpool to Philadelphia, and was described by Dr. Meredith Clymer. In September, 1869, it again visited Philadelphia, and New York in November, and a somewhat extensive epidemic prevailed in the former city, in which I had some experience with it.

Etiology.—That the specific cause of relapsing fever is a bacterium of the group spirobacteria, genus spirochæta, is now generally acknowledged. First discovered by Obermeier in the blood of cases, it is known by his name as the *spirochæta Obermeieri.* It is a narrow spiral about .025 to .05 mm. ($\frac{1}{1000}$ to $\frac{1}{500}$ of an inch) in length, that is, its length is three to six times the width of a red blood disc. It is found floating among the blood discs during the fever. Before the crisis and in the intervals the organism is not found, but small, glistening spherules, said to be its spores, take its place. Confirmation of the contagious nature of the disease is found in the fact that it has been communicated from one human being to another by inoculation of blood, and to monkeys in the same way. It may be supposed that the organism is given off in the breath or from the skin. The operation of the cause is undoubtedly favored by overcrowding, by filth, and by destitution. Yet the disease is not confined to the poorly fed. This was especially proven in the Philadelphia epidemic of 1869–70, where a considerable number of fairly well-to-do persons were affected, although they always resided in crowded districts. Neither age, sex, nationality, nor season are factors in its causation.

Morbid Anatomy.—There is no essential morbid anatomy, and such as is found corresponds with that of typhus. Most conspicuous is enlargement of the spleen.

Symptoms.—The *period of incubation* varies greatly, so that it is put down at from two to fourteen days. According to Murchison there may actually be no interval between exposure and the invasion. The latter is sudden by a *chill, fever, intense pain* in the back and limbs, with *dizziness.* This abrupt invasion is a distinctive feature, and in perhaps none of the contagious diseases is it as a rule so marked. Exceptionally only is there a short period of *malaise* with *loss of appetite.* On invasion the temperature rises rapidly and quickly reaches 104° F. (40° C.). The patient cannot retain his feet and promptly takes to his bed, feeling very sick, rather than profoundly weak; there may be *nausea* and *vomiting* and even *convulsions* in the young; the pulse rises rapidly, more rapidly than in typhus, reaching 140 on the second day, and later 150 and 160. The patient may be *delirious*, but the typhoid symptoms are not usually as profound as in typhus, and the tongue remains moist. *Jaundice* appears in a certain number of cases on the third or fourth day, usually in one out of every twelve cases, occasionally as often as one in every four or five. The temperature during the paroxysm fluctuates slightly, being higher in the evening. *Sweating* and *sudamina* are often present, and occasionally *petechiæ*, but there is no characteristic eruption. Rarely, Murchison says, in eight out of six hundred cases, a

roseolous rash appears in small spots, or it may be a mottling like that of typhus, which, however, always disappears on pressure, and disappears altogether in three or four days, differing in these respects from the similar eruption of typhus. *Herpes* may be present. There is occasionally *abdominal tenderness* in the epigastric or iliac region and the *enlarged spleen* may be easily detected, but there are no active intestinal symptoms. The *liver* is also *slightly enlarged*, extending lower than in health.

The *spirillum* is to be found in the blood and should always be looked for. It may readily be detected with a power of 500 diameters without any special preparation of the blood, care being simply taken to secure a thin film.

If the invasion of relapsing fever is sudden, its termination is no less so. It is by *crisis*, beginning usually with sweating. After five or six days of unabated fever, sweating sets in, which soon becomes profuse, the temperature falls rapidly to normal or even subnormal, the various discomforts fade away, and in the course of a few hours the patient is apparently well. Rarely the crisis may be ushered in by a diarrhœa, an epistaxis, or the appearance of menstruation.

The crisis does not always take place at the same stage of the disease. It may occur as early as the third day or not until the tenth, and even the fifteenth, but most commonly on the seventh. While the crisis is ordinarily followed by some relaxation and faintness, there soon ensues a rapid recovery of natural and healthful feeling. Occasionally, however, the depression is greater and a feeling as of collapse occurs, especially in delicate or elderly persons.

Again, in a week from the crisis, generally on the fourteenth day from the primary chill, another occurs, or a series of them, with *fever*, and the paroxysm repeats itself, to be again succeeded by a crisis at a somewhat shorter interval. There may be a third or even fourth and fifth paroxysm; more commonly they are limited to two, or at most three. Each succeeding attack is shorter than the previous one. Occasionally there is no relapse, the disease terminating with the first crisis. Convalescence, usually rapid, is sometimes prolonged, and the duration of the entire illness may be put down at from eighteen to ninety days, and the patient rarely returns to work within six weeks. One attack does not secure immunity from another.

Among the *complications* may be mentioned bronchitis, pneumonia, nephritis, and hæmaturia. The spleen may enlarge until it ruptures. It may attain a weight of four and a half pounds (ten kilos), and may be the seat of infarcts. Albuminuria occurs as in other fevers characterized by high temperatures. Pregnant women usually abort in the relapse, and the child if not still-born survives but a few hours. Post-febrile paralysis may occur and troublesome ophthalmia succeeds in some epidemics.

Diagnosis.—In its early stages relapsing fever is not unlike typhus. In suddenness of onset, rapid rise of temperature, habitat, and victims the resemblance is close. The readiness with which a patient takes his bed is characteristic of each, but in relapsing fever the adynamia is not as great as in typhus, and it is rather because of a dizziness that he cannot keep about. The crisis cuts short all doubt on this point of confu-

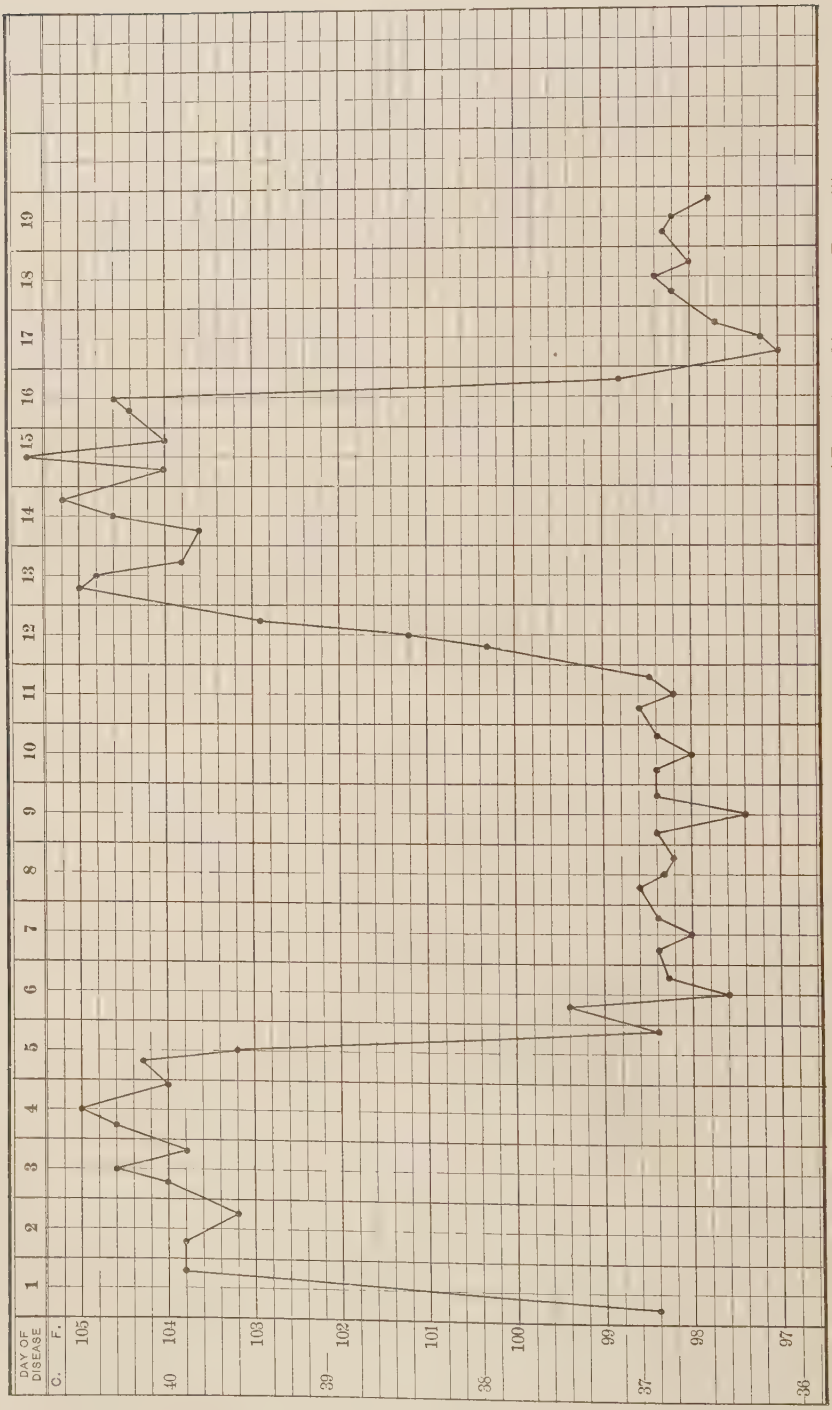


FIG. 6.—TEMPERATURE CHART OF RELAPSING FEVER, SHOWING RELAPSES.—(*From Murchison on "Fevers."*)

sion with typhus. In the intense muscular pains, especially in the back, relapsing fever resembles small-pox, but the eruption in the latter disease sets doubt at rest.

Malarial fever may be suggested by the relapse, but the presence of an organism in the blood of each of these affections widely different in appearance permits the settlement of such confusion by the microscope. The prevalence of an epidemic is, of course, of great assistance in the diagnosis between relapsing fever and any of the diseases with which it may be confounded.

Prognosis.—The prognosis of relapsing fever is not unfavorable. The higher mortality reported in some of the earlier epidemics in Great Britain and Ireland was doubtless due to an admixture of typhus. An average for several years in a number of cities in Great Britain and Ireland, according to Murchison, has been 4.3 per cent.; in the epidemic at Bombay in 1877–78, Vandyke Carter estimated the mortality at 18.02 per cent., while in the Philadelphia epidemic the studies of William Pepper and Edward Rhoads found it 14 per cent. There are some accidents which have been already alluded to, which are responsible for a few deaths. Thus, the spleen has ruptured from extreme congestion. Pneumonia sometimes causes a fatal termination. It has been said that the crisis sometimes terminates in collapse with its characteristic clammy coldness, pulselessness, unconsciousness, and fatal end. A fatal nephritis occasionally complicates the disease, death being preceded by uræmic convulsions. Certain cases associated with jaundice, called by Griesinger "bilious typhoid," are often fatal. Some striking cases of this kind were noted by Pepper at the Philadelphia Hospital in the epidemic of 1869–70.

Treatment.—The febrile paroxysm demands much the same treatment as typhus,—careful nursing, sponging or cool bathing, nutritious, easily assimilable food, and stimulation, although the latter is less important than in typhus. No drug has the power to prevent the recurrence of the relapse, although quinine is indicated, and, as in other adynamic fevers, is useful as a roborant. It is reasonable to expect that phenacetin, antifebrin, or antipyrin will relieve the muscular pains. Should they not suffice, morphine, hypodermically, can be relied upon.

THE MALARIAL FEVERS.

SYNONYMS.—*Ague; Fever and Ague; Chills and Fever; Marsh Fever; Swamp Fever; Paludal Fever; Miasmatic Fever; Intermittent, Remittent, and Pernicious Remittent Fever; Bilious Fever; Æstivo-autumnal Fever.*

Definition.—An infectious fever of intermittent or remittent type caused by a protozoon known as the plasmodium or hæmatozoon of malaria. The term "malaria," meaning, in the Italian, bad air, was originally applied to the supposed specific cause of the fever, but it is also used to express the consequences of such cause.

Varieties of Malarial Fever.—The malarial fevers are *intermittent* or *remittent*. The former is characterized by paroxysms of fever,

between which there are total intermissions. In the remittent form there are remissions or abatements in the fever, but not total intermissions. The remittent fevers exhibit much less regularity than the intermittent fevers, even in their remissions, and in consequence of their prevalence in the later summer and fall have recently among other irregular types been included under the head *æstivo-autumnal.* This term embraces also all the malignant types, which are rarely seen in the spring months. At the same time the term "irregular" malarial fevers would be quite as expressive and perhaps more accurate.

The paroxysms of fever may come on daily at the same hour, when they are called *quotidian;* they may occur every other day, when they are known as *tertian;* or they may occur every third day, that is, skip two days, when they are called *quartan.* More rarely occur quintan, sextan, septan, and octan fevers, with intervals of four, five, six, and seven days, respectively. It will be noted that in naming these periods the day of the paroxysm and that of the following paroxysm are both counted. The "*double tertian*" is a fever in which paroxysms occur each day but at different hours, the hours on alternate days corresponding with each other. In these cases the alternate paroxysms may also be of different intensities. In like manner there may be double quartans and even double quotidians.

Although the paroxysms in true intermittent fever commonly occur at the same hour, they may happen a little earlier each day, when they are named "anticipating," or they may occur later each day, when they are called "retarding." The former is apt to occur when the disease is becoming more severe, the latter when it is abating. The paroxysm varies in length in the different varieties. In the quotidian form it lasts from ten to twelve hours, in the tertian six to eight hours, and in the quartan four to six hours.

Etiology.—The malarial fevers are to-day believed to be caused by a protozoon known as the *plasmodium malariæ, hæmatozoon or hæmatomonas malariæ.* These fevers have ever been a field which seemed to promise reward to the seekers after such a cause. In the days of Hippocrates, B. C. 460–357, it was recognized that marshes breed malaria. John K. Mitchell,* an eminent American physician, was, however, the first to suggest, in 1849, in a scientific manner, the parasitic origin of malarial fever. In 1859 John K. Barnes,† of the U. S. Army, also called attention to such a mode of origin. Massy, Basa and Wiener, Polk, Holden, Salisbury, and others, all suggested a fungus origin of malaria on more or less unstable foundations. It was not until 1879 that Ed. Klebs and C. Tommasi-Crudeli succeeded in isolating a germ which they called *bacillus malariæ,* from the low-lying atmosphere over marshes and from the soil itself, the inoculation of which into rabbits, they alleged, produced malarial paroxysms with enlargement of the spleen and pigmentation. No permanent impression was, however, made by this announcement. A. Laveran followed the very next year, 1880, with the discovery of the plas-

* "On the Cryptogamous Origin of Malarious and Epidemic Fevers," Philadelphia, 1849.
† U. S. Army Reports, 1859, p. 163.

FIG. 7.—ILLUSTRATING DIFFERENT FORMS OF THE MALARIAL ORGANISM WITH THEIR STAGES OF DEVELOPMENT.

A_1, A_2, A_3, A_4. Sporulation stage. B_1, B_2. Sporules separating. C_1, C_2. Free sporules. D_1, D_2. Epi-corpuscular forms. E_1, E_2, E_3, E_4. Intra-corpuscular forms. F_1, F_2. The large extra-corpuscular body. G_1, G_2. The flagellate forms. H_1, H_2, H_3, H_4, H_5, H_6. The crescent-shaped parasite.

modium referred to, which he announced to the Paris Academy of Medicine in 1881 and 1882. His researches were made in Algiers, and his results were confirmed by Richard in 1882. E. Marchiafava and A. Celli published, in 1885, their observations on the same organism in the blood of malarial patients in Rome. These observers first insisted upon the amœboid property of the intra-cellular form. From this time our knowledge has been enormously extended by many observers, among whom may be especially mentioned Vandyke Carter (1887) and Patrick Manson (1893–96) in England, Golgi (1885–92), Canalet (1893), and many others in Italy, and in this country A. C. Abbott, W. T. Councilman (1885), William Osler (1886), Walter James (1888), George Dock (1889–92), and William Sydney Thayer and John Hewetson as authors of a masterly monograph in 1895.

The malarial organism * is best studied as follows :—

A drop of blood is taken from the finger of a case of ordinary tertian intermittent fever during the chill, or an hour or two previous, while the temperature is gradually rising, and suitably spread on a slide. On careful examination by the microscope there will probably be found a number of pale, mulberry-like bodies analogous to those in Fig. 7, *a*, *a*, made up of from 12 to 20 segments, massed about a clump of black pigment granules. Careful focusing will show that this body lies within a red blood corpuscle, the delicate walls and pale substance of which surround it. If now a little solution of gentian, violet, or fuchsin be added to the preparation, the stain will impart itself to each of the little segments, differentiating a deep tinted central nucleolus and more lightly stained surrounding protoplasm. In the same slide may be seen other similar bodies, *b*, *b*, unenclosed in the ring of hæmoglobin, loosely arranged, apparently falling apart; also small, pale spherules, *c*, floating alone in the liquor sanguinis, apparently derived from the same source.

Careful searching may also discover certain red corpuscles, *d*, *d*, on which may be found minute pale spots, epi-corpuscular, which exhibit amœboid movement. These also take a stain which develops a central nucleolus and lighter surrounding area, deepening in color at its periphery. The colorless intermediate area is usually interpreted as a bladder-like nucleus, the deeper-stained dot at one side representing the nucleolus. The nucleolus is not apparent in the unstained specimen. Sometimes there is more than one disc-like body. A few hours later, after the chill, none of the bodies described are visible, but within the corpuscles are seen actively moving amœboid bodies, *e*, *e*, of considerable size, constantly changing shape and sending out pseudopodia into the substance of the blood corpuscle. These intra-corpuscular bodies stain with the same differentiation as the smaller disc-like bodies. Many of these contain one or more dark granules in active motion.

This movement of the granules in the intra-corpuscular body is

* These organisms are readily seen with a power of 500 to 600 diameters, but are best studied when magnified 1000 times, say, by an oil immersion $\frac{1}{12}$th. The finger-end should be thoroughly cleaned before taking the drop of blood, which should be placed thinly on a cover glass without reagent. If the study is to be prolonged the edge should be sealed with paraffin or ring of oil. Preliminary studies may be usefully made on the *drepanidium ranarum*, a similar parasite in the red corpuscle of a frog.

regarded by some as Brunonian, by others as due to the moving protoplasm which carries them along. Staining of the intra-corpuscular bodies produces the same differentiation as in the smaller bodies, but the nucleolus is large and less distinct. Still later, toward the next paroxysm, the pale body fills the entire corpuscle, its amœboid movement ceases, while the pigment granules are more numerous and stationary. The protoplasmic mass containing numerous granules now occupies the entire area of the corpuscle, and has lost its nucleus and nucleolus. The pigment now tends to mass itself into clumps or radiating lines, and just before or at the time of the chill the picture first described, of rosettes or loosely attached and free spherules, is seen. The same cycle of successive steps is kept up from paroxysm to paroxysm.

The conclusion reached by all observers is that the large intra-corpuscular body is the mature parasite ready for sporulation, and the mulberry mass presents the sporules perfectly formed, which a few seconds later become free spherical spores. These attach themselves to the red discs, penetrate them, and grow at the expense of the hæmoglobin, leaving the black granular residue as excrementitious substance.

The time required to attain the perfect growth from the free sporule to the last stage of sporulation varies in the different varieties of malarial fever, in each of which certain groups, represented perhaps by myriads of corpuscles, pass through the same stages at the same time, and the sporulation of such a group of parasites is always followed by the malarial paroxysm. This is probably due to some toxic substance developed at the time of sporulation. Thus with the quotidian type sporulation takes place every day, with the tertian type every other day, the quartan type every seventy-two hours, and so on. If, however, two groups ripen at different hours, we have the double forms, be it double tertian or double quartan. The cycle of existence of the æstivo-autumnal type has an undetermined duration, and probably varies from twenty-four to forty-eight hours. The large granule-holding organisms may be found at any stage, because of the different period of ripening of the different groups. Such sporadic ripening of different groups would explain the irregularity of the æstivo-autumnal forms of fever.

With the act of sporulation immense numbers of pigment granules are let loose into the blood, and as a consequence of this the presence of pigment in the blood and viscera is one of the most characteristic features of malaria.

Attempts are made further to differentiate the parasite of the different types of fever by its dimensions, rapidity of amœboid movement, size and number of pigment granules, number of segments in sporulation, etc. Thus the full-grown *parasite of tertian fever* is about as large as a normal red blood corpuscle. In the sporule stage the segments number from 15 to 20, or even more. The *parasite of quartan fever* is very similar, but it is smaller; its amœboid movements are slower and the pigment granules coarser, darker, and less active in motion. The red corpuscle embracing it shrinks about the parasite and assumes a deeper greenish hue, instead of expanding and losing its color. The sporulation segments are fewer, only five or six in number, and arranged with great regularity about the central pigment (Fig. 7). The drawings in the

upper part of the plate are from a case of quartan fever met in the fall of 1895 in the wards of the Hospital of the University of Pennsylvania. Those in the lower portion are selected.

The *parasite of the æstivo-autumnal fever* is still smaller, often less than half the size of a red blood corpuscle at its full growth. Only the early stages of its development, represented by small hyaline bodies, often with one or two pigment granules, are found in the peripheral circulation, the later stage of development being seen in the blood of certain internal organs, such as the spleen and bone marrow. The fewness of the pigment granules is characteristic. The corpuscles containing the parasite are also apt to become shrunken, crenated, and brassy in color. After the process has lasted a week or more, the larger crescentic ovoid and round bodies to be next described make their appearance and are characteristic of this form of fever.

It remains to describe three forms of the parasite, the precise significance and origin of the last two of which are not yet definitely settled. These are the free *extra-corpuscular* body, the *flagellate*, and the *crescentic* forms.

The large *extra-corpuscular* body, *f*, *f*, which presents otherwise the same pigmentation and other features of the intra-corpuscular body, is the latter escaped from the corpuscle, and the event of its escape may sometimes be observed while studying blood taken about the time most of the intra-corpuscular bodies have disappeared by sporulation. Such escape is not, however, supposed to take place in the living blood in the vessels, since any number of preparations, dried or "fixed," immediately after the removal of the blood from the body may be examined without finding a single one.

The same is true of the *flagellate organism*, *g*, *g*, which may come into view on the slide some fifteen or twenty minutes after the blood is mounted, but is never seen on slides "fixed" immediately after the blood is drawn. As seen, however, it is a very interesting object, the tentacular-like prolongations lashing about the central mass and agitating the surrounding corpuscles in a seemingly violent manner, throwing the latter and its own melanin particles into a state of extreme commotion. Sometimes portions of these tentacles break loose and float away in the blood plasma. They are more frequently found in the blood of internal organs, especially the spleen, as first shown by W. T. Councilman.

The *crescent-shaped* parasite (plate, h_1, h_2, h_3, h_4, h_5, h_6) is also a striking object far less frequently met, at least in the latitude of Philadelphia, than the ordinary pigmented form. Unlike the flagellate form, it is apparently a constituent of living blood, and, according to the recent studies of Thayer and Hewetson, as well as the earlier ones of Marchiafava and Celli, appears in most of the cases of æstivo-autumnal fever after a certain time, generally during the second and third week, and not in the cycle of development of either tertian or quartan fevers. The crescent develops in the interior of the red corpuscle from the small hyaline forms, which gradually increase in size, lose their amœboid movement, and assume a crescentic shape, while pigment granules collect in a group at the centre. The corpuscle itself gradually becomes decolorized and ultimately totally destroyed, though for some time a delicate line can be seen run-

ning between the horns of the crescent, a shell, as it were, of the corpuscle in which the parasite is developed. The crescents in turn change into elliptic, ovoid, and finally round forms. When the round form is assumed, the pigment starts into active motion, and sporulation may take place. Such sporulation may also, according to Canalis and Patrick Manson, be by flagellation, though the latter holds that while crescents are found on wet slides immediately after removal from the body, neither ellipsoids, round forms, or flagellate shapes exist in the blood before its withdrawal. He believes that the crescent is intended to carry on the life of the species out of the body, and that the flagellated body is the first phase of the extra-corporeal plasmodium, which Manson regards as a parasite of the mosquito, and that, therefore, malaria is a disease of insects. While with the early study of the subject many observers were inclined, and some still are inclined, to look upon the crescent and flagellate forms as degenerate, the tendency of later studies is to regard them as developmental.

The most important facts with regard to the crescents are these :—

(1) They are associated especially with the irregular and severe forms of fever known as æstivo-autumnal.

(2) They occur late in the attack, generally during the second and third weeks.

(3) On the other hand, their presence in the blood is often unassociated with fever.

(4) They are a very resistent form, with a longer cycle of development than the smaller variety.

(5) In many instances where they have been previously seen in the afebrile period, relapses have occurred, though in such relapses the crescents are associated with small hyaline and amœboid forms.

(6) They may change into round bodies from which flagellation may be observed. Thayer and Hewetson say, however, that they have never seen sporuloid forms which they believe to have developed from crescents. They believe, however, that they are not degenerate, though their true nature is not determined.

Favoring Causes and Geographical Distribution of the Malarial Fevers.—Such is the belief as to the immediate cause of malarial fever at the present day. As yet we do not know how this organism originates or how it enters the system. We do know, however, what conditions favor malarial fever, and these, notwithstanding exceptions, are hot climates and hot seasons plus decomposing vegetable matter, and low river banks frequently covered and uncovered with water and exposed to the sun. Wherever these conditions occur malaria is rife. Especially are they found in the southern borders of the north temperate zone, as in our Southern United States, Southern Italy, and along the lower Danube; the northern border of the south temperate zone, in the tropics, as Central America, the West Indies, Central Africa, and Southern Asia. A freshly upturned soil may furnish, under a sufficiently high temperature, a minimum of 60° F. (15.6° C.), as favorable a focus almost as a marshy river bank. Yet malaria may originate over a sandy soil and even rocky beds and scanty vegetation, but never without high temperature. All ages are susceptible, but

children are especially liable to take the disease on exposure. More men have it than women for evident reasons.

Currents of air carry the miasm to localities in which it is not engendered.

Morbid Anatomy.—The morbid anatomy of malaria includes, mainly, changes in the blood, the liver, and the spleen, and these changes vary with the duration and intensity of the disease, to which, however, they do not always correspond.

In the true *intermittent* fevers there is a loss, sometimes considerable, of red corpuscles after each paroxysm, which is regained during the intermission. In the æstivo-autumnal form the blood-losses are greater and more permanent.

In *pernicious* malaria, a form characterized by the intensity of the poison and severity of the symptoms, the morbid changes are often not very striking if the patient die in the first attack, though more marked after a second. The blood is described as hydræmic, the serum is sometimes tinged with hæmoglobin, and the corpuscles, while containing the parasite, present all stages of destruction. The spleen is enlarged, but not nearly so much as in chronic recurring forms of malaria. It is, further, soft and the pulp dark from accumulated pigment in the intervascular cords. The liver is enlarged and dark-hued, not necessarily so to the naked eye, though there may be no difficulty in recognizing the excess of pigment within and without the small vessels, some of which may be occluded. In fact, by the aid of a microscope, almost all the tissues may be found abnormally pigmented, even the brain, some small vessels of which may also be occluded. The kidneys are the seat of pigment deposits, and the cells of cloudy swelling.

In *chronic* malaria, of whatever form, the *enlarged spleen* is the most characteristic morbid product. It may weigh as much as ten pounds (4.5 kilos) and measure ten inches (25 cm.) long and four (ten cm.) to six (15 cm.) in width; its capsule is thickened, its substance firm, and the trabeculæ prominent. Pigmented areas abound, due to the plugging of the intercommunicating lymphoid spaces of the pulp with pigment, and in some cases the melanosis is general. The pigment particles resulting from the disintegration of the hæmoglobin in the vessels are retained in the spleen as by a filter.

The *liver* is also enlarged, to a less degree, however, than the spleen. It is indurated and presents various degrees of pigmentation, which may reach a slate-grey tint. The pigment is contained in the portal canals and beneath the capsule.

The *kidneys* may be similarly pigmented, the pigment lying about the smaller blood-vessels and the Malpighian bodies, and in the cells lining the tubules. In protracted cases of malarial cachexia, other tissues may be pigmented. Thus the small vessels of the brain may be surrounded by pigment and even occluded, so that hemorrhagic infarcts may occur. Even the mucous membrane of the stomach and the peritoneum may be pigmented in extremely chronic cases. Malarial poisoning is included among the causes of chronic nephritis, but in considerable experience with renal diseases I can recall but one or two cases of nephritis doubtfully traceable to this cause.

The *blood changes* in chronic malaria are more marked. There is a positive secondary anæmia in which, as usual, the hæmoglobin is decreased rather more than the corpuscles. The leucocytes are almost invariably diminished, the polynuclear leucocytes most, while the larger mononuclear forms are relatively increased. Pigment deposits are abundant.

Clinical Varieties.

The chief varieties of malarial fever admit of easy separation by their symptoms.

Intermittent Fever.

Symptoms.—This, the well-known fever and ague, characterized by distinct paroxysms of chill, fever, and sweat, has a distinct period of incubation, which may be as short as twenty-four hours or even less, though usually it is a week to fourteen days. Sometimes it is very much longer, and even months are said to elapse after exposure before the first paroxysm sets in. The paroxysm is usually preceded by a prodrome of uneasiness and discomfort, sometimes languor and yawning, sometimes headache, sometimes nausea, which forewarns the patient of its coming. As often as not there is no such prodrome. The paroxysm consists of the chill, or cold stage, the fever, and the sweating.

The *chill* commonly begins gradually. First there is a creep, then another a little more severe, then another, each growing in severity until the teeth chatter and the body shakes violently. There is, however, great difference in the severity of the chill. It may be a barely noticeable creep or such a chill as will cause the bed and even the windows of the room to shake. At the same time the patient's lips are blue, his face pale and pinched, and he looks very cold. Yet he will have fever. Even before the chill there is a slight rise in temperature, and during it the temperature may reach 105° F. (40.5° C.) and 106° F. (41.1° C.) in the axilla or mouth. A surface thermometer may show a lower temperature of the skin, but the internal heat is in strong contrast to the apparent coldness. There may be nausea or vomiting and severe headache. The pulse is small, hard, and frequent. The hands are pale, cold, and the nails blue. The urine is increased, light-hued, of low specific gravity, though before the chill it may be concentrated and the specific gravity high. The length of the chill varies from a few minutes to an hour or more.

To the chill succeeds the *fever*. The skin is intensely hot and dry and the face flushed. There is intense thirst. The mouth is dry, the tongue coated, the breath foul. There is no mistaking this stage any more than the first. Yet the actual temperature is but little higher than that during the chill. This is well shown in the appended chart, in which it will be seen that the temperature during the *chill* at two successive observations was 104.2° F. (40° C.) and 104.4° F. (40.18° C.); during the succeeding *fever* it reached at the first observation 104.8° F. (40.4° C.), and at the second 105° F. (40.45° C.). The duration of this stage is from half an hour to four or six hours.

The *sweating stage* follows with the appearance of drops of sweat on the face, whence it extends all over the body and is various in quantity. With it comes relief to all the symptoms; indeed, a sense of great comfort supervenes. It may be a mere suggestion of moisture, or it may be very profuse, drenching the patient's clothing and even wetting the bed. It is commonly proportionate to the severity of the chill. During the sweat the temperature falls rapidly, but if the paroxysm is severe several hours elapse before it attains the normal.

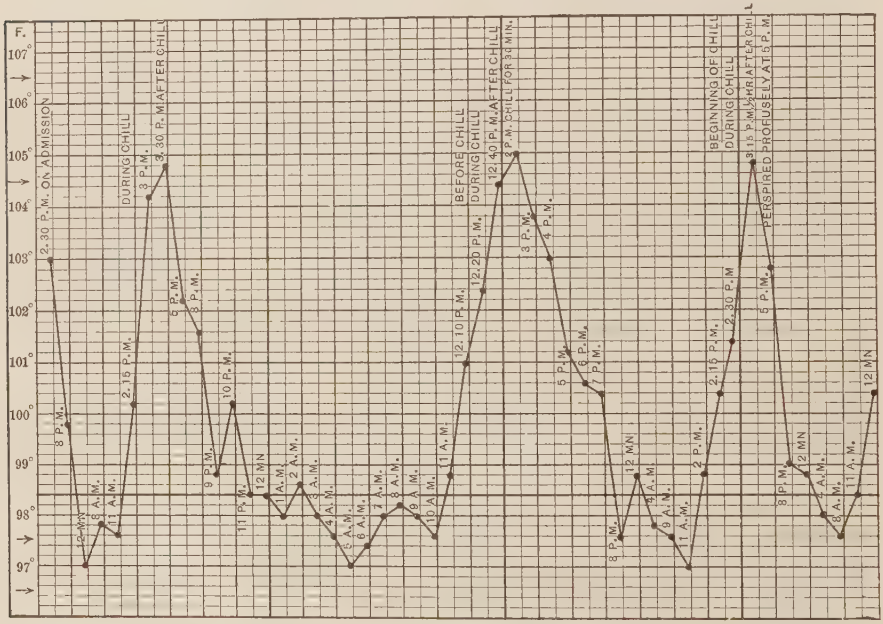


FIG. 8.—TEMPERATURE CHART IN INTERMITTENT FEVER, showing paroxysms and intermission. It will be noted that the temperature has been taken during the chill, and during the fever, just after the chill, and that although, as is well recognized, the fever is very high during the chill, while the patient *feels* cold, it is still a little higher in the fever just after the chill.

It is not easy to give a satisfactory explanation of the mechanism of the three stages. The first and second are undoubtedly the direct result of the same cause,—the plasmodium or a toxic product, since the actual fever which characterizes both is irritative. The superficial coldness and sense of cold of the first stage may be due to a vaso-motor spasm contracting the blood-vessels of the surface of the body with the usual impression on the nervous system of such spasm. The third stage is probably a reactive vaso-motor paralysis with the usual leakage from the skin incident to it.

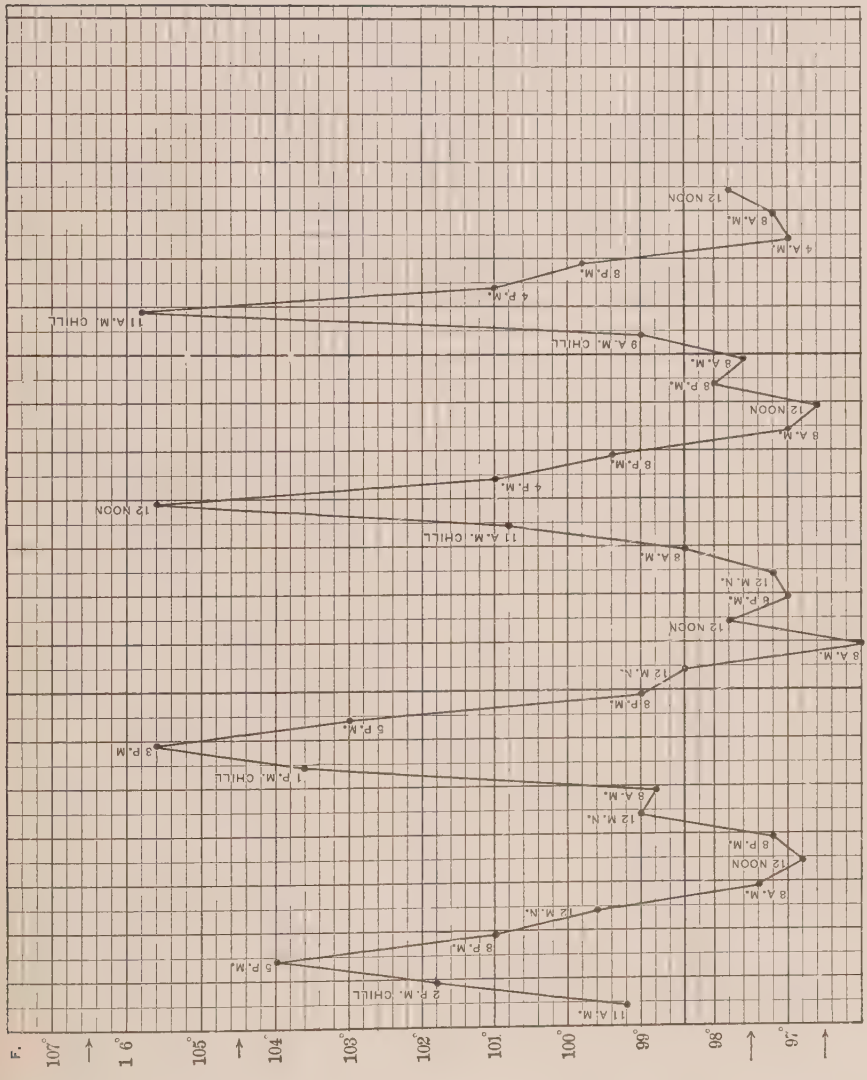


FIG. 9.—TEMPERATURE CHART IN INTERMITTENT FEVER, showing the paroxysms and intermission.

The total duration of the paroxysm is from ten to twelve hours, and usually between the paroxysms the patient feels perfectly well. During the paroxysm the spleen becomes enlarged and there is often *herpes labialis*, a symptom which is almost pathognomonic of malarial fever. The size of the spleen subsides after the paroxysm, although with its repetition there is a disposition to permanent enlargement resulting finally in the ague cake.

The types of the paroxysm so characteristic of intermittent fever have already been referred to. The order of frequency is quotidian, tertian, and quartan, the first being by far the most frequent.

Diagnosis.—The diagnosis of intermittent fever is most easy, and the disease should be easily recognized after the second paroxysm, if not after the first, if the case be typical. If it be less typical and the chill is omitted or so slight as to escape recognition, a certain resemblance between such a paroxysm and the hectic fever of tubercular consumption, with its subsequent sweat, must be admitted, and it not unfrequently happens that such fevers are declared to be malarial by the attending physician, more frequently, it is to be hoped, for the sake of comforting the patient than as a matter of accurate diagnosis.

Still more close is the resemblance of the paroxysm to the chills, fever, and sweats of septicæmia and pyæmia, while suppuration is frequently ushered in, and its progress associated with like symptoms. Other conditions calculated to produce these symptoms may generally be discovered on careful inquiry, though they may escape notice for a time. In addition to suppuration, surgical operations, catheterization, the parturient state, the event of empyema, and the like, all produce chill, fever, and sweat. The so-called nervous chill is easily distinguished, because with it there is no rise of temperature,—at least nothing at all comparable to that of the malarial chill. The possible combination of malaria with other causes of chill and sweats is to be remembered. A search for the hæmatozoon, scarcely necessary for diagnosis in typical cases, may under such circumstances prove extremely useful.

Prognosis.—The prognosis of simple intermittent fever is always favorable. Very frequently if the disease is not treated by medicine it will exhaust itself in a couple of weeks and disappear, while the administration of suitable doses of quinine always puts an end to it. The worst that can happen is the conversion of the disease into chronic malaria or the malarial cachexia. This may happen where treatment is neglected, or where constant exposure to the cause operates to produce such a state notwithstanding suitable treatment.

Treatment.—The treatment is preëminently by quinine. Not only does it promptly break up the paroxysms, but it causes also the rapid disappearance of the plasmodium, which is responsible for them. The dose required varies, but 15 to 30 grains (one to two grams) are usually sufficient for an adult. Sometimes larger doses may be needed in inveterate cases. It does not matter very much how the drug is administered, but there is a best way for each case. I prefer, as a rule, to give an hourly dose of three to five grains, beginning long enough before the expected paroxysm to get the quantity previously decided upon administered at least two hours before the chill is expected. If the first dose

selected fails, the second should be made larger. It is to be remembered, however, that quinine, like other drugs, acts more efficiently after a free aperient, while constipation decidedly interferes with its prompt and efficient action. Some prefer a mercurial, as eight to ten grains (.5 to .666 gm.) of blue mass, or six to ten grains of calomel, but provided a free movement is secured it does not matter much how it is accomplished. Having broken the paroxysm, it is well to continue the quinine for a few days in smaller doses, and to anticipate the seventh day subsequent to the last chill by another full dose of the drug, and to do so at intervals of seven days for some weeks. Under ordinary circumstances the freshly prepared pill of quinine made with aromatic sulphuric acid is to be preferred. This is easily soluble and is not as unpleasant to take as the solution, which is, however, more readily absorbed. The sugar-coated and gelatine-coated pills are not so certainly efficient, as they sometimes, especially with deranged digestion, pass through the bowel undissolved.

The treatment of the paroxysm itself is by measures calculated to combat each stage. During the chill, to satisfy the patient artificial warmth should be supplied, though it does no good and the temperature is already a fever temperature; during the fever, if the temperature is above 102° F. (38.9° C.), the body may be sponged to reduce the heat, and during the "sweat" the patient should be carefully dried. If there be any reason why quinine should not be exhibited, the other alkaloids of cinchona, as cinchonidine, are equally effectual in doses about one-fourth larger. No substitute for quinine has ever been suggested which has stood the test of trial. See, also, treatment of remittent and pernicious malarial fever.

Remittent Fever—Æstivo-autumnal Fever.

Symptoms.—Remittent fever is the form of malarial fever characterized by a continued fever with paroxysmal exacerbations. It is also known as bilious fever. It scarcely occurs at the present day in the North Atlantic States of America, but is confined mainly to the Southern United States, Italy, and tropical countries. It occurs in the late summer and fall and hence is included among the *æstivo-autumnal* fevers.

It generally begins with a chill after a period of incubation analogous to that of intermittent fever. It is more likely to be preceded by prodromal symptoms than is intermittent. There is malaise, intense headache, a coated tongue, and nausea to a very decided degree. Vomiting of bilious matter is a conspicuous symptom. There may be jaundice, the liver may be tender on pressure, the chill may be less severe, and the other stages of the paroxysm less characteristic. The fever does not pass off, but continues with a full, bounding pulse and a temperature of 102° to 103° F. (38.9° to 39.5° C.). There are daily remissions, as in typhoid fever, but they do not follow the same rule of tidal rise. Yet the two diseases are very similar and often thoroughly try the diagnostic skill, even of those who are accustomed to meet bilious remittent fever. The temperature rises quite as high as in typhoid fever, and the patient is really very ill usually. The two diseases occur at the same time of the year, the autumn. It is not impossible for them to be concurrent, constituting the so-called typho-malarial fever.

The urine in remittent fever is high-colored, with high specific gravity, depositing a copious sediment of urates, and sometimes contains biliary coloring matter. Not infrequently it contains blood-corpuscles or hæmoglobin.

Diagnosis.—As intimated, it is with typhoid fever that remittent fever is most likely to be confounded, except, occasionally, yellow fever. To us who study typhoid fever in the North this seems surprising; it is ordinarily so easy, after watching the temperature chart for a few days, to recognize typhoid fever. In the South it is, however, different, and, before the discovery of Laveran's plasmodium, the therapeutic test, the administration of quinine was frequently needed to settle the question; for remittent fever, like intermittent fever, yields to quinine. In such cases a successful search for the hæmatozoon will settle the question promptly. This is the form in which we have the small, actively motile hyaline forms of organism, while the larger crescentic, ovoid bodies appear as soon as the disease has existed over a week. An unsuccessful search may still leave the matter in doubt, but if the nasal hemorrhage, the typhoid spots, the diarrhœa of typhoid, and the temperature are not sufficiently characteristic, the quinine test ought to put an end to all doubt. And, although it may seem presumptuous for one who has not practiced in the South to say it, such study as I have been able to give to the subject impels me to say, with Osler, that all of the continued endemic fevers of the South may be resolved into typhoid or malarial fever.

The diagnostic features between remittent fever and yellow fever will be given when considering the latter disease.

Treatment.—The treatment of remittent fever is essentially that of intermittent fever. It is in this form that the mercurial aperient is deemed especially valuable as a preliminary by those having large experience in its treatment. The continued nature of the fever, and the tendency to a typhoid state which often develops, demands a liquid diet, with the careful addition of stimulants.

Pernicious Malarial Fever—The Congestive Chill.

This form of malarial fever still presents itself occasionally in the North, but is much rarer than thirty years ago. Up to a few years ago it was not uncommon to hear of the death from this cause of a prominent citizen at his country seat on the banks of the Delaware above Philadelphia. Later the cases became confined to the servants and others out late at night or early in the morning, and, more recently still, even such cases as these are seldom reported, although the milder forms of malaria prevail. Two principal types present themselves—the *comatose* and the *algid*. Other forms named from special features more or less characteristic are the hæmaturic, the bilious, and the asthmatic type. As malarial hæmaturia is not confined to the pernicious variety, it will receive separate consideration. The bilious type is that of the ordinary severe form of remittent fever, while the asthma characterizes the comatose and algid types.

(1) The *comatose* type may or may not begin with a chill, but in its more serious forms the chill is a conspicuous feature, being severe. To it

succeeds the comatose state, whence the term congestive chill, often used in the South, where the popular notion prevails that if the first paroxysm does not kill, the second will. This is, of course, an exaggerated idea of its seriousness, although it is certainly a very grave affection and often terminates fatally. A low, muttering delirium may supervene, the eyes are bloodshot, the skin hot and dry, the temperature rising to 105° or 106° F. (40.6° or 41.1° C.). The comatose condition is probably a toxic one and lasts until a partial elimination of the poison has taken place, usually from twelve to twenty-four hours later. The patient may, however, perish without return to consciousness, or consciousness may return to be followed in a short time by fatal relapse.

(2) The *algid* type is also characterized by gastric symptoms, extreme nausea and vomiting, which seem to be followed by collapse, for there is intense prostration with coldness of the surface and extremities. The symptoms are, indeed, comparable to those of the collapse of cholera. There is the same small, feeble pulse, frequent, shallow breathing, cramps, vomiting, purging, husky voice, and thirst, with suppressed urine, and with these the same clearness of intellect, until death steps in, the last scene in the drama, in which asthenia also plays a leading rôle.

In these cases there may or may not be a chill, yet the patient feels cold and the temperature is never high, rarely exceeding 101° F. (38.3° C.), and falling as low as 96° F. (35.6° C.). The internal temperature is, however, high.

Pernicious malarial fever is distinguished from typhoid fever and yellow fever, to which one or other of its forms bears a more or less close resemblance, by the presence of the plasmodium and pigment in the blood. According to the Italian observers also, pernicious malarial fever is associated with the small plasmodium.

Treatment.—The pernicious forms of malarial fever are treated by quinine, as are the other varieties of the disease. In the congestive variety advantage must be taken of the first lucid interval to push the drug in very large doses. Sixty grains or more may be necessary, and advantage may be taken of rectal or even hypodermic injections, but abscesses are almost sure to occur if the latter are used. Extreme cases, however, demand extreme remedies. Soluble salts should be used, such as the tannate, hydrochlorate, and hydrobromate, of which 15 grains (one gm.), dissolved in distilled water, with a grain of sodium chloride, may be injected as a dose. Double this dose may be given. The bisulphate of quinine may also be administered hypodermically with tartaric acid, 30 grains (two gm.) of the former to five grains (0.333 gm.) of the latter. The muriate of quinine and urea may also be given hypodermically in 10, 15, or 20 grain (0.666, one, and 1.33 gm.) doses.*

In addition to the use of quinine, prompt measures must be taken to combat the symptoms which add to the dangers of the situation. Stimulants for the asthenia, and artificial heat for low temperature, morphine hypodermically, to relieve the pain and allay nausea; cool sponging or bathing to reduce the temperature, and saline cathartics to relieve congestion in the comatose form.

* My experience in the hypodermic use of this drug has been satisfactory from the therapeutic standpoint, but I have not escaped abscesses.

Malarial Cachexia, or Chronic Malaria.

Symptoms.—This is a condition which often supervenes in cases imperfectly or ineffectually treated or in persons living in malarial districts where there is constant exposure to the cause and consequent repeated attacks.

The most striking symptom of this condition is *anæmia* of a peculiar kind. The incident changes in the blood have been referred to on page 33. The skin exhibits a dirty yellow or sallow appearance, often erroneously characterized as "bilious," as though it were a form of jaundice, which it is not, although there may be sometimes slight jaundice also. Such persons have also, in addition, deranged digestion. The tongue is pale, flabby, and coated, and the breath sometimes foul. The bowels are constipated. The hands and feet are cold, the circulation generally bad, and the temperature subnormal, though it may alternate with the feverish state. In consequence of the hydræmic blood there is sometimes œdema of the feet and even general anasarca.

Some very unusual symptoms are included in the symptomatology of this form of malaria, as, for example, *paraplegia* and *orchitis*. The former condition may be the result of deranged circulation in the spinal cord, but it is difficult to regard the latter as anything except a coincidence.

Such persons have *enlarged spleen*, the "ague cake," which sometimes extends down as far as the crest of the ilium and as far forward as the median line, and its outline may be felt through the abdominal walls.

The plasmodium is found in this form of malaria also, and the crescent is said to be the form more or less characteristic of it. The recognition of the plasmodium is of value in the diagnosis of this form and other forms of anæmia, although the history of the case and the presence of enlarged spleen are also important factors in diagnosis. It is to be remembered, however, that in leukæmia there is also enlarged spleen, but the microscopic examination of the blood reveals at once in the latter disease the excess of colorless corpuscles.

Treatment.—It is in this form of malaria especially that arsenic becomes a useful remedy, Fowler's solution being the best preparation. It should be given in ascending doses. Iron is often advantageously associated with it, and for such a combination the solution of the chloride of arsenic and the tincture of the chloride of iron are especially suitable. Here, as elsewhere, I am disposed to believe that needlessly large doses of iron have been given and that the constipating effect of iron so justly complained of would be obviated by giving doses little in excess of what can be absorbed. For it is this excess remaining in the alimentary canal which works the mischief. Five minims (.333 gm.) of the tincture of the chloride combined with as many of the solution of the chloride of arsenic are a proper dose, but the arsenic should be increased until a slight puffiness of the face results. The carbonate of iron or the reduced iron or iron sulphate may be given in doses not exceeding one grain (.066 gm.). Iron should be kept up a long time.

Quinine should not be omitted in this form of malaria, but there is no advantage in giving it in large doses. Strychnine and mineral acids are also useful remedies for the gastric derangement, while constipation

may be treated by an occasional mercurial purge, say a couple of grains (.132 gm.) of blue mass, to which may be added a couple of grains (.132 gm.) of compound extract of colocynth, and as much extract of hyoscyamus or one-eighth grain (.008 gm.) of extract of belladonna.

Irregular Forms of Malarial Fever.

It sometimes happens that the paroxysm in intermittent fever omits one or more of its stages. Especially is this the case with the chill, in which event the disease has received the characteristic name of "dumb ague." Frequently, however, what receives the name of "dumb ague" is something altogether different. The "malarial cachexia," for instance, is sometimes spoken of as "dumb ague." Like the malarial cachexia, the "dumb ague" is found among the older residents of a malarial district.

Quite often it happens that the malarial paroxysm consists of nothing but a state of drowsiness, which recurs at regular intervals and is very characteristic. The temperature in these cases is elevated but not very high, 100° F. (37.8° C.) or perhaps 101° F. (38.3° C.). There may be slight delirium. Or there may be intermittent neuralgia, which is clearly malarial, the proof of it being the facility with which it is broken up by quinine. There is not usually fever in this variety. The nerve commonly involved is one of the branches of the trigeminal. The intercostal nerves are also the seats of such an attack, giving rise to one of the forms of pain in the chest, but any nerve trunk or its branches may be affected, as the sciatic or brachial. Such forms of malaria are sometimes called "masked malaria."

The term "latent intermittent fever" is applied to a combination of symptoms affecting persons living in malarial districts—consisting in a weary, languid feeling, associated with want of appetite, headache, nausea, vomiting, constipation, and coated tongue. Sometimes the so-called "bilious attacks" which exhibit the above symptoms in an aggravated form, especially the headache and vomiting, are malarial in their origin and may be broken up with quinine. Such attacks might be called "malarial migraine."

Malarial Hæmaturia, or Hæmoglobinuria, or Intermittent Hæmaturia.

This form of hæmaturia is the direct result of malarial poison. While it is a very frequent symptom of the pernicious or malignant malarial type fever, it also occurs as a symptom, and, indeed, sometimes the sole symptom, of the milder varieties of malaria, such as occur in the Middle States of the United States. I have met a number of these cases. Rarely are they accompanied by a chill. More frequently there is a cold feeling and the tips of the nose and of the fingers become cold, and the lips become blue, immediately after which the urine is found to be bloody. Microscopic examination will recognize in some instances blood discs, in others no corpuscles can be found. It is a hæmoglobinuria.*

* The presence of hæmoglobin can be easily shown by making Teichmann's hæmin crystals in the following manner: The earthy phosphates are precipitated, filtered out, and a small portion placed on a glass slide, and carefully warmed until completely dry. A minute

The hemorrhage occurs daily or on alternate days, more rarely at longer intervals. Sometimes it is continuous with exacerbations at regular intervals. When a symptom of pernicious malarial fever, it may be associated also with hemorrhages from the nasal and oral mucous membranes, and even from the stomach.

Treatment.—The treatment of the milder varieties of hæmaturia is most satisfactory. The administration of quinine in almost any way, say three grains every three hours for several days, will effectually break up the paroxysms, and its use in smaller doses for some time longer will prevent a return. Not every case of intermittent hæmaturia is a case of malarial hæmaturia. But this question is promptly settled by the therapeutic test. If quinine fails to break up the paroxysm of the milder form, when administered as directed, the hæmaturia has an origin other than malarial.

As a symptom of the graver forms of malarial fever, treatment of hæmaturia is that of these forms of fever.

Prophylaxis of Malarial Fever.—Much may be done to avert malarial infection. While it is possible that the organism may enter the system by the stomach, it is more than likely it enters by the respiratory passages. Little can be done to prevent this, but the blood can be made an uncomfortable habitat for the plasmodium by charging it with quinine. To this end a few grains of quinine, say five to ten (.333 to .666 gm.), should be taken daily, especially by newcomers and by all residents at times. It seems pretty well founded, too, that malaria is more active after nightfall and in the early morning. Hence exposure at these times should be avoided. It is also a notion, probably founded on experience by residents in malarial districts, that exposure while the stomach is empty is apt to invite the poison. Hence exposure under these circumstances should be avoided.

CHOLERA.

SYNONYMS.—*Cholera asiatica; Cholera algida; Cholera maligna; Cholera infectiosa; Epidemic Cholera.*

Definition.—Cholera is an acute infectious disease caused by the invasion and proliferation of the comma-bacillus or spirillum of Koch, characterized especially by vomiting, purging, painful cramp, and collapse.

Historical.—Cholera is a disease long endemic in certain localities in India, whence it has made periodical visitations to Europe, and in 1831–33 for the first time to North America. It entered the United States in 1832 by two channels of immigration, first, from Great Britain by the way of Quebec and the Great Lakes, reaching the then limits of settlement, the military posts of the upper Mississippi; second, by the way of

granule of common salt is carried on the point of a knife to the dried mass and thoroughly mixed with it. Any excess of salt is then removed, the mixture is covered with a thin glass cover, a hair interposed, and a drop or two of glacial acetic acid allowed to pass under. The slide is then carefully warmed until bubbles begin to make their appearance. After cooling, hæmin crystals can be seen by aid of the microscope. These, though often very small and incompletely crystallized, are easily recognizable by an amplification of 300 diameters. They are chemically hydrochlorate of hæmatin.

New York. In 1835–36 another visitation occurred, and in 1848 another by way of New Orleans and the Mississippi valley, extending even to California. In 1854 a severe epidemic raged through the United States, for which immigration was also responsible. In 1865 Arabia and Egypt were severely visited in the spring, Constantinople in July, and thence all Europe. In 1866 it again appeared in Egypt, spread over all Europe, reaching the United States the same summer, during which there were quite a number of cases, some of which came under the observation of the writer in the Philadelphia Hospital. An extensive outbreak prevailed in Europe in 1884, extending to Italy, Spain, and France, but it did not reach the United States.

Much more serious than any of the more recent epidemics was that of 1892, which started in March or April in the northwestern provinces of India, attacking with great violence the pilgrims at the great Hurdwar fair, near the source of the Ganges, and extending thence through Cashmere and Afghanistan to Persia, where it arrived in May or June. Thence it crossed the Caspian Sea and spread rapidly through European Russia into Prussia, seating itself most stubbornly and savagely in Hamburg in August. Havre, Antwerp, Berlin, Vienna, and especially Buda-Pesth in Hungary were also visited. A few cases occurred in Southampton, London, and Liverpool, and it reached New York Harbor in September, 1892. A few cases were also reported in New York City, but there was no further spread.

Etiology.—It is now generally acknowledged that cholera owes its existence to the comma-bacillus or spirochæta of Koch, a semi-spiral rod-bacillus discovered by him in 1884. Its multiplication is favored by heat, moisture, and filth. It is easy of destruction, even by weak acids and a temperature of 140° F. (60° C.). It can produce cholera only when it is taken in by the stomach, where, however, a normal gastric juice is always able to destroy it, while weak digestion induces a vulnerability which is promptly availed of by the bacillus, which quickly passes into the intestine, where the alkaline reaction of the secretions favors its development in enormous numbers. Bacilli are rarely found in vomited matters, but are numerous in the fecal discharges and are found in the intestines after death. They may invade the follicles and intestinal wall, but some time is required for this, and such invasion does not occur in cases speedily fatal.

Drinking water is the acknowledged medium through which the bacillus is commonly introduced into the human organism, but it may be conveyed in clothing or food, on the hands, and may even enter the mouth while floating in the air. It frequently follows in the train of moving masses of human beings, such as in emigrants and pilgrims, but it prefers the sea level and lower altitudes, especially less than 1000 feet (30.5 meters) above the sea.

While at the present day the views of Koch as to the origin and spread of cholera are largely dominant, it should not be overlooked that an authority as high as that of Pettenkofer, of Munich, still holds that the germ of cholera develops in the soil-water of the earth during the heated months, and rises in the atmosphere as a miasm. He claims that the conditions peculiarly favorable to its development are a low soil-

water, associated with porosity, moisture, and a contamination with organic matter, especially sewage.

As stated, anything that enfeebles digestion favors its permanent lodgment and multiplication. Hence general ill-health, fatigue, the alcoholic habit, depression of spirits, fright or anxiety, any one or all may be predisposing causes. All ages and sexes are liable to be infected, but young children seem most vulnerable.

Morbid Anatomy.—The appearance of a man dead of cholera may present no peculiarity. More commonly there is a shrunken aspect of the whole frame, the skin of the exposed and non-dependent parts is grey or ashen hued, while the dependent portions are livid. The eyes are deeply sunken, the temples hollow, the nose pinched, and the skin clings closely to the bones beneath it. The appearances of such a body, in brief, are those of a wasted body long immersed in the pickling vats of the dissecting room.

Very striking are the post-mortem elevations of temperature and the phenomena of post-mortem muscular contraction. The former has reached 109° F. (42.8° C.) and higher. The latter include movements of the lower jaw, rotation of the eyes, contraction of the arms and legs, sometimes startingly life-like.

On section of the body the subcutaneous tissue is found dry, the blood in the vessels thick and dark. The *stomach* is commonly but not always filled with a turbid liquid, greyish-white in color, resembling rice-water. In this the microscope may recognize columnar epithelial cells, isolated and in flakes; also the remnants of partially digested food, such as disintegrating muscular fasciculi and oil globules. The mucous membrane of the stomach appears congested, and the course of the larger vessels can be readily traced in consequence of their being full of thick blood. A papillated appearance ascribed to enlargement of the solitary follicles is often present. The epithelium is detached in places, in others intact.

The mucous membrane of the *small intestine* may also be much congested; the bowel is filled with rice-water fluid. On its surface lie numerous patches or flakes of detached epithelium, while the papillated appearance produced by the enlarged lymphadenoid follicles is everywhere present. The villi are largely denuded of epithelium, but in places they are intact.

If death takes place during imperfect reaction, the gastro-intestinal mucous membrane is still more congested and dark red in color from hyperæmia and blood extravasation. At such times, too, the solitary glands are conspicuous, as also are Peyer's patches, and the same denudation of epithelium from the villi and elsewhere is present. The signs that suggest an inflammatory process are a slight cellular infiltration of the intestinal walls, and the enlargement of the solitary follicles.

The *liver and kidney* are natural in size, but may be congested and darker hued than in health, while the cells exhibit cloudy swelling. The *spleen* is usually small, certainly not enlarged.

The *heart* is normal in size, but its walls flaccid. The right cavities are commonly filled with dark, liquid blood, the left cavities empty.

In many instances the *lungs* also present an appearance more or less characteristic, being shrunken and small, lying back in the thorax as

though collapsed. Like the other tissues, they are empty of blood except in their dependent portions, which are the seat of hypostasis. They have been compared by Parkes to fœtal lungs. Sutton found the two organs to weigh but 20 ounces (1.33 gm.), as compared with 45 ounces (three gm.) when death occurred after reaction had been established, that is, after the blood had again occupied the pulmonary artery and its branches. Collapse may be interfered with by adhesions, in which event it is only partial.

Such appearances could, of course, occur in death from hemorrhage and, after all, the only distinctive condition is the presence of the rice-water fluid in the stomach and intestine, or in both, containing the "comma" bacillus and desquamated epithelium. The last, to which the earlier descriptions attached great importance, is now generally regarded as post-mortem in origin. The flakes thus produced are also what the older authors described as patches of lymph.

Symptoms.—After a period of incubation ranging from two to five days, the symptoms of cholera commonly present themselves sufficiently gradually to admit of arrangement into three distinct groups or stages :—

(1) The stage of preliminary diarrhœa.
(2) The stage of collapse, and
(3) The stage of reaction.

The stages are by no means always recognizable, and the severity of the symptoms varies greatly, such variations being reasonably ascribed to the varying quantities or virulence of the specific poison, while mildness in a given case is no guarantee against virulence in another caused by it.

(1) *The stage of preliminary diarrhœa* * is characterized by moderate diarrhœa, which may be painless or may be associated with colicky pains. The stools are yellow or yellowish throughout this stage and are alkaline in reaction. Nausea and vomiting are not usual in it and the patient may feel but slightly indisposed. There is generally a feeling of restless discomfort and depression, to which headache may contribute. The temperature remains normal. The first stage may last for a week or longer, or for a few hours only, or it may be entirely absent.

(2) *In the stage of collapse* the diarrhœa has become profuse. The discharges have lost their yellowish color and resemble thin gruel or rice-water. The fluid gushes out with great profuseness and apparent force. There may be griping or tenesmus, but more characteristic are the very painful muscular cramps, which usually begin in the fingers and toes and extend thence to the calves of the legs and abdominal walls. Vomiting, bilious at first, is soon added to the diarrhœa. The fluid vomited soon assumes the rice-water character and gushes from the mouth as from the bowel in enormous quantities.

Extreme weakness and exhaustion are by this time present. The skin is blanched and shrunken, the lustreless eyes are sunken and bounded below by great circles of blue. The nose is pinched, the lips thin, the cheeks hollow, and the countenance pallid to bluish greyness. The extremities and entire body become clammy and cold, the superficial

* To this stage the term cholerine has also been applied, but this word is now more commonly used to indicate a mild form of cholera.

temperature falls five to six degrees, while that of the rectum rises to 103° and 104° F. (39.5° and 40° C.). Speech is husky, whispering, and labored. The pulse is feeble or frequent or absent at the wrist, and the patient appears to be dying. Even the heart-beat and sounds are almost gone, but the breathing continues. Through all this consciousness may be maintained to the end or coma may supervene. Death commonly occurs in this stage.

On account of the scantiness of blood certain secretions cease and there is neither urine nor saliva, while power to perspire remains, and, it is said, also the lacteal secretion in nursing women.

A more close examination of the rice-water vomited matters and bowel discharges reveals flakes of epithelium, mucus, and granular debris, and, with sufficiently high powers and suitable preparation, the cholera bacillus, together with numerous other bacteria. Occasionally a little blood is present. The fluid is albuminous and contains the salts of the blood, among which sodium chloride is conspicuous. Sometimes, however, there may be no vomiting or purging, whence the term *cholera sicca*. In these cases, however, the stomach and bowels are commonly found containing the characteristic fluid after death.

This second stage is generally of shorter duration, commonly a few hours only, but it may be prolonged to twelve or twenty-four. The disease is sometimes ushered in with the symptoms of this stage. It has been ascribed to the action of a ptomaine produced by the bacilli, which, when absorbed, produces the systemic effects of this stage, but it is likely that the flux is the principal factor in its production.

(3) *The stage of reaction* is characterized by the return of warmth and color, the latter more slowly, and the re-establishment of secretions. Especially favorable is the return of the urinary secretion. Along with these changes the vomiting and purging occur at longer intervals. Such improvement is, however, often delusive. The diarrhœa may return, the collapse repeat itself, and the patient die. Or there may supervene *cholera typhoid*, a state characterized by a frequent feeble pulse, dry tongue, delirium, and sometimes an erythematous or roseolar eruption on the extremities. This may end in recovery. Or there may be superadded symptoms of nephritis, including uræmia, coma, and death. Or there may be inflammation, diphtheritic or catarrhal, of the bowels.

Diagnosis.—In the matter of the diagnosis it is well known that, so far as symptoms are concerned, cases of cholera morbus, *cholera nostras*, or sporadic cholera, as we may prefer to name it, have occurred with symptoms absolutely identical with those of true cholera, including the fatal termination. It is not worth while, therefore, to contrast the clinical features of true cholera with those of cholera morbus, with which alone it is likely to be confounded. By bacteriological investigation only, including also cultivation on artificial media, can a given case be identified with absolute certainty. Not less than thirty-six hours are, however, necessary to complete such a bacteriological diagnosis. Further, such investigation can only be made by those who are expert and provided with proper facilities. Such expertness and facilities, moreover, are not found in the hands of the general practitioner, and the bacteriological investigation is, therefore, of limited application. Doubtless, however,

should occasion demand, the authorities in the large cities, at least, will furnish the same assistance they now do in the case of diphtheria.

As to the microscopic examination of the dejecta, which is more feasible for the practitioner, it may be said if the examination reveals a preponderance of curved bacilli, comma-shaped, and sometimes joined end to end, so as to form figures somewhat resembling the letter S, and again appearing in long threads, we may feel justified in considering the case one for careful study by bacteriological methods. Although there are found in the alimentary tract other bacilli, the morphology of which is much like that of the cholera bacillus, they are not numerous. The bacillus of Prior and Finkler, found in the stools of cholera morbus, while closely resembling the true comma-bacillus of Koch, is larger and thicker. More easily distinguished are the cultures. That of the former grows more rapidly and its shape is *saccular*, while that of the cholera bacillus is *conical*. It also liquefies the gelatine much more rapidly.

How, then, shall we know a case of vomiting, serous diarrhœa, severe colicky pain, followed by collapse, to be a case of cholera? In this country, where such a thing as endemic cholera is unknown, it goes without saying that any isolated case, even if fatal, cannot be one of true cholera, unless there be traceable some connection with an acknowledged focus of cholera elsewhere. Second, such communication must have taken place within the period of incubation required for the development of the case, say within six days. Of course, such communication need not be a personal one. It may be by clothing, merchandise, and probably even letters.

These conditions being fulfilled, the patient suffering with the symptoms of cholera must, for the time being, be regarded as a case of the true disease, and isolated until the bacteriological investigation can be made, but the rapid occurrence of similar cases increases the probability of its being true cholera, and finally establishes its certainty. Yet local epidemics of cholera morbus do sometimes take place, severe and grave in character, due to local causes, and favored by extreme and long-continued heat. Thus it is still the question whether the recent epidemic of cholerine that prevailed in Paris in May, June, and July, 1892, was true cholera or cholera morbus, and there seems much reason to believe it to have been the latter, notwithstanding the prevalence of true cholera elsewhere in Europe.

There is one very important etiological difference in the two diseases which is also of great diagnostic value, and that is that almost invariably cholera morbus is traceable to a severe and irritating exciting cause, such as a meal of indigestible fruits or vegetables, or imperfectly cooked or decomposing fish or shell-fish, while cholera comes on without any such cause, or succeeds trifling derangements of digestion, which in other than cholera seasons pass away without harmful results. As a rule, too, the symptoms of cholera morbus are much more severe at first than those of true cholera, and the substances first vomited are undigested articles that have acted as exciting cause, succeeded by green, bilious matter. The discharge from the bowels is first also of a more bilious character, and, above all, the mortality is much less serious; indeed, recovery is the rule. Yet these differences are not to be relied upon.

(See, also, appendix to section on Cholera, p. 84.)

Symptoms similar to those of cholera arise from poisoning by corrosive sublimate, tartar emetic, arsenic, and mushrooms, but their symptoms are rarely confounded with those of cholera.

Prognosis.—The prognosis, always grave, varies with the stage of the epidemic. It is well known that in the beginning a very large proportion of cases die, fully 80 per cent., but as the epidemic is prolonged the ratio of deaths to persons attacked grows less, the mortality falling to 30 per cent. or less. The habits and morals of the patient have an important influence. Intemperance and dissipation diminish greatly the powers of resistance, as do also fatigue, indigestion, fright, and fear.

Treatment.—The treatment of cholera is very appropriately divided into prophylactic and medicinal, the former, when properly carried out, being more effectual than the latter.

Prophylaxis.—In the first place, it has been shown that a certain degree of immunity from cholera is secured by a first attack, through a collective investigation directed by the Academy of Medicine of Paris in 1884, and information collected by E. O. Shakespeare during his residence in Spain in 1885, appointed by the United States Government to investigate the subject. From this standpoint it has been sought to secure immunity by inoculation of protective virus by Ferran and others. The former, using a pure culture in bouillon of the comma bacillus, practised the method in Spain during the epidemic of 1885, but a French commission appointed to investigate it reported unfavorably and it fell into disuse, although Dr. Shakespeare in his "Report on Cholera in Europe and America" is inclined to think there are possibilities in Ferran's method which make it worthy of further consideration. Gamaleia, Löwenthal, Brieger, and Wassermann* secured immunity in animals, and more recently G. Klemperer† obtained results which went to show that immunity could be conferred on man by a previous treatment with cultures of cholera bacilli sterilized by heat; also by the subcutaneous injection of the milk of immunized goats, though the immunity is considerably less by the latter than by the former. It was found, too, that the blood serum secured from persons treated with injections of from .1 to one c. c. of such cultures had the property of conferring immunity on guinea pigs.

A. Lazarus soon added (1892) that the blood of man, after recovery from an attack of cholera, has the property of protecting guinea pigs from fatal infection when injected in very small quantities into the peritoneal cavity. Issaif, in 1894, confirmed the latter observation, but showed that the property was temporary. The same year this observer, with R. Pfeiffer, showed that the specific properties of the serum are due not to a bactericidal action of the serum, but to a specific bactericidal action of the exudate produced in the peritoneal cavity by such injection. This view Pfeiffer founded on his experience that the destruc-

* Brieger's experiments were upon guinea pigs, which he succeeded in making immune to virulent cultures of cholera bacilli. The method consisted in making intraperitoneal injections of comma spirilla cultures prepared in watery extract of calves' thymus or in beef bouillon.—*Deutsche Med. Wochenschrift*, 1892, No. 31.

† *Berliner Klin. Wochenschrift*, 1892, No. 39, p. 969; Med. News, Phila., October 29, 1892, p. 496.

tion of the living cholera bacilli quickly takes place in the peritoneal cavity of the guinea pig, if at the same time a minute quantity of the serum from an immune animal is injected. Pawlowsky, Buchstal, and Federoff extended these observations, using the serum of immunized dogs with similar success. Up to the present time, however, apparently but three cases of cholera have been treated by subcutaneous injections of blood-serum from persons who recently suffered attacks of cholera. Of these, one died and two recovered.

The results of protective inoculations by cultures by M. Haffkine's method are more to the point. His researches at the Pasteur Institute, and published in 1892, started with the inoculation of the animal in the peritoneal cavity and exposure of the resulting exudate to the open air for several hours. A transfer of this to the peritoneum of another animal secures an "exalted" or "fixed" vaccine, after the Pasteur method, whose subcutaneous injection secures anticholeraic immunity, but with necrosis of the cutaneous tissues. This necrotic effect is removed by cultivating it at a temperature of 102.2° F. (39° C.) in an atmosphere constantly aerated. Successive inoculations result in an attenuation of cultures, which, injected under the skin of animals, even in exaggerated doses, produces only a passing œdema, while it leaves the animal immune to the "exalted" or "fixed" virus. The same harmlessness attends its inoculation under the skin of man, where the microbes die and disappear, setting free a substance which acts upon the organism and confers immunity on it. The same result follows the injection of their dead bodies only. Thus he was enabled to prepare vaccine, preserved in weak solutions of carbolic acid, which remains efficacious for six months, and may be used by persons without bacteriological training.

From the summary of cases, some 32,000 in India during 1893–94, treated by his method, published by M. Haffkine in January, 1895, we note a reduction of mortality from 6.51 per cent. in cases not treated to 3.8 per cent. of those treated. No other method has been tested by anything like as many cases, and though the correct treatment may not yet have been arrived at, it seems reasonable to believe that we are in close pursuit of one which is most promising of results.

Until these processes are perfected we must be satisfied with a prophylaxis which, in point of fact, is little, if at all, less efficient in securing immunity than the most efficient inoculation methods as yet suggested. By means of it cholera has been virtually kept out of England and the United States since 1873, though brought to certain ports where it has been held at quarantine. It consists mainly in the isolation of the patient and in certain precautions against the spread of infection by sterilizing the discharges. To this end:—

(1) The vomited matter and the discharges from the bowels are to be gathered in carbolic solution, one to 20, or chlorinated lime, one to 10, some of which should be in the vessels before it is used. After use, more should be added. The matter thus collected should be gently stirred and allowed to remain twenty minutes before being poured into the water-closet hopper. Where the excreta can be thrown into a pit, or even, as may be done in the country, on the manure pile, milk of lime, or, what is the same thing, ordinary whitewash, is a very suitable and cheap medium with which to disinfect them.

(2) After vomiting, the mouth of the patient should be rinsed with a solution of hydronaphthol, one to 5000, care being taken that none is swallowed. After each evacuation from the bowels, the buttocks, thighs, and anus should be washed with soap and water.

(3) All body and bed linen soiled with the discharges should be immediately moistened with carbolic solution, one to 60, and removed in a covered vessel from the apartment, placed in a wash boiler, and boiled for half an hour in a one per cent. solution of washing soda.

(4) Napkins, towels, and table linen should be placed in a similar vessel or canvas bag for removal and similarly boiled.

(5) All dishes, knives, forks, spoons, etc., used by the patient should be boiled after each meal in a one per cent. solution of soda.

(6) The remains of meals should be thrown into a vessel containing milk of lime or whitewash, and removed at the end of the day.

(7) Door-knobs are liable to be soiled by the hands of one carrying out excreta, and should be carefully watched and cleaned and sterilized lest they, in turn, communicate the infectious material to another handling them.

(8) In case of death, the body, without being washed, should be wrapped in sheets wet in a solution of bichloride of mercury, one to 1000, and allowed to remain until removed for prompt burial.

Special Directions to Nurses:—

(1) In like manner nurses of cholera patients should not hold any direct communication with others during attendance on such cases.

(2) They should, under no circumstances, take their meals in the same apartment with the patient, and before leaving the room the hands should be cleansed with soap and bichloride solution, and such portion of the dress as is liable to be soiled should be changed. The hands should be again rinsed in bichloride solution one to 1000 after leaving the patient's room. A very convenient plan is to wear a slip or "over-all" with a hood to cover the hair, which can be easily thrown aside before leaving the room. A canvas slipper or overshoe readily removed should also be worn in the sick-room.

(3) The food of the nurse should be wholesome and plain, freshly cooked, and served hot. No uncooked vegetables should be eaten. Milk should be boiled and, if desired, cooled before using. Cold drinks should be taken moderately if at all. Coffee and tea may be taken hot.

(4) Teeth should be cleansed after each meal, as the mouth affords a peculiarly favorable nidus for decomposing matters and a favorable nidus for the multiplication of pathogenic fungi. A daily bath in warm water, with the use of soap, should be taken by each nurse.

(5) Care should be taken to keep the body from being chilled by drafts or other cool exposure, and to this end woolen underclothing should be worn.

(6) Courage and cheerfulness are amply justified because it is really almost impossible to take cholera if the above precautions are carried out.

The Treatment of the Attack.—The indications in the management of cholera apart from isolation of the patient and the sterilization of the discharges are to relieve the pain, check the flux, counteract the effects, and if possible destroy the organism responsible for it.

For the relief of cramps morphine hypodermically is to be preferred

because of the promptness of its effect and because absorption from the gastro-intestinal mucous membrane is much hindered if not altogether prevented in true cholera, while the vomiting is a further obstacle to the administration of medicine by the mouth. Full doses should be given, a sixth (.016 gm.) to a fourth (.014 gm.) of a grain, which may be repeated if necessary. If circumstances compel the administration of anodynes by the mouth, chlorodyne is one of the most suitable and is well administered in brandy or whiskey. Such administration, too, fulfills any indication for opium to control the bowels.

Considerable difference of opinion exists as to the propriety of giving opium for this latter purpose, but there can be no doubt about its usefulness in the earlier stages and in cholerine. A combination of salol and subnitrate of bismuth in large doses with wine of opium or deodorized tincture is a valuable remedy.

It has long been the practice to prescribe in cholera as well as cholera-morbus a mixture of stimulating aromatics and irritants with opiates, and there is no doubt that in the early stages of cholera such combinations may be of value. The following is one of them :—

℞. Sp. Menth. Piper.,
Tr. Opii,
Sp. Chloroform.,
Sp. Camphor.,
Tr. Capsici, ... āā . . . . . ℥ss (1.8 c. c.)
Tr. Zingib.,
Sp. Vin. Rect., ... q. s. ad . . . . . f℥ij (60 c. c.).
M. et S.—Teaspoonful in hot water or black tea every fifteen minutes until relieved.

Paregoric in drachm doses (3.7 c. c.) similarly administered early in the diseases is often sufficient to control the symptoms.

The following is the well-known cholera mixture or diarrhœa mixture of Squibb, which is given under the same circumstances :—

℞. Tr. Opii,
Sp. Camphoræ,
Tr. Capsici, ... āā . . . . . f℥j (30 c. c.)
Chloroformi pur., ... ℥iij (11.1 c. c.)
Alcohol, ... q. s. ad . . . . . f℥v (150 c. c.).
M. et S.—Teaspoonful every hour or two hours.

The chief reason given against the checking of the discharges is that the bacilli, whose presence is directly or indirectly the cause of the flux, are thus retained. But such object is offset by the fact that the flux itself is the greatest source of danger and that if it can be controlled the bacilli are comparatively harmless. Unfortunately, in the later stages, when the flux is established, nothing avails to control it, and the opiate may as well be limited to that hypodermically administered for the relief of pain.

The effect of the copious discharge is to produce the intense exhaustion referred to under symptomatology, and it is imperative to counteract this if possible by stimulants freely administered. Champagne, brandy, and ammonia, combined with ice and carbonated waters, are suitable. If not retained by the stomach, whiskey, ether, and ammonia may be given hypodermically in 30 minim (two gm.) doses frequently repeated. The

hope of benefit from these remedies is justified if reaction once sets in. The application of heat to the body by hot baths, hot water bags, and hot water bottles is an important adjunct.

More serious still is the drainage of moisture from the tissues, and the most serious consequences ensue from the resulting stagnation in the blood. To restore its liquidity is, therefore, of the greatest importance. Transfusion of watery solutions suggests itself, but the difficulty and delay involved in practising are opposed to its use. Hypodermic injections of hot saline solutions or hypodermoclysis, also enemata or enteroclysis of similar fluids, slightly astringent, were practised successfully by Cantani in Italy in 1892, and have been continued with various results in Europe, and in a more limited manner, with satisfactory results, at Swinburne Island in New York Harbor. The method practised at the latter place, as described by Judson Daland, is as follows: Water at 104° F. (40° C.)

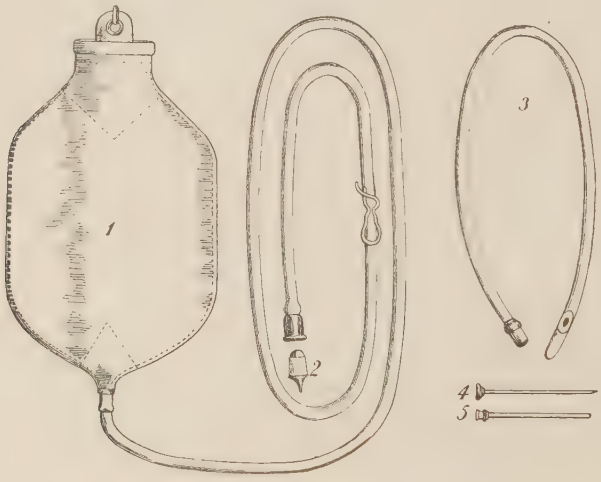


FIG. 10.

1. Rubber reservoir and tube of fountain syringe for hypodermoclysis. 2. Attachment for canula. 3. Soft rubber rectal tube. 4. Needle 5. Canula.

previously sterilized, and containing 0.8 per cent. of sodium chloride and one per cent. of brandy, was introduced under the skin in the midaxillary line in the region of the floating ribs through a long hypodermic needle and canula attached to the tube of a fountain syringe or Davidson syringe. The former is preferred, because the pressure may be neatly regulated by raising or lowering the bag. One or two quarts may be introduced, thirty to forty-five minutes being required in favorable cases for the former quantity. Where absorption is slow, it may be facilitated by manual manipulation at the seat of the swelling which results at the point of injection. In unfavorable cases a much longer time is required to introduce this quantity, as much as four hours, whence the rate of absorption becomes of prognostic value. The operation may be repeated in two hours, or in severe cases one quart may be injected in each flank, repeated as soon as absorption is complete. The quantities to be used may be laid down at, for an

adult, two pints (one liter), an adolescent one pint (.5 liter), and an infant half a pint (.250 liter). Other sites may be selected for injection, as the buttocks, inner surface of the thighs, or below the pectoral muscle. The neighborhood of the neck should be avoided because of the possible œdema of the larynx, such an accident having occurred at Swinburne Island. The benefit derived from the use of this measure under other circumstances, as, for example, succeeding large hemorrhages and uræmia, together with the facility with which it can be carried out, commend it strongly. A teaspoonful of common salt to a gallon of sterilized water furnishes with sufficient nearness the proportion desired.

Whenever the discharges have been so copious as to make it reasonable that the vessels are becoming drained, hypodermoclysis is indicated and may be repeated every two, four, or six hours as required.

Enteroclysis is made with a one or two per cent. solution of tannic acid at a temperature of 45° C., or 113° F. For an adult two quarts (two liters) may be administered, for an adolescent one quart (one liter). It is introduced slowly by a fountain syringe or Davidson syringe through a rectal tube by a medium syringe closed at the end, but having two lateral outlets one a half inch from the end, the other two inches from the end. The tube is introduced gently by a combined rotary and pushing motion to the depth of ten inches, when the fluid is allowed to enter slowly, allowing not less than ten minutes. The patient should, of course, be encouraged to retain the fluid, and may be aided by pressure on the anus with a napkin. Enteroclysis is said to be useful in any moderately severe case of cholera, and may be given night and morning, more frequently in severe cases. According to Daland, experiments made at Swinburne Island in the autumn of 1892 showed conclusively that when thus introduced fluid can be made to pass through the ileocæcal valve into the small intestine. In fact, several patients vomited the solution.

It is in the algid stage that this treatment is more particularly used, but other means must be taken to keep up the warmth of the body. To this end the patient is immersed in the hot bath at a temperature of 38° to 42° C., or from 100.4° to 107° F. In favorable response the warmth of the body returns, the pulse is fuller and stronger, and the respiration deeper. Hot-water bottles, hot-water bags, and hot bricks may be applied alongside the body.

In the stage of reaction, indicated by the return of warmth, pulse, and heart-beat, and especially the establishment of the urinary secretion, the restorative measures are continued with the addition of judicious nutriment, preferably in the shape of peptonized foods, especially peptonized milk. Great care must be exercised lest diarrhœa be induced by too liberal feeding. Convalescence is necessarily very slow in serious cases, and relapses are prone to occur.

The third indication is that for destroying the bacilli and producing conditions unfavorable to their development. Salol is the remedy which has chiefly been employed for this purpose of late by Löwenthal and others because of its ready decomposition into salicylic acid and carbolic acid. Doses of 15 grains every second and third hour were given. No opportunity has, as yet, occurred to use guaiacol, but it is likely to prove a valuable antiseptic, so far as this treatment is of use.

The greater or less usefulness of calomel in cholera, as attested by experience in so many epidemics, is more rational at the present day by reason of its antiseptic qualities, although it is probably as efficient in controlling vomiting as any other drug. The plan pursued at the New Hamburg Hospital and at the Moabit Hospital in Berlin was to give an initial dose of four grains (.266 gm.), after which ⅓ to ¾ grain was given every two hours through the first and second stages. On Swinburne Island a single dose of ten grains was usually given, but it was not repeated or continued in small doses.

Appendix.—The Examination for Cholera Bacillus.

I add the method practised for this purpose in the Bacteriological Institute at Berlin furnished by Dr. Louis Fischer.

The articles necessary are :—

(1) A microscope with Abbé's condenser and an oil-immersion lens of $\frac{1}{12}$ in. focal distance.

(2) A solution of fuchsin one gm. in 90 c. c. distilled water and ten c. c. alcohol.

(3) A few pipettes, glass rods, object glasses, or cover glasses or slides.

(4) A few platinum wires melted or soldered to the ends of glass rods.

(5) A few hollow slides.

(6) Ten to 12 glass plates or glass panes about 12 cm. long and nine cm. wide.

(7) About a dozen ordinary flat plates.

(8) An alcohol lamp or gas, preferably a Bunsen burner.

(9) A number of test-tubes with sterilized gelatine.

(10) A number of test-tubes with sterilized nutrient bouillon.

(11) A few Erleymer's glasses, about one-third filled with one per cent. pepton solution,—one gm. pepton, 0.5 gm. chloride of sodium, 100 gm. distilled water.

(12) Concentrated sulphuric acid.

The dejecta of the suspected patient are scattered in as thin a film as possible on a glass plate, and this is carefully examined by the aid of a platinum wire for a mucous flake, "Schleimflocke," which is laid on the edge of the plate and isolated. From this is taken a piece the size of a pin's head by means of a platinum loop sterilized by drawing it through a Bunsen burner. The fragment is rubbed on a cover glass until it is evenly divided; superfluous material is removed by pressing another cover glass over it; the two are separated and allowed to air-dry.

The glass cover is then drawn three times through the flame of the Bunsen burner in the same manner as for the examination of sputum for tubercle bacilli, and by means of a pipette a few drops of fuchsin solution are placed on the cover glass, allowed to remain one or two minutes, and then washed off in distilled water. A drop of water is put on the cover glass, which is laid on a slide and examined with the oil-immersion system. If it be desired to preserve the specimen, after staining with fuchsin solution wash off the excess of stain with distilled water, allow it to get thoroughly air-dry, add Canada balsam, and mount.

In some of the fulminating cases where the intestinal contents have a colorless or pale red color, with slimy flakes or with a flour-soup mass, especially in the period of reaction, the cases running a slow course, no mucous flakes will be found, but large quantities of blood. Here may be found, besides cholera bacilli, large quantities of other micro-organisms, while the cholera bacilli are but sparingly present. To render a diagnosis absolutely positive in such cases, "cultures" are necessary.

Cultures can be made in "hollow slides" by smearing the border with vaseline, then bringing a small drop of sterilized bouillon into this hollow groove of the slide by means of a platinum wire loop, and inoculating the bouillon with the smallest possible particle of the suspected mucous flake. The cover glass is carefully laid on the vaseline, which serves to render the groove air-tight, and also prevents evaporation of the drop of sterilized bouillon. The slide is then laid aside at a temperature of 20° to 22° C. (68° to 70° F.). The room can be heated if the temperature is below this. In about twenty-four hours the bouillon becomes turbid, and the slide can be examined with the oil-immersion lens without disturbing the culture. The best place to examine is the border line, and even if but few cholera bacilli were originally present, they grow so rapidly that they can be easily recognized by their curved shape.

Culture Method by Schottelius: Take 100 to 200 c. c. of the suspected dejecta from the intestinal contents and place them in a beaker glass containing 250 to 500 c. c. of mild alkaline meat bouillon and mix thoroughly; then let this mass stand twelve to twenty-four hours at a temperature of 30° to 40° C. (86° to 104° F.). After this time the cholera bacilli have usually increased in numbers, and are found on the upper layer of the fluid. Introduce at the upper layer a platinum loop, take out a small drop the size of a lentil seed, rub it on a cover, and allow it to dry thoroughly in the air; then stain, as previously described, with the fuchsin solution.

Post-mortem Tests.—To examine suspected intestinal contents, open the abdominal cavity carefully and ligate at two places with stout twine a piece of the ileum well filled with fecal contents, about three to four centimeters in length, and taken from near the cæcum. A double ligature should be applied at each end and the cut made between the two ligatures, so that the intestinal contents will not be spilled in the abdominal cavity. It is well also to cut out a piece of the intestine three to four centimeters in length from the upper portion of the ileum, and to lay the excised portions in ordinary water until ready for examination. The method is the same as has been described; that is, take a small piece of flocculent mucus the size of a pin's head, etc.

Gelatine stroke and stick cultures, and also potato cultures, can be made for examination. The spirilla also grow on blood serum and agar.

Cholera bacilli require for their growth a mild alkaline nutrient medium, and are very quickly destroyed by mineral acids. They do not develop readily in ordinary water, owing to the presence of other bacteria, which destroy them; they do develop, however, very rapidly in sterilized water.

YELLOW FEVER.

SYNONYMS.—*Febris flava; Bilious Remittent Fever* (Rush); *Kendall's Fever; Barbadoes Distemper; Indiæ occidentalis; Elodes icterodes; Typhus icterodes; Typhus tropicus.*

Definition.—Yellow fever is an acute infectious disease, characterized by a febrile paroxysm succeeded by remission, and a relapse, terminating in death or recovery, and associated more or less constantly with a yellow coloration of the skin, and less constantly with hemorrhages.

Historical.—The birthplace of yellow fever is unknown. It appeared in Barbadoes in 1647; prevailed in Jamaica in 1671; at St. Domingo in 1691; at Pernambuco, Brazil, in 1687 to 1694; in Martinique in 1690; in Boston Harbor probably in 1692; Philadelphia and Charleston in 1692; Rocheford, France, in 1694; and Philadelphia again in 1699, when a severe epidemic prevailed. Philadelphia was again visited in 1741 and 1762; New York in 1791, and Philadelphia in 1793, during which time there reigned one of the most frightful epidemics history records, 4040 dying out of a population of 40,000. After 1793 the disease prevailed more or less every year in the United States until 1805. From 1805 to 1820 the epidemics were limited or cases isolated until 1820, when Philadelphia was again severely attacked. After 1820 there was a period of comparative immunity, but not without cases and small epidemics in various cities of the United States until 1853, when there raged a violent outbreak through the southern cities of the United States. In New Orleans alone in that year nearly 8000 died. In 1867 and 1873 there were other epidemics of moderate severity, and in 1878 another severe epidemic appeared, chiefly in Louisiana, Alabama, and Mississippi, during which nearly 1600 died. Thus it will be seen that the United States as well as the West Indies have always been favorite seats of this still imperfectly understood and fatal malady.

Etiology.—The analogy between yellow fever and the other forms of contagio-infectious disease, in its origin, spread, and conditions, render it more than likely that it, in common with them, is the result of a specific organism. Yet by common acknowledgment none has as yet been isolated, although Domingos Fréire, of Brazil, Carmona, of Mexico, and Gibier have described organisms as possibly responsible. Nor is the contagium bearer known. In this respect the disease is more like typhus fever than like cholera and typhoid fever, where it is known to be the alvine discharge. The contagium is exhaled someway from the body of the patient, and may be carried in fomites to an indefinite distance,—it is said even by letter. Ordinarily it is disseminated through the air. The predisposing causes are much better understood. It is a disease of hot countries and hot weather. At the present day it is endemic only in the West Indies and the west coast of Africa. At one time it was so in New Orleans, U. S. A., but rigid hygienic regulations have excluded it, and it now appears only in the southern cities of the United States in periodic epidemics the result of accidental transmission, while

in northern cities it is almost unknown, being limited to isolated and accidental cases brought in ships from southern ports. Freezing weather terminates the activity of the germ, but does not destroy it, since it is revivified by return of warm weather. In accordance with these facts John Guitéras makes three areas of infection :—

(1) The focal zone, in which the disease is never absent, including Havana, Vera Cruz, Rio de Janeiro, and other Spanish-American ports.

(2) Peri-focal zone, or region of periodic epidemics, including the ports of the tropical Atlantic, in America and Africa.

(3) The zone of accidental epidemics, between the parallels of 45° north and 35° south latitude.

Its development and multiplication are also favored by filth. Hence the foul and confined air of ships is apt to be contaminated, and its tenacity is shown by the fact that a ship once infected can with difficulty be purified, the disease having been known to return again and again after the most careful disinfection. The densely populated and unhealthy portions of a city are favorite localities.

A very interesting fact in connection with yellow fever is its limitation to the sea and the sea-coast, as it rarely invades interior cities, or altitudes higher than 1000 feet (meters).

It attacks all races, both sexes, and all ages, except the very young. Yet it is through the young that the disease is maintained in a native population because protection is secured by a previous attack or long residence in a locality in which it is endemic, and it is the young who, as they grow up, furnish the pabulum for fresh cases. The negro and the creole, although not immune, are comparatively so. More men are attacked than women, because of their frequent exposure. Strangers are especially liable to attacks.

Morbid Anatomy.—Yellow coloration and hemorrhagic extravasations under the skin are present. The yellow coloration is a hæmatogenetic jaundice. The serum of the blood is red tinted, because of its containing dissolved hæmoglobin. The liver is the organ which has always been regarded as exhibiting the most characteristic change. It resembles the yellow of admixed coffee and milk—a *café au lait* appearance—as contrasted with the more bronzed appearance of the liver of remittent fever, though this appearance occurs in other diseases. I have seen it very strikingly reproduced in the liver of pernicious anæmia. The liver cells present various stages of fatty degeneration, with necrotic masses in and between the liver cells, described by George M. Sternberg. The kidney may exhibit cloudy swelling or even acute nephritis. Bacteria of various kinds are found in both organs.

The stomach after death contains more or less of the "black vomit," which is a mixture of transuded serum and altered blood pigment. The mucous membrane of the stomach is hyperæmic and more or less swollen.

Symptoms.—Yellow fever has a period of incubation of twenty-four hours to very rarely exceeding five days. It is usually three or four days. After this the onset is sudden, generally with a chill, promptly followed by headache and severe pain in the back and limbs. These symptoms may come while the patient is at work or appear at

night. There is fever which rises rapidly to 102° (38.9° C.) and even 105° F. (40.5° C.). The pulse corresponds. The skin is hot and dry, but less pungently so than in typhus. The face is flushed, the tongue is furred but moist, the throat sore, the bowels constipated, the urine scanty and often albuminous. There may be nausea from the beginning, but it is not until the second or third day that it is aggravated and the characteristic "black vomit" makes its appearance. This resembles an infusion of coffee, and deposits a sediment comparable to coffee grounds, and which consists of broken-down corpuscles and hæmatin. In the worst cases the vomited matter may be tarry in appearance and consistence. On the other hand, "black vomit" is not always present, being generally confined to the severe cases. In some it is watery or bilious. These are the phenomena of the febrile stage, or *stage of invasion*, which lasts from a few hours to two or three days.

Then follows a decline in the fever while the severity of the other symptoms also generally subsides, and this may be the beginning of convalescence in the mild cases. But in severe cases this second stage, or *stage of calm*, is of short duration, from a few hours to one or two days. Then the third stage, or *stage of febrile reaction*, sets in. The temperature now rises again, although the pulse may continue to fall; the nausea and vomiting return and the latter becomes hemorrhagic, and may be accompanied by abdominal pain. Black and offensive stools occur. Jaundice makes its appearance; the tongue becomes dry and brown and there may be bleeding of the gums—indeed, from all the mucous membranes. To albuminuria may be added hæmaturia. The strength rapidly fails, the pulse grows weaker, there is nervous trembling, suppression of urine, mental wandering, convulsions or stupor, and death.

Such, however, is not always the termination, even when there has been "black vomit." The symptoms may all gradually subside and the patient recover, although the jaundice may persist for a long time. In mild cases the calm stage may be succeeded by convalescence.

Diagnosis.—Yellow fever is most likely to be confounded with severe fever of bilious or malarial remittent type. Indeed, the resemblance is sometimes very close, especially when the latter is accompanied by hæmaturia. But the remission occurs earlier in remittent fever and the chill is of much longer duration, while the presence of Laveran's plasmodium in the blood settles the question in favor of the latter. Acute yellow atrophy of the liver is a disease more insidious in its approach and more febrile. The urine in acute yellow atrophy is loaded with bile.

Relapsing fever resembles yellow fever only in the fact of the relapse, but this occurs much earlier in yellow fever. The similarity of the mild forms of yellow fever to thermic fever has been emphasized by John Guitéras.

Prognosis.—Yellow fever is a grave disease, and in its severe forms one of the most fatal of the infectious diseases. The mortality ranges from 15 per cent. to 85 per cent. Among the dissipated, the wornout, the poor, and in hospitals the mortality is higher. It is less in the colored race. "Black vomit" is not necessarily a fatal symptom. Many malignant cases terminate in a couple of days.

Treatment.—There is no specific treatment for yellow fever and the symptoms are to be met as they arise. The practice quite general in the Southern United States to give an initial dose of castor oil is harmless and may be useful. This is followed by efforts to cause perspiration. Quinine is indicated on general principles. We may seek to stop vomiting by ice internally and externally and hypodermic injections of morphine. The hemorrhagic tendency may be combated by astringents, including iron. The failing strength is to be supported by alcohol and digitalis, the high temperature by sponging and cool baths. Nutrient enemata are to be relied on where vomiting is uncontrollable. Toward a specific purpose is the following prescription of Sternberg :—

℞. Sodium bicarbonate, ... 150 grains (10 gm.)
Mercury bichloride, ... ½ grain (.022 gm.)
Distilled water, ... 1 quart (a liter).
M. et S.—Three tablespoonfuls to be given every hour.

The mixture is antiseptic and antacid, and its object is to reach the specific germ in its supposed habitat, the intestine, and prevent its further development. Good judgment should be exercised in discriminating against the over-use of drugs.

Prophylaxis is more effectual than direct treatment of the disease. Pretty definite rules to this end have been laid down in recent times through the studies of Sternberg, who directs the following precautions :—

(1) Exclusion of the exotic germs of the disease by sanitary supervision at the port of departure of ships sailing from infected ports, and thorough disinfection at the port of arrival when there is evidence or reasonable suspicion that they are infected.

(2) Isolation of the sick on shipboard, at quarantine stations, and, as far as practicable, in recently infected places.

(3) Disinfection of excreta and of the clothing and bedding used by the sick, and the localities into which cases have been introduced or infected in any way.

(4) Depopulation of infected places, that is, the removal of all susceptible persons whose presence is not necessary for the care of the sick.

Those who through the force of circumstances are compelled to remain in infected districts should live temperate and wholesome lives, exercising care in bathing, eating, and drinking, avoiding all over-fatigue and securing sufficient sleep.

The same principles and method applied to the disinfection of the excreta and clothing and apartments as in the management of other infectious diseases should be applied also to those of yellow fever. See page 79.

Protective inoculations have been followed by less satisfactory results in the case of yellow fever than any other disease, though success for them has been claimed by W. L. de Humboldt as far back as 1854, Caromon in 1881, and Fréire in 1884.

MEASLES.

SYNONYMS.—*Rubeola; Morbilli.*

Definition.—Measles is an acute, highly contagious disease, characterized especially by a mottled eruption and naso-bronchial catarrh.

Historical.—Measles and small-pox are first recognized in the writings of Ahrun, a Christian priest and physician of Alexandria, A. D. 610–641. It was, however, first accurately described by Rhazes, A. D. 900, and Avicenna, A. D. 980–1037. Rhazes is accredited with distinguishing it from small-pox. It continued, however, to be confused with the latter disease as late as the middle of the seventeenth century. The two were clearly separated by Withering as late as 1792. The distinction of having separated the disease from scarlet fever has been awarded to Thomas Sydenham about 1665, but this separation is also said to have been made one hundred years earlier, that is, in 1563, by Forestus, of Holland. Rhazes and Avicenna described it under the name *hhasbah.* The term *rubeola* or its equivalent in Arabic is said to have been first used by the Arabian, Haly Abbas, in the latter part of the tenth century, but was replaced by the Italian word *morbilli*, meaning little disease, up to the middle of the eighteenth century, when Sauvages reapplied the name rubeola, which was adopted by Cullen and Willan. Within the last few years some English writers have reapplied the name morbilli. The disease has prevailed in Asia and Europe for centuries, and was imported into the United States with the first settlers.

Etiology.—This disease is in all probability due to a micro-organism, which, however, has not as yet been isolated, although micrococci have been found in the blood and tracheal mucus by Babes and by Klebs. More recently, 1892, P. Canon and W. Pielicke * found with considerable constancy a bacillus in the blood, the expectoration, and nasal and conjunctival mucus of cases of measles. Whatever it is, it is very virulent, since the disease is more unfailingly communicated to those unprotected by previous attacks than scarlet fever. Nor is the contagium bearer definitely known, but it is likely to be the nasal and bronchial discharges, and probably also the tears. The contagium has been transmitted by the inoculation of morbillous blood and nasal mucus, and it is most active when the breath is its medium. It is communicable by a third party and by fomites; though more active and unfailing than the contagium of scarlet fever, it is less so than that of small-pox. It is not, however, so tenacious as the causes of these. Measles is a disease of childhood, but adults often get it, and that very severely. Repeated attacks are possible, but as other eruptive affections resemble it and diagnosis is often careless, some of the repeated attacks may be thus explained. It is milder and rarer in sucklings under six months, while the age during which the disease is more commonly contracted is one to five years.

Morbid Anatomy.—There is no essential morbid anatomy of measles beyond the nasal and bronchial catarrh, and the signs of these

* *Berliner Klin. Wochenschr.*, Vol. XXIX, 377.

generally disappear with death. Where death occurs it is usually the result of complications, and the morbid anatomy of such is present. The most frequent is broncho-pneumonia, sometimes lobar pneumonia, and among the morbid phenomena are to be included sometimes those of collapse of the lung. In those rare instances of hemorrhagic or "black" measles there is the usual discoloration of hemorrhagic extravasation. Rarely also the morbid states of intestinal catarrh are found.

Symptoms.—The *period* of *incubation* of measles varies, but is commonly between seven and fourteen days. Rarely it is a day or two longer. A prodrome if present in measles is of short duration. It may be manifested by sneezing, fretfulness, chilliness, and feverishness, or, if the child is old enough to express itself, by headache. Then comes the initial or prodromal fever, a peculiarity of which is a remission on the

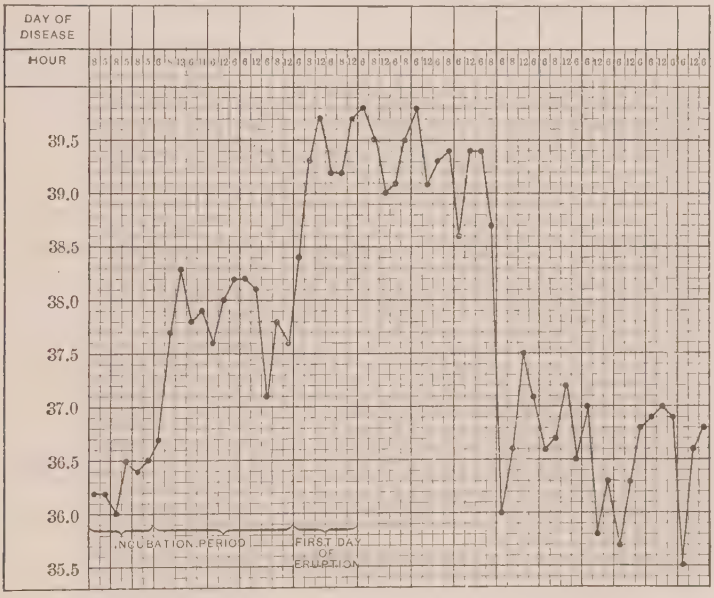


FIG. 11.—TEMPERATURE CHART OF MEASLES.—(*Eichhorst.*)

second day. This is shown by the appended cut from Eichhorst. But very early, and even almost suddenly, *coryza* with red and watery eyes and photophobia present themselves, closely followed by troublesome *cough* and corresponding feverishness reaching 103° and 104° F. (39.4° to 40° C.). Much less frequently than in scarlet fever is there *vomiting*, but the *tongue* is apt to be furred; the cough is sometimes croupy. Convulsions very rarely usher in the disease.

On the fourth day from the onset of the first symptom, the *eruption* makes its appearance, first in the face. It first appears in papules and blotches which coalesce more or less imperfectly, leaving sometimes islands of white skin between them. Under any circumstances the boundary between the eruption and the sound skin is uneven and crescentic. The eruption is somewhat raised above the surface, and the whole effect is to make the face appear swollen. This elevation of sur-

face at times becomes distinctly papular and even shot-like, resembling closely the papular stage of small-pox. In fact, this appearance has quite often led to the diagnosis of small-pox which twelve hours later had to be withdrawn. From the face, the eruption spreads to the neck, thorax, abdomen, and extremities. It is bright red, as a rule disappearing on pressure. Sometimes, however, even in mild cases, there are petechiæ, and in malignant cases the extravasations are extensive. At the same time the mouth and fauces are bright red in color and not infrequently there is diarrhœa, as though the eruption extended throughout the entire mucous tract as well as over the skin. At the maximum of the eruption there may be slight swelling of the cervical sympathetic glands. At the end of two or three days the rash fades gradually and a fine, branny desquamate occurs, easily overlooked. The fading takes place in the order of invasion.

The other symptoms described continue until the eruption begins to fade,—that is, on the fifth or sixth day, when they abate; though the *cough* often hangs on quite stubbornly, especially in scrofulous children, sometimes even persists as the catarrhal symptom of a tuberculosis, whose development seems peculiarly favored by the disease. Hence the cough of measles should never be slighted and early exposure should be prohibited.

It has already been intimated that a malignant form of measles sometimes occurs called also "black" measles, which is very serious, often, indeed, fatal. It is generally epidemic, occurs in institutions and camps, and its attack is characterized by subcutaneous extravasations of blood and hemorrhages from the mucous membranes.

Complications and Sequelæ.—These furnish most that is serious in the disease, and of these the most frequent and dangerous is catarrhal pneumonia or broncho-pneumonia, the bronchitis creeping into the smaller air tubes. The occurrence of catarrhal pneumonia seems to be favored by bad hygiene. Collapse of the lung is also prone to occur caused by an accidental valve-like plug of secretion. Broncho-pneumonia is recognized by the persistent and aggravated cough, the continued high temperature, and physical signs of a circumscribed pneumonia. More rarely lobular pneumonia supervenes and is recognized even more easily. In view of these possible complications, frequent physical examinations of the chest should be made.

Among the complications may be mentioned laryngitis, catarrh of the middle ear leading to suppuration and perforation of the drum, and chronic or intractable ophthalmic trouble. Ulcerative and even gangrenous stomatitis (*cancrum oris*) are met under unfavorable hygiene conditions.

Nephritis, although not often a complication of measles, does, however, occur, and I have met serious instances of such. Tuberculosis has long been recognized as a sequel of measles, yet it is not very common. Any of the varieties of pulmonary tuberculosis may be present. Even nervous lesions are reported, such as hemiplegia, paraplegia, neuritis, and myelitis.

Diagnosis.—Measles is easy of diagnosis; but the physician must not be too precipitate. Allusion has already been made to the possibility of mistaking it for small-pox, on account of the similarity of the

eruption in the early stage, an error which a few hours' delay would have averted. From scarlet fever there is sometimes difficulty, as there is occasionally slight sore throat and the eruption may be diffuse, while the difficulty is increased if there be glandular swelling in measles; but the catarrhal symptoms of measles are essential to it. The mildest cases are probably those which give most trouble. The distinction between measles and rubella is sometimes more difficult, but this will be considered when treating of rubella.

Typhus fever and measles have been confounded, and it must be admitted that in the asthenic variety of measles the eruption may resemble that of typhus fever. It will be remembered that the eruption of typhus is described as "rubeoloid." Confusion is further favored by the fact that the eruption occurs at about the same time in each disease.

Prognosis.—The vast majority of cases of measles get well. It is only in epidemics of the malignant form, in hospitals, camps, and foundling asylums, that death occurs as a direct result of the disease. In these the mortality is sometimes very high. Epidemics among the aborigines in North and South America, in the Mauritius and Fiji Islands, and in the Confederate Army in the War of the Rebellion in America were of signal fatality. Other deaths are due to the complications, especially pneumonia. Out of 24 fatal cases collected by Pott, 21 died of bronchopneumonia and pneumonia, and three of croup.

Treatment.—After surrounding the patient by a uniform warm temperature, best secured in bed, the treatment of measles is mainly that of the fever and the cough. The former is sufficiently treated by the simple diaphoretics and febrifuges, such as citrate of potash and sweet spirit of nitre, or tincture of aconite. The latter is efficient and tasteless. The cough calls for positive anodyne measures, of which for children paregoric is the best because the safest. Laudanum or deodorized tincture of opium may be used in smaller doses, but not morphine. They may be combined with the febrifuges just mentioned. It is comparatively rare that cool sponging is needed to reduce the temperature, but cold water should be allowed *ad libitum*. I recently knew a case of measles to receive the cold tub-bath treatment under the impression that it was typhoid fever. The rash came out brilliantly at the proper time and the case did splendidly. Complications should be treated as they arise. Stimulants and tonics are necessary in the adynamic form. Where the cough is prolonged cod-liver oil is a valuable remedy. Watchfulness during convalescence is more important than is supposed by many, and carelessness and indifference are sometimes responsible for unfortunate results.

It sometimes happens that the appearance of the eruption is delayed or "suppressed." Under these circumstances the hot pack is very effectual. The child is wrapped in flannel wrung out in hot water and then enveloped in a macintosh. Copious perspiration soon sets in, the eruption appears, and general reaction commences.

I have never seen any good reason for isolating measles as usually occurring in families. The disease is so mild in children, and so much more serious in adults, that I think it is desirable that all the children of a family should have it as soon as possible.

RUBELLA.

SYNONYMS.—*Rötheln; Rubeola*; German Measles; Rubeola notha; Epidemic Roseola; False Measles; Hybrid Measles; Hybrid Scarlet Fever.*

Definition.—Rubella is a mild, acute, contagious disease, characterized by a punctiform rash which fuses into patches less markedly crescentic than those of measles. It is less markedly contagious than measles or scarlet fever. There is often slight sore throat, more rarely mild catarrhal symptoms, and trifling fever. Many so-called second attacks of measles and scarlet fever are attacks of rubella.

Historical.—The existence of rubella as an independent and separate infectious disease was for a long time disputed, some regarding it as a variety of measles and others as a mild form of scarlet fever, although as far back as 1752 Bergen, a German physician, claimed for it a distinct individuality. A sufficient foundation for such claim was not, however, advanced until 1815, when Maton, an English physician, substantiated that, while one attack protected ordinarily against its own recurrence, it did not protect its subject either from measles or scarlet fever, while an attack of either of the latter diseases did not protect from rubella. A few held out for the original views, among whom was the eminent Hebra, but there seem to be none at the present day who deny that it is an independent disease. The name rubella was first suggested by Veale in 1866.

Etiology.—The relation of the disease seems rather closer to measles than scarlet fever, and may be said to bear to it the same relation as varicella to variola. It is, however, much less contagious than measles or scarlet fever. It affects children chiefly, very rarely adults, sucklings less frequently than school children, because the latter are more exposed to contagion. Isolated cases occur, but it is apt to prove in large cities epidemic, which are sometimes widespread.

Symptoms.—After a *period of incubation*, ranging from two to three weeks, the disease sets in, as a rule, with no distinctive symptoms of invasion prior to the eruption. There may be chilliness, moderate muscular pain, mild catarrh, and slight fever, with temperature barely reaching 100° (37.8° C.), for a day or two previous to the eruption. More frequently a pale rose color—an indistinct macular *eruption*—is first noted. The papules are scarcely elevated and vary in size from a pin's head to a split pea, the smaller being more numerous, much smaller than the papules of measles. They may, however, fuse and form large, irregular patches, with little or no disposition to form small crescentic-shaped groups like those of measles. The rash may appear as late as the second day, rarely on the third.

Two types of the spread of the eruption are possible. In the one it appears almost simultaneously all over the body, reaching its maximum by the second day, after which it rapidly fades. In the second mode of invasion the rash appears first on the face and extends rapidly thence all

* It is unfortunate that the Germans have selected for their technical term for this affection the word *rubeola*, which is the word used in English for measles.

over the body, reaching the hands and feet last, and beginning to fade on the face and trunk before attaining its maximum on the extremities, or even before it appears there at all. Thus it has a wave-like course, reaching its maximum in twenty-four hours, when it begins to decline rapidly. It is, therefore, of shorter duration than the eruption either of measles or scarlet fever. It may terminate in a branny desquamation, less evident even than that of measles.

The most constant symptom after the eruption is the *sore throat.* It varies in severity, but is for the most part mild, never becoming ulcerative. It is really, perhaps, the eruption in the throat. Somewhat less constant than the sore throat, though it varies somewhat in different epidemics, is swelling of the lymphatic glands of the neck, especially the superficial cervical and post-cervical glands. This swelling is present during the eruptive stage and may occur even earlier. Its possible, though rarer, occurrence in measles also is to be remembered.

The remaining symptoms of rubella are not marked or distinctive. There is little or no constitutional disturbance, and, as already mentioned, rarely any *fever* above 100° F. (37.8° C.), although 102° F. (38.9° C.) and even 103° F. (39.4° C.) have been noted. There may be slight catarrh, watering of the eyes, and running at the nose, all much less marked than that of measles. There are no complications, as a rule, though albuminuria, nephritis, pneumonia, colitis, and icterus have been reported, but it would seem as though measles or scarlatina must have been mistaken for rubella in these cases.

Diagnosis.—Such are the symptoms of a typical case. Unfortunately, there are many deviations, some approximating measles and some scarlet fever, differing from either mainly in mildness. The absence of decided catarrhal symptoms, the earlier appearance of the eruption, its more diffuse character, and the swelling of the lymphatic glands are its chief differences from measles. The careful studies of J. P. C. Griffith * show the latter of less significance than is usually supposed. The course of the eruption differs also, measles lasting longer. The same mildness and absence from fever, with the more distinct mottling, distinguish it from scarlet fever. In rubella the symptoms of invasion are all very much milder than in either measles or scarlet fever, even mild cases of the latter.

Prognosis.—The prognosis of rubella is invariably favorable.

Treatment.—Very little if any is required except rest in bed. A simple febrifuge with potassium chlorate may be useful.

SCARLET FEVER.

SYNONYM.—*Scarlatina.*

Definition.—Scarlet fever is an acute contagious disease, especially characterized by faucitis and a diffuse scarlet eruption, terminating in more or less membranous desquamation.

Historical.—Although it has been claimed that the pestilence of

* "Differential Diagnosis of Rubeola and Rubella, with Special Reference to Enlargement of the Glands of the Neck," University Med. Magazine, June, 1892.

Thebes, 600 B. C., and the plague at Athens, 430 B. C., were each epidemics of scarlet fever, no accurate knowledge of this affection as a separate disease was obtainable prior to the seventeenth century, when Sydenham and his contemporaries described it in a manner which permits its easy recognition as the scarlet fever of to-day. It pervades the Old World everywhere, having been recognized in England in 1661, Scotland in 1716, Germany and Italy in 1717, Denmark in 1740, and was introduced into North America by shipping in the year 1735. It did not, however, reach South America until 1829, Iceland 1827, and Greenland so late as 1847.

Etiology.—The organism that causes scarlet fever has not been isolated. Streptococci have been found in the blood and *post mortem* in the glands and the kidneys. Whatever the agency, it is the most tenacious of all the contagia, retaining its power to infect for at least a year after the occurrence of a case. It is especially difficult to dislodge from organized substances, such as bedding, clothing or straw, letters and books, and the disease has been communicated to new comers even after an infected apartment has been thoroughly cleaned and fumigated with sulphur. Physicians have doubtless conveyed it, and the beard and hair are contagium bearers more frequently than is supposed. Hence physicians should not wear long beards, and nurses before passing from one case to another should disinfect their hair as well as the rest of their body.

While the contagium itself has never been isolated, there is every reason to believe that the bearer is the exfoliated epithelium. Hence it is not until desquamation takes place that the disease is communicable, and the ease with which the scaly particles are disseminated through the air and the tenacity with which they adhere to textures readily explain the communicability of scarlet fever and the difficulty in destroying its cause. On the other hand, until the eruption makes its appearance the disease cannot spread. Accordingly, it is not likely to be communicated to those exposed prior to this stage. Therefore, children promptly removed from association with the disease after its discovery and kept apart generally escape it.

The route of communication is probably for the most part the respiratory tract, although the alimentary canal may also convey it. In confirmation of this is the fact that in two instances, at least, in England, it has been conveyed by milk, the milk having been infected by being exposed for a time in an apartment occupied by patients. In one instance the disease appeared in six out of twelve families supplied from the infected source. The readiness with which milk absorbs volatile substances kept in the same refrigerator apartment and retains their flavor is quite in accord with such transmission.

Children of either sex are more subject to the disease, because a single attack, as a rule, protects against a second. Infants, however, even under exposure, are less liable to the disease, and it would seem, too, that adults who have escaped exposure during childhood are less liable. I have never had scarlet fever, and have been exposed many times to the most virulent forms. In my own experience the disease is most common about the age of seven.

Morbid Anatomy.—There is no morbid anatomy peculiar to scarlet fever. The eruption fades after death unless there happens to be hemorrhagic extravasation. There may be lesion the result of ulcerative destruction in the neighborhood of the throat. The intensity of the fever sometimes produces granular fatty change in muscles, which is pronounced in the case of the heart; also cloudy swelling in the cells of the kidney and liver. Glandular swellings present at death maintain themselves afterward. The morbid anatomy of the complications and sequelæ is appropriately considered under the diseases constituting them. The primary attack is not always protective; second and third attacks are reported. But here, again, careless diagnoses and defective memory are responsible for a certain number.

Symptoms.—The *period of incubation* varies greatly. It is sometimes as short as twenty-four hours, and again as long as twelve days. More frequently, perhaps, two to four days. At the end of this time there is usually a very short prodrome, sometimes none at all. Vomiting, occurring either as an initial symptom or a couple of hours later, is often present; more rarely a convulsion, still more rarely a chill. Sore throat is early complained of and high fever is conspicuous. The face is flushed, and the temperature rapidly rises to 103° F. (39.4° C.), 105° F. (40.5° C.), and even 108° F. (42.2° C.), and the pulse to 110, 120, or more.

The *eruption* appears, as a rule, on the second day, and it generally happens that if it is not present at the physician's first visit it is sure to be found at his second. Its striking character is its uniform redness. It is like a diffuse, broadly spread blush, appearing first upon the neck and chest and extending thence rapidly over the whole body, so that at the end of the third day it has completed its invasion. The appearance of a child covered with a frank scarlet-fever eruption is very characteristic. It has been well compared with that of a boiled lobster in its bright redness. It is further characterized by the readiness with which it disappears on pressure and the promptness with which it returns after the pressure is removed. It is, however, no sooner complete than it begins to fade, and does so with great rapidity in the order of invasion. The eruption is not, however, always thus typical, and presents every degree between that described and that which is barely recognizable. It is also at times more "patchy," but never presents the crescentic or otherwise irregular edges or mottled appearance of the eruption of measles. In the lower and more malignant forms the redness is of a darker or dusky hue, and in the worst of these petechiæ are present. Vesicles are even found with turbid contents, producing *scarlatina miliaris*. The eruption is sometimes entirely absent from the face; hence no conclusion should be based upon its inspection only. The thorax and inner surface of the thighs are more favorable sites. The eruption, when severe, is constantly accompanied by an itching or burning more or less intense, and there is a feeling of slight roughness at times.

The *tongue* is red at the edges and tip, furred at the centre, but through the fur stand out the papillæ in distinct points, producing the appearance known as the "strawberry tongue," which is regarded as more or less characteristic. The rest of the mouth, including the roof and the palate and tonsils, is bright red, as though the eruption extended

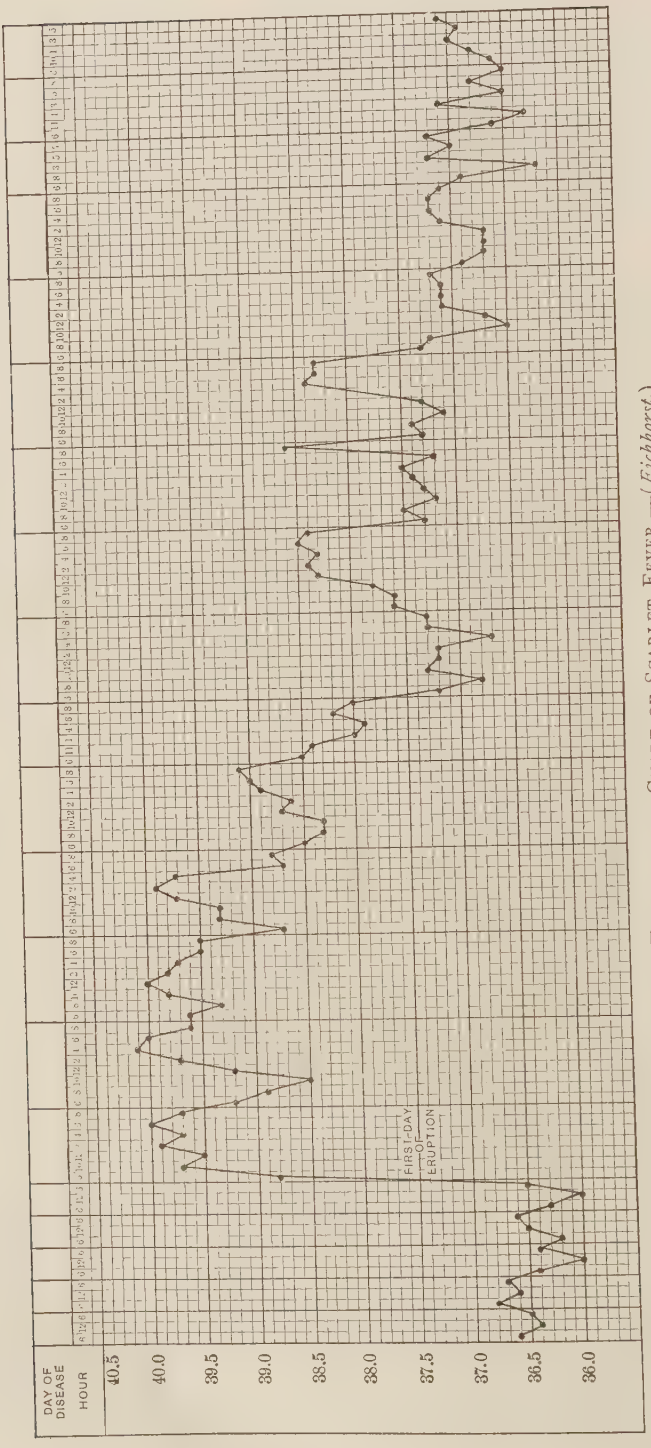


FIG. 12.—TEMPERATURE CHART OF SCARLET FEVER.—(*Eichhorst.*)

to it, as it doubtless does. After a few days the fur desquamates, leaving the tongue red and raw-looking.

With the abatement of the eruption comes *desquamation,* and it is generally proportionate to the intensity and extent of the former. When the eruption is slight the little scales are scarcely noticeable, and the closest examination is necessary to discover them, while where there is a vivid and extensive eruption the amount of desquamation is enormous. Glove-like casts of the fingers, including the nails, are sometimes exfoliated, and the bed contains each day numerous flakes of epiderm which have come off, while many days are required for complete separation of the dead skin. Great care should be taken in gathering it up, as in the desquamation resides the contagium. On the other hand, when slight it should be carefully sought for, as it has great diagnostic value. At the same time it should not be regarded as something peculiar and confined to scarlet fever, for every dermatitis is followed by desquamation, as especially exemplified in the exfoliation which follows an attack of erysipelas on the face or irritation by iodine or mustard.

The *urine* from scarlet fever proper is like that of fever cases generally, scanty, high colored, and precipitating uric acid and urates on cooling. The chlorides are diminished during active fever.

The duration of simple uncomplicated scarlet fever ranges from three to fourteen days, according to the degree of severity. Its decline is, however, gradual as compared with the suddenness of onset.

Such is a general picture of scarlet fever in its simple, uncomplicated form, so characteristic that early in its history it received the name *scarlatina simplex;* owing to further combinations of symptoms, there have been added three other varieties, the anginose form, or *scarlatina anginosa,* the malignant form, or *scarlatina maligna,* and the *hemorrhagic* form.

In the *anginose* variety the throat symptoms are conspicuous and severe. In no well-developed case is there an absence of throat redness. On the other hand, there is intense soreness with swelling of the fauces and tonsils, giving rise to extreme dysphagia. The neck may be so swollen as to fill up the space beneath the jaw. There may be a false membrane involving the fauces, the posterior pharynx, the nasal cavities, the trachea and bronchi. The throat may present all the features of a severe diphtheria. Abscess and destructive ulceration may result, which may proceed even to perforation of the carotid artery and rapid death ensue therefrom. The inflammation almost certainly ascends the Eustachian tubes, producing severe ear symptoms. The false membrane is usually the result of the intensity of the inflammatory process, due to the specific cause of the disease, and not that of diphtheria, but there may be true diphtheritic membrane containing the Loeffler bacillus. Especially is this true of the cases in hospitals for infectious diseases. The *streptococcus pyogenes* is perhaps the most frequent cause of the throat inflammation. It has been found also in the skin, the blood, and the glandular organs in fatal cases. Scarlet fever has, indeed, been called a streptococcus infection. Follicular tonsillitis may also be one of the forms of sore throat.

In the *malignant* variety there is an overwhelming intensity of the cause, which may result in almost immediate prostration and death of the

patient, giving no time for the development of the usual symptoms, or these may be so feebly manifested that they present no distinctness. When the disease is not immediately fatal, there is intense adynamia, the heart and pulse sharing it. The breath is rapid, the capillary circulation is feeble, the skin dusky, the eruption imperfectly developed, the temperature is very high, reaching 105° to 108° F. (40.5° to 42.2° C.), there is delirium, which may pass over into coma, and convulsions may occur. The pulse ranges from 120 to 150.

In the *hemorrhagic* form there are more or less extensive hemorrhagic extravasation, epistaxis, and hæmaturia. It attacks, for the most part, the feeble and badly nourished, and, like the previous variety, is almost invariably fatal.

Epidemics of scarlet fever vary greatly in severity. In some all the cases appear to be mild, in others all are of extreme severity. Families of children may be exterminated. Again, a mild case may give rise to one of the most intense forms.

Complications.—*Acute nephritis* is the most frequent complication of scarlet fever. It makes its appearance usually after desquamation, is more or less complete in the second, third, or fourth week. A small albuminuria which is common at the height of the fever is not to be confounded with that of nephritis, and probably does not predispose to it, although the cells lining the tubules are at this stage in a state of cloudy swelling. The rationale of its production is not precisely understood. It is commonly ascribed to cold or a draught of air upon the skin, which is young and tender after the desquamation. But when it is remembered that the mildest cases are as susceptible as the most severe, and probably more so, and that children have been found barefoot in the street with the eruption upon them, and yet have escaped the Bright's disease, it must be admitted that we do not know all about it. The fact that the disease is usually more severe the earlier it appears would go to show that the specific poison has something to do with it. It is true, too, that with the skin functionally dead the complemental work thrown upon the kidney increases its susceptibility to the ordinary causes of nephritis, of which cold is one. It is to be remembered also that other diseases in which the skin is seriously affected predispose to nephritis. This is preeminently true of burns and scalds.

However it may be brought about, the result is generally a typical example of parenchymatous or tubal nephritis, although instances of interstitial inflammation are also found. Every grade of severity is met with, but early recognition increases our power to control this severity. The majority of cases thus recognized get well, and I have known recovery to take place after suppression of urine has lasted for a week. The clinical picture is that of acute nephritis otherwise caused, and its consideration may be deferred until that disease is studied. This complication was formerly often overlooked, but in modern times cases are more closely watched for it. The possibility of Bright's disease without albuminuria must be borne in mind.

Adenitis producing a moderate degree of glandular enlargement occurs in almost all cases of scarlet fever, but in severe cases it becomes a painful and grave complication. A majority of cases subside, but some

go on to extensive and destructive suppuration, of which I have known ulceration through the carotid artery a consequence.

Arthritis ensues in a certain number of cases and closely resembles that of acute rheumatism. The term rheumatism is justified as much as the term gonorrhœal rheumatism, and no more. Each is the result of the specific cause of the disease and not of the cause of rheumatism. It occurs usually at defervescence and recovery is almost invariable. Suppuration in the joint has, however, occurred.

Otitis is one of the most serious and permanently harmful of the complications. It is commonly considered the result of an extension of inflammation from the throat through the Eustachian tube to the middle ear and is associated with the streptococcus. I have known it to occur after recovery was supposed to have taken place immediately after a child had been sitting on a cold step. Suppuration and perforation of the membrane of the tympanum are common, and more rare is destructive suppuration of the mastoid cells. As a consequence of one or both of these it almost always leaves impaired hearing or total deafness. The facial nerve may become involved in the disease of the labyrinth, producing facial palsy, while thrombosis of the lateral sinuses may be another result of the same condition. Meningitis and death may be later consequences.

Various *nervous affections* develop as rare complications. Among these may be mentioned chorea, convulsions, hemiplegia, and Osler mentions two cases of progressive paralysis of the limbs due to ascending spinal paralysis or multiple neuritis and subacute ascending spinal paralysis as coming under his observation.

Of *thoracic complications* endocarditis and pericarditis not infrequently develop during convalescence from scarlet fever. Endocarditis is not always discovered, and a few unexplained chronic valvular defects may have originated in this way and thus be accounted for. Pericarditis is less likely to be overlooked. Mycotic endocarditis is not as frequent as might be expected from the virulence and widespread character of the responsible germ. Pleurisy may also occur, and more rarely pneumonia.

Diagnosis.—The diagnosis of scarlet fever is easy if the symptoms are well developed, for it is the mild cases which escape detection. In the absence of the eruption in a distinctive form it is sometimes impossible to aver the presence of the disease. If there be doubt as to the eruption, close watching will sometimes discover signs of desquamation in the shape of branny scales beneath the underclothing or in the stockings. In the absence of this the question must sometimes remain forever unsettled. At others the development of a nephritis unfortunately sets the matter at rest. If there has been exposure to the contagion, it is best to regard every case of sore throat as a possible case of scarlet fever and treat it accordingly. While the throat affection of diphtheria closely resembles at times that of scarlet fever, where this symptom is at all conspicuous in scarlet fever the eruption is not generally wanting, or is, at least, present to such extent as to permit recognition of the disease. The fact that the one or the other of the two diseases is prevailing may settle the question. It must be admitted, too, that the two affections

may succeed each other, and even, perhaps, coexist, both events being, however, exceedingly rare.

The coryza and cough in measles characterize the stage of invasion, while the eruption occurs later than in scarlet fever. When it does come it is very different, being at first, at least, in patches bounded by irregular and crescentic outlines, more uneven and elevated, and is conspicuous in the face, where the scarlet fever eruption is faintest. The absence of sore throat is distinctive of measles, though its occasional presence in mild degree must be admitted.

Rötheln, or rubella, has an eruption more like that of scarlet fever than is the typical measles eruption, but it is not usually followed by desquamation. There are no uncomfortable throat symptoms and the constitutional disturbance is much less. It is possible, too, that these affections may succeed each other, as is true of real measles and scarlatina.

Acute exfoliating dermatitis resembles scarlet fever during the eruption, but the exfoliation in the latter is not like that of scarlet fever. As in erysipelas, it has more the appearance of scales and crusts before it is thrown off, and there is more apt to be a moist surface left behind, followed by a second exfoliation. There are no throat symptoms and the tongue characteristic of scarlet fever is wanting. The eruption of belladonna, both on the skin and throat, resembles that of scarlet fever, but it is of short duration and without constitutional symptoms.

Prognosis.—The prognosis of scarlet fever varies greatly. There are epidemics of great severity, in which the mortality is large, and certain fulminating cases are beyond treatment. Yet most physicians of large experience in surveying their work will recall that the percentage of deaths in their scarlet fever cases has not been large, and that it has been greatest among the very young. The percentage of deaths is put down at from five to ten per cent. in mild epidemics and 20 to 30 per cent. in severe ones. The mortality is greater in hospitals than in private practice. In the fulminating cases death takes place before a chance for treatment is offered; but in the next grade of cases, characterized by high temperature and severe throat symptoms, a survival of five or six days generally means recovery, unless the supervening complications carry off the patient. Among these nephritis and glandular swelling passing over to abscess are conspicuous, but even of those so afflicted a majority recover.

Treatment.—The treatment of scarlet fever is, in the main, a symptomatic one, associated with a vigilant nursing which will guard against complications. The patient should be isolated, if possible at the top of the house, and all communication with those of the family who have not had the disease interdicted. The temperature of the room should be uniform, while effective ventilation should be secured. The diet should be liquid as long as the fever persists, and the best of all liquids is milk, though light broths are allowable with an abundance of water.

If the fever is high, say above 103° F. (39.4° C.), cool sponging may be resorted to, but it is to be remembered that high temperature in this disease is of short duration and incapable, therefore, of the mischief it may cause in the long-continued febrile diseases like typhoid fever.

Very high temperature, such as 105° F. (40.5° C.), with meningeal symptoms may require the tub-bath or cold pack, but the temperature of the former should not be as low as that for typhoid fever. It is safer to put a patient in a bath at 90° F. (32.2° C.) and gradually reduce the temperature. The warm bath allays the irritation of the skin, but this is as well accomplished by inunction with cold cream or sweet oil, and this unguent is important for another purpose as soon as desquamation takes place, to keep the scales from flying about and spreading the contagium. An ice cap may be applied to the head if the temperature is high, and especially if there are head symptoms. While cool applications are allowable during fever, they are positively contraindicated in its absence, as they may act in the development of complications of nephritis and otitis.

Fever is best controlled by these measures, but it is desirable to give medicines which tend to the same purpose, especially if they dispose to diuresis as well. Hence the officinal solution of citrate of potassium or of the acetate of ammonium combined with the spirit of nitric ether with a little flavoring syrup is useful.

The throat symptoms require to be treated according to the degree of their severity. Iron and potassium chlorate may be added to the above mixture. Constipation should be guarded against and frequent aperients are indicated. If more active local measures are needed, the throat may be sprayed frequently with peroxyd of hydrogen one to three, or with a weak bi-chloride of mercury (one to 5000) solution, or carbolic acid spray one to 60. The first is the best. Cold water applications and even ice to the exterior of the throat are very comforting to the patient. Very efficient and soothing is a bandage for the throat with pockets opposite to the tonsils, into which pieces of ice are placed and the whole covered with a dry towel; or little India rubber ice bags may be similarly used. In adynamic cases stimulants and restorative treatment in general are indicated. Due regard should be had to the tendency of the disease in severe forms to produce degeneration of muscle and the liability of the heart to share in this.

The proper treatment of the throat tends to save the ear, but should the middle ear become involved the membrane should be watched daily, and if the tension is extreme, perforation practised, even more than once, if needed. Too little attention has been paid to this complication, and if the circumstances permit, an aural surgeon should be called in.

The *prophylaxis* against *nephritis* should be most careful. Whatever may be the immediate cause of the renal involvement, it is certain that cold often becomes its exciting cause. Hence the patient should be scrupulously guarded against drafts, and tedious as it may sometimes seem to mother and child, "six weeks in the room" is a precaution which will avert many a case of nephritis. In addition to the milk diet, which is an efficient prophylactic against nephritis, I am in the habit of giving a moderate dose of digitalis, say three to five minims (.333 to .666 gm.), two or three times a day to aid in maintaining a free movement of blood through the kidney.

The treatment of complicating nephritis is the treatment of that affection under other circumstances, and the reader is referred to the appropriate section for it.

DIPHTHERIA.

SYNONYMS.—*Membranous Croup; Angina maligna; Angina membranacea; Cynanche contagiosa; Diphtheria faucium.*

Definition.—Diphtheria is an acute contagious inflammatory disease due to inoculation with the Klebs-Loeffler bacillus, and especially characterized by the formation of false membrane and by secondary constitutional infection. It may attack any mucous membrane and even the skin, but as usually employed the term means diphtheritic inflammation of the oral, faucial, nasal, laryngeal, tracheal, or bronchial mucous membrane. The term *diphtheroid* is applied to such membranous inflammations as are not due to the Klebs-Loeffler bacillus.

Historical.—Diphtheria was certainly recognized by Aretæus, of Cappadocia, who lived in the latter part of the first century and the early part of the second, and by Galen in the latter part of the second century. Paralysis of the soft palate was also recognized as one of the consequences of diphtheria in the fourth century by Coelius Aurelianus, in the fifth or sixth by Ætius,—and from that time to the present medical literature teems with mention of cases and epidemics, although one age seems frequently to have forgotten what another had learned. Diphtheria appeared in this country, in New England, in the seventeenth century. An admirable account by Samuel Bond appeared in the "Transactions of the American Philosophical Society" at Philadelphia in 1770. We owe the name by which the disease is now generally known to Bretonneau, who applied it in a paper read before the French Academy of Medicine in 1821, wherein he declared, also, that "Angina suffocativa," "Cynanche maligna," "putrid" and other forms of sore throat were one and the same thing.*

Etiology.—The specific organism which by common consent at the present day is the cause of diphtheria is the so-called Klebs-Loeffler bacillus, a bacillus non-motile, slightly bent, with rounded ends, 2.5 to 3 μ in length, and from .5 to .8 μ in thickness. It stains readily by Loeffler's methylene alkaline blue in cover-glass preparations and sections. Its cultures in blood serum are small, round, greyish-white colonies that are characteristic. These, with the clubbed ends of the bacillus and clear spaces in its interior, giving it an appearance as if broken, suffice for its recognition. It grows on all the usual culture media, but ceases to grow below 20° C. (68° F.). If inoculation cultures are practised on the lower animals, the nature of the virus is declared by the exudation, the bacilli, the swelling of adjacent lymphatic glands, and the invariably fatal results of such inoculation. The bacillus produces in its growth a potent toxic substance or toxalbumin, the absorption of which from the seat of local infection produces the general symptoms of the disease, which are therefore due to this toxin and not to an invasion of the blood by the organism producing it. This toxin is an albuminous substance, but its composition is unknown. When injected into animals, it produces

* The history of diphtheria is one of the most interesting chapters in medicine, and is more fully considered in the classic paper of Abraham Jacobi in the "System of Medicine by American Authors," Philadelphia, 1885.

paralysis, nephritis, and albuminuria. Roux and Yersin were the first to show, in 1888, the pathogenic property of cultures which had been filtered through porcelain.

The successful implantation of the bacillus of diphtheria is, however, dependent on various circumstances. Certain temporary states of the individual doubtless favor it, while others retard it. While general weakness or feeble resisting power may be one of these, it is likely also that purely local states, such as uncleanness of the mouth, teeth, and fauces, as well as chronic inflammatory conditions, may act as predisposing causes. Enlarged tonsils and nasopharyngeal catarrh predispose to it. It has been shown that there are different degrees of virulence in the contagious organism itself.

The bacillus of diphtheria is associated with other pathogenic bacteria, such as *streptococcus pyogenes* and *staphylococcus albus* and *aureus*, *micrococcus lanceolatus*, and *bacillus coli communis*, which are probably responsible for suppurative processes often associated, as well as for certain deep-seated inflammatory conditions, and certain forms of pseudodiphtheria, which often complicate the disease and are sometimes mistaken for it. The streptococcus is probably the most active.

It was formerly believed that defective drainage, and to a less extent also the upturning of soil, are conditions favoring the production of diphtheria, but such views are not sustained by modern studies. On the other hand, army statistics seem to show that foul air causes simple follicular sore throat, which in seasons of epidemics makes an excellent nidus for the growth of the diphtheria bacillus. The contagion is communicated, as a rule, through the air and not by fluids ingested, although epidemics have been traced to milk in which the bacillus multiplies. In the vast majority of instances the source of the contagion is the throat or nose of another individual affected, whence it is propelled by acts of coughing or expectoration. Hence it happens that the physician and nurse are not infrequently infected. Perhaps in this disease, more than any other, excepting typhus, are doctors and nurses the victims of contagion, as there is no way of securing immunity from it. Much may, however, be done to secure protection by caution during such ministrations, as by keeping the mouth closed and carefully cleansing the hands after contact. The practice of examining throats through a plate of clear glass is a further protection against inoculation of the examiner. The contagion is less tenacious than that of scarlet fever, but is still highly so, having been found to live on blood serum for one hundred and fifty-five days, dried on silk threads one hundred and seventy-two days, and in gelatin for eighteen months. It has been found on a child's toy which had been kept in a dark place for five months, and in the hair of nurses. It resides also in the healthy throats of immune persons, in simple catarrhal angina without membrane, and in simple lacunar tonsillitis. Whence it is plain how the disease may arise without apparent cause in certain sporadic cases.

It is believed by some that diphtheria affects the lower animals, especially the cat, and may be transmitted from them to children. It is said, also, that such an affection attacks calves and heifers, and is from them communicable to man.

The disease is much more common in children than in adults, though no age is exempt. It is rare in very young children, and more girls are attacked than boys. Jacobi, whose experience has been very large, has seen only three cases in the newly-born. Several cases in children about six months old have come under my notice. Epidemics vary in severity, and winter is the season in which the disease is most prevalent. While crowding in cities favors it, it is often widespread and virulent in the country.

Morbid Anatomy.—The morbid anatomy of diphtheria consists, on the one hand, in the presence of the false membrane and of the more ordinary phenomena of inflammation, most of the latter of which disappear after death; and, on the other, of the deep-seated ulcerative processes which sometimes result; and of the results of the complications and sequelæ, the most common of which are broncho-pneumonia, fatty degeneration of the myocardium, nephritis, and more rarely endo- and peri-carditis, and the articular inflammation. The paralyses do not furnish palpable morbid results.

Under morbid anatomy the constitution of the false membrane is suitably considered. At its first appearance it is yellowish-white, but later may assume a greyish hue. Whether superimposed on a mucous membrane, or set into it as in a frame, depends much upon the character of the epithelium with which the surface is normally covered. To squamous epithelium the membrane is more deeply and thoroughly attached; to columnar epithelium, such as lines the larynx or bronchi, it is more loosely adherent, but in both situations it tends to become looser with the lapse of time.

The membrane itself is to-day considered a product of what is known as coagulation-necrosis, our knowledge of which is based on the studies of Wagner, Weigert, and especially of Oertel. The mechanism of its production is as follows: The diphtheritic poison infiltrates the wandered-out leucocytes and the epithelial cells of the part, especially the more superficial, causing first their death and then a hyaline transformation, and simultaneously coagulation. The resultant is a plate of necrotic tissue and coagulated fibrin. Hence the word coagulation-necrosis. It presents, also, a laminated structure, probably due to the involvement of successive layers of tissue and wandered-out cells. If forcibly separated, especially when recent, it is apt to leave a bleeding surface, on which new membrane is generally promptly deposited. The process proceeds from without inward, and, though usually superficial, may extend more deeply, invading lymphatic glands and adjacent tissue, producing foci of necrosis, which may be extensive. Blood-vessels may also be invaded, especially the capillaries. Bacilli are everywhere present, but they do not directly produce the mischief. It is the toxin which they generate. The same results may be produced experimentally.

Inflammatory membrane of this kind is not the product of the toxin of diphtheria only. Any intense irritant is capable of producing it. Such are the corrosive poisons like nitric acid and ammonia, although the necrotic product is here partly the result of the direct action of the agent itself on the tissue. Similar in its effect is the toxic agent of scarlet fever, which often produces a pseudo-membranous angina difficult to

distinguish in its coarser characters from that of diphtheria, but in which is not as a rule found the Klebs-Loeffler bacillus. In this membrane are, however, found staphylococci and streptococci, the latter especially. The membrane may be truly diphtheritic if this disease happens to be prevailing also.

It is even likely that a form of pseudo-membranous angina exists, due neither to the toxin of scarlet fever nor diphtheria, but in other respects similar to the membranes thus caused, and accompanied by a constitutional disturbance much less marked. It is to this that the term *diphtheroidal* is peculiarly applicable, but it is applied to any one of the varieties of membranous inflammation not due to the Klebs-Loeffler bacillus. Some cases of so-called diphtheroidal membrane are probably truly diphtheritic.

Symptoms.—The period of incubation varies from two days to twelve. It, however, seldom exceeds one week.

According to what may be the primary or principal seat of invasion we may speak of the *pharyngeal*, *laryngeal*, and *nasal* forms of diphtheria.

In the *pharyngeal variety*, *fever* and *sore throat* appear simultaneously, sometimes preceded by a chill or chilliness. Both increase rapidly. There may be aching or a sense of weariness. More rarely a convulsion ushers in the attack. At times at the beginning, at others on the second or third day, an erythematous eruption more or less extensive appears on the skin and may lead to the diagnosis of scarlet fever. Usually, as soon as attention is called to the throat, white patches are found on one or both tonsils, which spread with varying rapidity. It is this spread from the original focus by which the disease is especially characterized as something distinct from follicular tonsillitis. Commonly, the extension is anterior, over the anterior half-arches to the uvula, and to the palate, or up into the nasal passages, or both. With the invasion of the uvula and palate, commonly reached about the fourth day, the diagnosis becomes certain, even without the bacteriological examination. More serious is the extension backward into the larynx, producing croup.

The *temperature* rises to 103° or 104° F. (39.4° or 40° C.), but is not characterized by extreme or persistent elevation. The *pulse*, which ranges from 120 to 140, is never very full and strong, but tends early to smallness and weakness. *Delirium* is rarely present. *Deglutition* becomes more and more painful, and is increased by external glandular swelling, involving the lymphatic and salivary glands, although this swelling is not invariably present. As the nasal passages become involved, breathing becomes more and more obstructed, until, finally, it is possible through the mouth only. The Eustachian tube, middle ear, and even the antra may be invaded. So, also, there may be diphtheritic conjunctivitis, and even keratitis, and, though rarely indeed, dermatitis. Should there be, however, excoriations or wounds, they are apt to be invaded by the diphtheritic pseudo-membrane. Such false membrane may, however, be due to the streptococcus, which requires a bacteriological examination for its recognition.

If the inflammation and membrane formation extend downward, *laryngeal cough* and the signs of laryngeal obstruction become superadded—in a word, the symptoms of *pseudo-membranous croup* supervene.

Or if the process begins in the larynx we have croup at the outset, which differs from spasmodic croup in being less sudden in its onset. The seriousness of the disease is greatly aggravated by the possibility of complete obstruction and suffocation unless averted by operative interference. Not the larynx alone, but the trachea and bronchi, may be invaded by false membrane.

In three to five days after the onset, if the case is one of ordinary severity, the phenomena of constitutional infection make their appearance in extreme *adynamia*, *feebleness of pulse* and *heart-beat*, while a sense of intense weariness is complained of. From this time a new period of danger begins, the danger of death from heart failure. This is a distinct and separate cause from heart paralysis due to neuritis of cardiac nerves. At times in diphtheria, as in scarlet fever, the signs of constitutional poisoning appear at the outset, and the patient is struck down as by a blow, but this is less common than in scarlet fever. In such cases the temperature may not rise, and may even be subnormal.

As intimated under the head of morbid anatomy, the *ulcerative process* may extend much more deeply, producing destruction of tissue and even gangrene, resulting, as in scarlet fever, in fatal erosion of blood-vessels. Usually, the membrane gradually disappears from the fauces as convalescence is established, or is coughed up if deeper in the respiratory passages. Sometimes, on the other hand, it remains on the tonsils several days after all constitutional disturbance has disappeared.

Nasal diphtheria, in which the nares are especially invaded by the false membrane, requires special allusion. It is more apt to succeed upon acute catarrh with little secretion than on chronic catarrh. The effect of the invasion is to increase any previous discharge, which is also rendered acrid and irritating. In this form glandular swelling of the deep facial glands at the angle of the jaw is particularly prone to occur, probably on account of the richness of this locality in lymphatics, and persists as induration, while a chronic pharyngeal and nasal catarrh may persist a long time after disappearance of the membrane. Abraham Jacobi, who also especially emphasized the diagnostic value of this peculiar glandular swelling, called attention to the fact that this form of catarrh is not only liable to be a focus of fresh attacks, but may also be a source of spread to others, long before modern bacteriological studies showed that this is the case. Suppuration in these enlarged glands rarely occurs.

Symptoms of the Diphtheroid Sore Throat.—With the exception of a few instances of pseudo-membranous wound affections the diphtheroid inflammations are almost invariably of the throat. They occur alone or in conjunction with scarlet fever, measles, whooping cough, and typhoid fever. The diagnosis can only be made with absolute certainty by the bacteriological examination; yet before the latter became available, physicians of experience made the diagnosis many times without error. The exudation commonly remains localized; it does not spread. The fever may be as intense or more intense than in true diphtheria, but it is of shorter duration and usually abates within twenty-four or forty-eight hours. Then the circumscribed pseudo-membrane is apt to drop off and leave a clean ulcer which heals rapidly. In many cases the

exudate occupies one or more lacunæ of the tonsil, constituting lacunar or follicular tonsillitis. In other instances there are several foci on the tonsils seemingly beneath the epithelium and they remain a day or two longer. They do not, however, enlarge or fuse. In other cases the superficial resemblance to the pseudomembrane of diphtheria is more close, but the harmless membrane retains with constancy its limitation. On the other hand, it may happen that the true diphtheritic membrane remains circumscribed, but in this event the constitutional symptoms are correspondingly mild. The chief danger from the latter cases is that they may be a source of infection, and on this account, where no bacteriological examination is possible, every mild case should be isolated until the true nature of the disease is determined. The diphtheroid inflammations are not regarded as infectious, yet it must be admitted that a few of them are grave affections. Most important is it to remember that there are grades of diphtheria so mild that the local symptoms attract little attention, but which are yet capable of spreading the contagion.

Complications and Sequelæ.—The most frequent complication of diphtheria is *nephritis*, which pursues a course somewhat similar to the nephritis of scarlet fever, but is less apt to be accompanied by dropsy, and generally terminates more favorably. On the other hand, albuminuria is present in almost every severe case. There may be the other signs of nephritis, blood casts, epithelial casts, scanty and even suppressed urine. Capillary bronchitis and broncho-pneumonia are serious complications, especially if the result of insufflation of the virulently laden membrane. Endocarditis and arthritis sometimes occur.

The most important sequel of diphtheria is *paralysis*. This is now generally regarded as the result of a toxic neuritis. It may come on as early as the seventh or eighth day, or as late as the second and third week, when convalescence is apparently established. It is quite as likely to follow mild cases as severe ones. It may even follow wound diphtheria. It most frequently affects the palate, producing nasal speech and permitting the passage of fluids into the posterior nares and through the nose. There is simultaneous anæsthesia of the pharyngeal mucous membrane, destroying reflex excitability. Next in frequency of involvement are the muscles of deglutition; more rarely the eye muscles, especially those of accommodation, which is thereby rendered defective. There may be also ptosis and strabismus, or paralysis of the distribution of the facial nerves. Still more rarely the nerves of the lower extremities are involved, producing paralysis, partial recovery from which leaves lameness, which may last through life. Generally, however, recovery takes place in the order of involvement, usually in two or three weeks. Sometimes there are ataxic symptoms, with loss of the tendon reflexes, and no involvement of sensation.

The most serious of the local palsies is that of the heart, due to neuritis of the cardiac nerves. In this there may be bradycardia and tachycardia, but the most frequent evidence is the sudden cessation of the heart's action, and this tragic termination may take place during convalescence. Indeed, the event is more frequent during convalescence, and is often as late as the sixth or seventh week. At other times the

phenomena of heart-failure are more slow in their development. The pulse may become weak and rapid, or more rarely become slow, while the extremities become cold, the temperature falls, and there supervene in a few hours all the signs of collapse. A most striking instance of bradycardia in diphtheria was met by Baumgarten, wherein, toward the close, the pulse fell to 25, though very regular.

Diagnosis.—The only two conditions with which diphtheria is liable to be confounded are, first, the different forms of diphtheroid faucitis, including follicular tonsillitis, and, second, scarlet fever. The difficulty in deciding between the former condition and diphtheria at the outset is sometimes extremely great, and time or the bacteriological investigation will alone settle it. The primary fever, constitutional disturbance, and dysphagia are often equally as great in follicular tonsillitis, due to streptococcus or some other cause of infection. As a rule, however, the follicular exudate remains limited in size, it does not spread, and in the second or third twenty-four hours is apt to drop out, leaving a clean-cut ulcer which heals rapidly, while the constitutional symptoms disappear with equal rapidity. In the form of follicular tonsillitis attended by multiple white spots on the tonsils, the local resemblance to diphtheria is even greater, but the constitutional disturbance is less and the white spots remain isolated.

Sometimes, however, the mass of desquamated epithelium, fibrin, and fungous filaments which make up the contents of the follicles in follicular angina extend outside of the follicles and over the surface of the tonsils. Then it becomes more difficult to decide. It does not, however, pass the boundary of the tonsils. The follicular fungi are said to stain bluish-red with an iodo-potassic iodine solution. Further certainty is secured by making cultures from the membrane, a small portion being removed by the sterilized platinum loop, or cotton swab, and planted in gelatinized blood-serum. In the course of twenty-four hours characteristic colonies will develop, and the microscope will confirm the diagnosis.

From scarlet fever diphtheria is usually easily distinguished by the absence of eruption, although this aid is wanting in those few cases of scarlet fever in which there is no eruption, and in those of diphtheria where there is an erythematous redness. Under these circumstances the distinction becomes more difficult, if the throat symptoms are similar, as they sometimes are. The prevalence of an epidemic of one or the other disease aids in the decision. Later on, the desquamation which takes place in scarlet fever, but not in diphtheria, also settles the question.

The larger cities in the United States now offer, through their health bureaus, to make bacteriological examinations for physicians in all cases of possible diphtheria. Outfits are left at Stations. They consist of a box containing a tube of blood-serum and another containing a sterilized swab. The following directions are issued by the Philadelphia Board of Health :—

"Inoculations should be made by rubbing the cotton swab attached to the end of the wire contained in the test-tube gently but freely against any visible exudate, and then drawing it over the surface of the culture-

medium without breaking the surface of the latter. The swab should then be replaced in the tube from which it was taken, and both tubes be replugged and put back into the box. Return the box to the Station from which it was obtained as soon as possible, or bring it directly to the Laboratory. The tubes will be collected every afternoon, examined the following morning, and reports will be mailed by one o'clock P. M. The attending physician can obtain information, however, by telephoning directly to the Laboratory after that hour."

Prognosis.—The introduction of the serum treatment for diphtheria, which may be dated April, 1893, when the first 30 cases treated by Behring's normal serum were reported,* marks an era prior and subsequent to which the prognosis of diphtheria presents very different aspects. Prior to 1893, while the prognosis was so unfavorable as to justify a wholesome dread of the disease the world over, many moderately severe and most mild cases got well. Allowing for the great variation in the percentage of fatal cases in different epidemics, and especially at different ages, the very careful and reliable studies of William H. Welch,† of Johns Hopkins Hospital, make it safe to put such mortality at a minimum of 40 per cent. Where the larynx was involved, it amounted to almost 100 per cent. Of the remaining cases probably one-third died. A fair ratio of the causes of death in 25 fatal cases prior to the use of antitoxin is given in a recent paper by William P. Munn ‡ as follows: from septic intoxication 8, laryngeal stenosis 7, cardiac paralysis 6, hemorrhage from the bowels 1, nephritis 1, unknown 2; total 25. Thus the causes of death are adynamia, laryngeal obstruction, heart paralysis, or suffocation from paralysis of deglutition. Occasionally nephritis is responsible, also broncho-pneumonia. Hemorrhage from an eroded blood-vessel is a possible cause of death. Since the introduction of the antitoxin treatment the studies of the same careful observer show a reduction of mortality of between 50 and 60 per cent. This improvement affects all classes of cases, including those operated upon as well. As near as it may be possible to put it, the mortality since the introduction of antitoxin has been eight to 25 per cent.

Treatment.—In the management of every case of diphtheria there are three principal indications—first, to prevent constitutional infection; second, to support the system, and, third, to combat the toxin. The first is accomplished by the local use of germicides and disinfectants; the second by resolvent supporting treatment; and the third by serum therapy.

I. *Local Treatment.*—Germicides and disinfectants are best applied when possible by the spraying apparatus at intervals of an hour, or, at most, every two hours. If the spraying apparatus cannot be used, as is often the case with children, a swab of cotton wool or a soft sponge may be used. The most satisfactory solution in my hands for this pur-

* The prior trials of immune serum in the treatment of human diphtheria, made in von Bergmann's clinic in Berlin in 1891, by Henoch and Hebner in Berlin in 1892, were tentative and made with weak serum and in insufficient doses.

† "The Treatment of Diphtheria by Antitoxin." Reprint of paper read before the Association of American Physicians, May 31, 1895, and published in the Transactions for that year.

‡ "Diphtheria, a Clinical Study," Medical News, Philadelphia, March 25, 1893.

pose has been equal parts of peroxide of hydrogen and Dobell's solution. Bichloride of mercury is also a suitable solution for spraying, of strengths of one to 4000, or even, in extreme cases, one to 2000. The objection to the corrosive sublimate solution is its extremely unpleasant taste. Carbolic acid may also be used in the proportion of 15 minims to an ounce of glycerine and water (one gm. to 30 c. c.). The stronger solutions are better applied by a swab than by the spray-apparatus, while with children it is often impossible to use the spray. Solutions of albumen solvents are also highly recommended by some, such as trypsin and papoid, in the strength of 30 grains to an ounce (two gm. to 30 c. c.) or lactic acid in the same proportion. Chlorine water, boric acid in saturated solution, and iodoform saturated solution in ether, or five per cent. suspended in equal parts of glycerine and water, are all useful local applications. Where there is laryngeal diphtheria, the patient should breathe an atmosphere saturated with the vapor of slaking lime. The comfort derived from such breathing is often very great. Lœffler's solution is highly praised. It is composed of menthol, ten grains, dissolved in enough toluol to make 36 c. c. ; liquor ferri sesquichloride, four c. c., and absolute alcohol, 60 c. c. Still another solution is tincture of the perchloride of iron, 1½ drachms (six gm.); glycerine and water, each an ounce (30 c. c.); carbolic acid, 15 to 20 minims (one to 1.3 gm.).

II. *Constitutional Treatment.*—This includes measures which also have for their object, first, the checking of the spread of the membrane, its loosening and solution; and, second, maintaining the strength of the patient. The former is accomplished by the preparations of mercury. Of these I prefer the bichloride of mercury in doses of $\frac{1}{48}$ grain (.0027 gm.) to $\frac{1}{12}$ grain (.005 gm.) for an adult, in conjunction with tincture of the chloride of iron and the chlorate of potassium, every two hours. The former dose makes one-quarter grain (.0162 gm.) of the bichloride in twenty-four hours, but as much as one-half (.032 gm.) may be given in that period. These doses are given to adults, and they need not be much reduced for children. There need be little fear of poisonous effects from the bichloride, as bowel irritation, pain, and loose movements give a warning before any more serious consequences supervene. When these symptoms appear the bichloride should be discontinued or the dose decidedly diminished. The calomel treatment is preferred by some. It is given in hourly doses of $\frac{1}{6}$ or $\frac{1}{8}$ grain (.016 or .008 gm.) until spawn-like stools are produced. Both remedies are supposed to have the effect of loosening the membrane.

While the chlorate of potash and iron have lost some of their former reputation, they are still, in my judgment, indispensable, and I always combine them with any other treatment I may care to use. As held by Jacobi, they are, at least, useful in the concurrent pharyngitis and stomatitis which invariably attend the disease. The chlorate of potash in saturated solution may be used as a simple mouth wash. Gargling is an ineffectual method of reaching the throat and has given place to spraying.

Jacobi recommends that in children too young to use the gargle the local effect of the chlorate of potash be secured by frequent administrations of small doses. Thus, regarding 1½ drachms to two drachms

(six to eight gm.) as a suitable twenty-four hours' quantity for an adult, 30 grains (two gm.) for a child two or three years, and 20 grains (1.33 gm.) for a baby a year old, he prefers the whole amount to be given in 50 or 60 doses rather than eight or ten, giving the weaker dose every hour or half-hour, or every fifteen or twenty minutes, being careful to take no water immediately afterward, for obvious reasons. But I have seen the thing overdone. I have seen a little child, exhausted for want of sleep, aroused every fifteen minutes for the administration of medicine, when what it wanted was sleep more than medicine.

Quinine should form a part of the medicinal treatment, ten to 24 grains (.65 to 1.50 gm.) in the twenty-four hours. Stimulating, nourishing, and easily-assimilated food is necessary. Milk is to be preferred to all else, fortified with full doses of whiskey or brandy, two drachms to an ounce (8 to 30 c.c.) every two hours being required in all cases of severity, and proportional doses for children. The milk may, of course, be alternated with nutritious animal broths or beef peptonoids. In extreme cases of difficult deglutition nutrient enemata may be useful, but nourishment by the stomach tube, if possible, is more efficient. For enemata, peptonized milk is the most suitable. To this brandy and whiskey may be added if needed. Rectal alimentation has sometimes to be discontinued because the enema is made too large and is too frequently administered. Once in four hours is often enough, and four ounces at a time are as much as the rectum will commonly bear.

Where laryngeal obstruction is imminent, intubation or tracheotomy should be performed. Lives have been saved by both of these operations. Intubation may precede tracheotomy, as its use does not preclude the more serious operation at a later date if the obstruction increases. Such cases should breathe an atmosphere charged with the vapor of slaking lime.

In the nasal variety of diphtheria special means must be employed to disinfect and cleanse the nasal passages. The solutions recommended to spray the throat may be used for cleansing the nasal passages. Gentle injections into the nostril may be more efficient than the spray, precaution being taken of keeping the mouth open, by which the entrance of fluid into the Eustachian tube is guarded against. The injections should be continued until the fluid has free exit either by the other nostril or through the mouth. Jacobi has seen cases where he has been compelled to bore a passage with a silver probe through a mass of membrane filling the nasal cavities, and then apply carbolic acid to remove the denser portions before injecting. He also recommends that when about to bring the injection to a close, the nasal cavities should be pressed together for an instant with the fingers, as in this way the fluid is forced backward into the pharynx and swallowed or ejected through the mouth, thus washing both at the same time.

III. *Serum Therapy.*—The treatment of diphtheria by antitoxin should be associated with the general and local treatment described. It is based on the fact that animals may be made immune to diphtheria by the injection of the diphtheria toxin attenuated by contact with iodine terchloride. This was shown by Behring in 1891, after some preliminary experiments by Fraenkel in the same year. In 1892 Behring

and Wernicke employed this method successfully in immunizing sheep, and also ascertained that blood-serum from an immune animal could be used with success in arresting diphtheritic infection in susceptible animals, to which was added this fact, of great importance in connection with the therapeutic use of antitoxin : *for producing immunity a smaller amount of serum was required than was necessary* for the cure of *an animal already infected. If the injection was made immediately after infection, from one and a half to two times the amount was required ; eight hours after, three times as much, and twenty-four to thirty-six hours after infection, the dose required was eight times the immunizing dose.*

In obtaining a uniform standard of strength Behring produced first his *normal therapeutic serum,* which when injected into guinea pigs, in the proportion of one to 5000 of body weight, saves the animal from the fatal effects of ten times the minimum dose of a culture two days old which would kill a control animal not thus treated. *One c. c. of this normal serum he calls an antitoxin unit.* The serum prepared by this method is labeled of three strengths : No. 1 is 60 times the strength of the normal serum ; No. 2, 100 times as strong, and No. 3, 140 times as strong. Of the No. 1 Behring's serum, for a child under ten years, a flask of ten c. c. is sufficient to arrest the progress of the disease and effect a cure if given within two or three days after the onset of the attack. The injection is made in the skin of the side of the buttock or flank or under the angle of the scapula. On the following day, if there is no improvement a flask of No. 2 should be used. In case of extreme severity, especially if seen late, a flask of No. 3 should be used at once.

Evidences of improvement are an abatement in the faucial swelling and loosening of the membrane with amelioration of the general symptoms. The former should take place within twenty-four hours. The latter is manifested by a slowing of the pulse, lowering of the temperature, feeling of diminished discomfort, and revival of spirits. The improvement is more rapid in the milder cases, but even in the severest forms those who are familiar with the slow course of the disease are struck with the marvelously rapid change for the better which sometimes takes place.

For producing immunity in children subject to infection one-tenth the amount (one c. c.) of the No. 1 serum is said to be sufficient to protect, while one c. c. of the stronger serum will arrest the disease during the period of incubation. As already stated, the later the treatment the larger the dose required.

Behring and others declare that the diphtheria antitoxin has no injurious effect upon animals in the largest dose in which it has been employed, and that, aside from its antitoxic powers, its properties are entirely negative, so far as human beings are concerned. Yet there is evidence to the contrary ; notably a fatal case reported in the Journal of the American Medical Association, April 4, 1896, that of a healthy boy five years old who received an injection of Behring's fresh serum as a prophylactic and died within five minutes ; also another case in Berlin, referred to in the Medical News, April 18, 1896, p. 443.

One of the objections to the serum treatment has been the necessarily large bulk of the injection. This is, however, being reduced by increas-

ing the strength of the serum, and the changes which are being made in this respect necessitate close examination of the preparation and the directions for their use. Other reliable preparations than Behring's are now to be had, and there is scarcely a large city in which antitoxin is not manufactured, in many instances under the regulation of Boards of Health. It is scarcely necessary to say that full antiseptic precautions must be taken in making the injections. It is probably the neglect of this which has produced abscesses in a few instances. Diffuse erythema and urticaria have also followed the use of antitoxin, and, it is said, albuminuria, but it is to be remembered that albuminuria is an incident of diphtheria otherwise treated.

Prophylaxis.—Most important are the precautions necessary to prevent a spread of the disease. To this end the patient should be isolated, all carpets and unnecessary furniture and hangings should be removed from the room, and all utensils used in treatment should be kept apart and separate for the patient's own use. Spoons and tongue depressors should be kept in carbolic acid solutions or, better, thrown into water kept boiling. All bed linen and clothing removed from the patient should be boiled, being immersed in water before removal from the room. Mattresses, pillows, and woolen garments too good to be destroyed should be exposed to super-heated steam in establishments provided for the purpose in the cities. Or they may be disinfected at the same time with the apartment occupied by the patient. They should be opened and suspended in this apartment, of which all the doors and windows must be closed tightly and the room fumigated with sulphur. To this end the sulphur, in the amount of two pounds to every ten feet (two kilos to every 2.5 meters) square, should be placed in iron pans supported by bricks, placed in wash-tubs containing a little water. The sulphur is then ignited by glowing coals or by burning alcohol. The room should be kept closed for twenty-four hours. After this fumigation the articles of clothing should be hung out in the open air for several hours, and the floors and woodwork washed well with a solution of corrosive sublimate one to 1000, while the walls should be wiped down with a similar solution.

Finally, physicians and nurses in attendance on the patient should carefully wash their hands before leaving the room, first in soap and water, and finally rinse them in corrosive sublimate solution one to 1000. Nurses in constant attendance should wear an overdress of washable material, which should be slipped off before leaving the room, and the physician while in the room should be similarly covered and should treat his hands as described.

The convalescent patient should also be kept isolated until thoroughly disinfected. This is accomplished by giving first a hot water and soap bath, then washing the body of the patient with a solution of bichloride of mercury one to 2000, or two per cent. solution of carbolic acid, or, what is more agreeable, 25 to 50 per cent. alcohol. This should be done two or three days in succession. The hair should be cut or similarly washed with these solutions. The regulations of the Board of Health of Philadelphia do not permit the children of a family in which diphtheria has been present to return to school until thirty days after the Board's physician has declared the patient's recovery.

The Treatment of Complications and Sequelæ.—Complications are treated as the same conditions under other circumstances, and the paralysis so frequently succeeding upon diphtheria alone requires special allusion. The prognosis is on the whole good, and time, under favorable circumstances, mainly effects the cure, and during this the most important measures are those which save the patient from accident. Thus, if there is paralysis of the muscles of deglutition liquid food only should be used, and it may be necessary to nourish for a time by the rectum or by means of the stomach tube. So, too, undue exertion should be avoided. Electricity and tonics, especially strychnine, are indicated. The former is applied to wasting muscles, and may be advantageously associated with massage. Strychnine should be given in full doses, ascending gradually to $\frac{1}{20}$ grain (.003 gm.) three and four times a day, with appropriate reduction for children. Iron and quinine should also be given.

The electrical treatment for paralysis of the pharyngeal muscles is applied in the following manner: An electrode is placed at the back of the neck and a very small electrode is touched to the velum palati, and a rapidly interrupted faradic current of moderate strength applied. Galvanism may be similarly used. A specially constructed electrode is also applied to the throat.

SMALL-POX.

Synonym.—*Variola.*

Definition.—Small-pox is an acute contagious disease especially characterized by an eruption which passes through the successive stages of papule, vesicle and pustule, desiccation and desquamation.

Historical.—Small-pox was first accurately described by Rhazes, an Arabian physician, in the ninth century, and distinguished by him from measles, but it is believed to be the same as the *pesta magna* described by Galen [A. D. 130–200]. It prevailed also in China many centuries before the Christian era. Sydenham's classic description was made in the seventeenth century. It is known to have prevailed in the sixth century and again during the Crusades. The disease is believed to have been introduced into America by the Spaniards early in the sixteenth century.

Etiology.—The contagium of small-pox, probably the most virulent of all the contagia in its effect upon the unprotected subject, has not yet been isolated. It is conveyed in the secretions and exhalations of the body, including those of the lungs. The pus of the pustule is its most fertile source, and the dust derived from the dried pus scales is the more usual medium of its distribution. The degree of mildness or severity of a case does not influence that of another caused by it, the severest cases being at times followed by the mildest forms, and *vice versa*. The contagium is very tenacious and may be dormant for months in clothing or furniture or hangings. No age or sex or race is exempt, but the number of cases in successive decades diminishes because of the immunity furnished by a previous attack. The *fœtus in utero* may acquire the disease from the mother, and the child may be born with the eruption

on it. Certain individuals are invulnerable even though unprotected by vaccination, while the mortality in aboriginal races is very great.

The immunity secured by a previous attack suggested to Lady Mary Wortley Montague the idea of inoculation for the purpose of protection, the practice of which was introduced in England in 1718. The still more thorough protection afforded by vaccination was discovered by Jenner in 1798. Regarding the infectious nature of the disease F. Colclough says: "Small-pox is not infectious until the second and third days of disease."

Morbid Anatomy.—The essential morbid anatomy of small-pox is that of the eruption as represented by its various stages and modifications, including hemorrhagic infiltration. To the anatomy of the eruption is added that of the complications which may occur.

The histology of the pustule shows that it starts in the *rete mucosum*, close to the true skin. The centre is a focus of coagulation necrosis, and about it the reticular spaces are filled with serum, leucocytes, and fibrin filaments. So long as the process does not extend

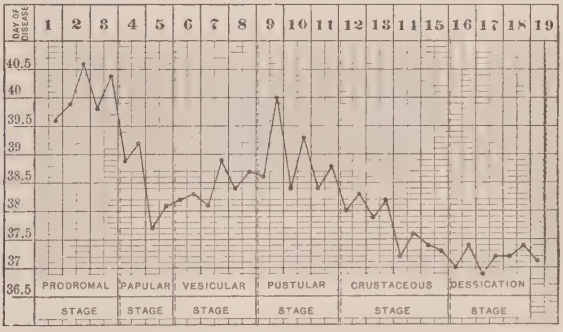


FIG. 13.—TEMPERATURE CHART OF SMALL-POX.—(*Eichhorst.*)

deeper, healing takes place without a scar. In the more severe cases the papillæ of the true skin are invaded to various depths by the infiltration and destroyed, producing a loss of tissue which constitutes the pit. Among other morbid phenomena may be mentioned a hardness and firmness of the spleen. Cloudy swelling of the secreting cells of the liver and kidney occur, as in other fevers with high temperature. True nephritis is rarely present.

Symptoms.—After a period of incubation of from seven to twelve days, and sometimes longer, the victim is seized with *violent muscular pains*, especially in the back. Often a *chill* or *chills* usher in the disease, and in children a *convulsion* may be the initial symptom. *Intense headache* is also present. *Fever* sets in rapidly and the temperature reaches 103° to 104° F. (39.4° to 40° C.) the first day. The *pulse* is rapid, hard, and strong at this stage. *Delirium* may be present and is sometimes very violent.

About the second day the *initial rashes* make their appearance.

These have been especially studied by Theodore Simon,* Knecht,† Scheby-Busch,‡ and William Osler,§ although they are mentioned by some of the older authors, including Sydenham, Wood, Watson, Niemeyer, Trousseau, Marson, Munro, and others.

They include a diffuse scarlatinous rash and a macular or measly form, dark red in color and occupying a variable extent of surface. Either may be associated with petechial ecchymoses. Sometimes they are general, but as a rule they are limited to the abdomen, the inner surface of the thighs, or the lateral region of the thorax and axilla. Among Osler's cases was one of a true urticarial prodrome. While it is to be remembered that the co-existence of small-pox and measles and of small-pox and scarlet fever is possible, it is more than likely that the eruptions on which the diagnosis was based were really the initial rashes of uncomplicated small-pox.

On the fourth day the *distinctive eruption* makes its appearance in the shape of small red spots, first on the forehead and wrists, whence it extends rapidly over the face and extremities, becoming quite general in the first twenty-four hours. At this stage the eruption is not unlike measles, but in another twenty-four hours it is decidedly different. The papules have acquired shot-like hardness. With the appearance of the eruption the fever falls and the patient feels comfortable. On the fifth or sixth day a clear or slightly turbid serum makes its appearance. Coincident with this a depression is seen in the middle of each vesicle. It is *umbilicated*, and this umbilication is the most characteristic feature of the eruption. Frequently a hair follicle passes up through the centre of it. By the eighth day the turbidity has increased until it is bright yellow and the umbilicus has disappeared. The pustule is complete. The maturation takes place in the same order as the eruption appeared. With the appearance of suppuration the fever again returns, known as the secondary fever, and with it the elevation of temperature and other signs of fever. There is a good deal of pain in the inflamed parts because of the tension. On the tenth or eleventh day the pustules become dry, and by the fourteenth are converted into crusts, which drop off, leaving in mild cases a simple discoloration, in severe a more or less deep ulcer, or, if cicatrization be complete, a simple pit. The eruption may be found on the tongue and buccal mucous membrane and even in the pharynx, larynx, and œsophagus, and pustules have been found in the stomach and rectum. In the trachea and thorax there may be ulcers. Sore-throat, nausea, hoarseness, vomiting, and diarrhœa may be consequences. With the drying of the eruption the fever disappears.

This description is typical of the course of the eruption in the simple discrete variety. It may be variously modified. The attack may be so virulent that the patient dies before the eruption makes its appearance, or it may be arrested at any stage. Sometimes blood forms the contents of the pustule and there may be subcutaneous infiltration of blood in

* "*Das Prodromal-Exanthema der Pocken.*" *Arch. f. Dermatol. und Syph.*, Prag, Heft III, 1870, 346; 1871, Heft II, 242; Heft III, 309; 1872, Heft IV, 541.

† *Arch. f. Dermatol. und Syph.*, Heft III, 1872, 372.

‡ *Arch. f. Dermatol. und Syph.*, Heft IV, 1872, 506.

§ "The Initial Rashes of Small-Pox," Canada Med. and Surg. Jour., 1875.

addition. Along with this there may be hemorrhage from the mucous surfaces of the nose, stomach, or bowels, or there may be hæmaturia.

The pustules may be so close to each other that they join, when the case is *confluent*, or they may be separate and distinct, producing the *discrete* form. The variety with bloody infiltration is called hemorrhagic. The diagnosis as to whether the confluent or discrete form is present is generally made by an examination of the face, for it is an interesting fact that nowhere are the pox marks more abundant than upon the face.

Sydenham early called attention to the fact that in the confluent variety the eruption appears earlier (on the third day), and its early appearance, according to him, is an indication that the case will be one of that variety. All the symptoms are much more severe. There is not the abatement of fever described as occurring on the appearance of the eruption. The face, hands, and feet present an almost continuous pus-vesicle, which often bursts in places, and the pus partly drying, there results a picture which is far from pleasant to look upon. Such pronounced morbid changes must produce wide systemic exhaustion, as is manifested by the tenth or the eleventh day by the growing weakness of the patient, an adynamia which frequently terminates in death. When recovery takes place the secondary fever is the more prolonged the more extensive the suppuration.

The *hemorrhagic form* of small-pox is still more severe. Two forms of it are described: one the *purpura variolosa*, or hemorrhagic variola, in which the hemorrhagic symptoms appear early in the shape of a hemorrhagic rash while hemorrhage takes place from the mucous surfaces, generally on the evening of the second or third day. The patient dies in from two to six days, sometimes before the eruption makes its appearance. In the second form, *variola hæmorrhagica pustulosa*, the case progresses at first like any other, and it is not until the vesicular or pustular stage that blood makes its appearance in the pocks.

A third variety of small-pox is *varioloid*, which is variola modified by vaccination or a previous attack of small-pox. In general, varioloid is small-pox bereft of all its serious features, each symptom being milder. The initial fever is less, the eruption is less general and may abort in its development, the secondary fever is less marked, convalescence sets in earlier. Yet it has happened that both classes of persons referred to, those having had small-pox and those having been vaccinated, have had very severe attacks, from which, indeed, the patients have perished. Generally, the longer the interval between the attack and vaccination the more severe the former is. Similar is the mildness which characterizes a small-pox produced by the direct inoculation of an individual from the pus of another, though the degree of mildness is less than after vaccination.

Other names given to less important varieties are *variolæ sine variolis*, or variolous fever without eruption; the crystalline pock, in which the eruption continues vesicular, and the "stone pock," "horn pock," and "wart pock," in which the vesicles dry up into tuberculated or warty elevations.

Complications.—Among complications of small-pox may be men-

tioned laryngitis with fatal œdema of the glottis, broncho-pneumonia, parotitis, vomiting, diarrhœa, albuminuria, but rarely nephritis. Prolonged delirium, and even insanity, may supervene, while neuritis may occur during convalescence. So may arthritis. On the skin may be boils and painful acne. A troublesome and painful conjunctivitis used to be the result of indifferent care of the eyes, but is now less common because of greater care in this respect. Myocarditis and pericarditis sometimes occur, and most rarely endocarditis.

Diagnosis.—With the appearance of the perfect papule all doubt in the diagnosis of small-pox generally ceases. Ignorance of the initial rashes, measly and scarlatinal, has often led to errors of diagnosis. On the other hand, the resemblance of the eruption of measles to small-pox has also given rise to errors, the result of which has been no less serious, because in consequence cases of measles have more than once been sent to small-pox hospitals with disastrous consequences. Never in measles is there such a severe pain in the back as in small-pox, while the early cough and coryza are only found in measles. The lesson taught is to defer a positive diagnosis, because less serious mischief can result from an error thus occasioned than as the result of an opposite course. The possibility of relapsing fever being taken for small-pox has been alluded to in considering the former disease. Cerebro-spinal fever may also be simulated by the hemorrhagic form of small-pox. Pustular syphilides and accidental croton-oil eruption have been mistaken for small-pox, as has also chicken-pox.

Prognosis.—Small-pox is a serious disease, and the death-rate is always relatively large. It varies, however, at different ages, in different races, and in different epidemics. The young die almost always. Thus in the Montreal epidemic of 1885, 86 per cent. of the deaths were children under ten years. The African and American Indian and native Mexican have perished by thousands. The range of the mortality in different epidemics is put down at 25 per cent. to 35 per cent. The hemorrhagic cases are always serious; those of *purpura variolosa* all die, and although some cases of *variola pustulosa hemorrhagica* get well, the majority are usually fatal on the seventh, eighth, or ninth day. The pregnant woman usually perishes, but not always. The complications of pneumonia and laryngitis are serious.

From the statistics of Dr. Gregory, based upon London hospital practice, the greater number die on the eighth day, but in private practice, according to the experience of the late George B. Wood, the greatest number of deaths occur between the twelfth and eighteenth days.

Treatment.—It is not possible to cut short a case of small-pox. The patient should be isolated and taken to a small-pox hospital if possible. An uppermost room should be selected, all hangings and carpet removed, and communication with the rest of the house cut off by closed doors fortified by a sheet dampened with a solution of carbolic acid one to 60. Separate dishes and utensils should be provided, and nurses should hold no communication with other members of the family. All clothing removed from the patient should be put in scalding water, and sweepings should be burned. The nurse should wear an overall, to be removed on leaving the room, and her head should be covered with a close-fitting cap.

The treatment must consist in combating the symptoms. Morphine or in less severe cases phenacetin, antifibrin, antipyrin, must be given to control the pain in the back. Nourishing liquid food and stimulants are required in adynamic cases. The fever is treated by sudorifics and by aconite, or by cool sponging or even by baths, as in typhoid fever, if the temperature is high. Cool drinks should be permitted *ad libitum.* The complications must receive the treatment appropriate to them. Tracheotomy may be demanded by œdema of the larynx.

It has always been the object of the physician to find some means of preventing the disfiguring scars which so invariably remain after very severe cases. No method is always successful. It has long been thought that the absence of light favored healing without pits. For the painful ophthalmia which so often attends small-pox, darkness is certainly a comfort, but that it diminishes the pitting I have much doubt. It is a comforting fact that even the deepest and ugliest pits gradually lose their distinctness with age, and that much of the marking disappears in the course of a few years. The surface should, however, be anointed with vaseline, cold cream, or similar substance, as it allays the burning and itching and keeps the scabs moist and prevents the contagium from spreading itself through the air. The odor, which is often intolerable, is perhaps best covered by adding carbolic acid to the vaseline or other unguent employed, say ten grains (.666 gm.) to the ounce (30 gm.); or a watery solution of carbolic acid may be made of the same strength and applied on cloths. Bichloride of mercury, one to 2000, may be used in the same way.

The eyes, nose, mouth, and throat should be kept clean, all crusts being carefully removed. As soon as convalescence is established the patient should bathe daily, using carbolic soap, the bathing being kept up until the skin is perfectly smooth, because only then does the patient cease to be a source of infection.

VACCINE DISEASE.

SYNONYMS.—*Vaccinia; Vaccina; Cow-pox; Kine-pox.*

Definition.—Under vaccinia are included the local and general symptoms which result from the introduction into the blood of man of the contents of the vesicle of kine-pox, or small-pox as occurring in cattle. Persons thus successfully vaccinated are, in the vast majority of cases, immune from small-pox. The local product of such vaccination is the vaccine vesicle, the contents of which when again inoculated are capable of producing the same disease with its immune effects in another person not previously vaccinated. It is pre-eminently characteristic of the vaccine disease that it can be communicated only when introduced directly into the blood.

Historical.—Edward Jenner, a physician practising in Gloucestershire, England, largely engaged in inoculating small-pox for the protection furnished by it, noticed that certain men who happened to acquire the kine-pox by inoculation, while milking cows, would not take small-pox. On this discovery he proceeded at once to make a series of experiments which led him to conclude that immunity could thus be

secured, and next that this protective disease could be communicated from one person to another. He announced his conclusions with proofs in a paper published in June, 1798. The practice was rapidly introduced, not without some opposition, some of which prevails even at the present day, although it is as certain as any demonstrable fact that thousands of lives have been saved by vaccination and that a thorough and continuous practice of the operation would sooner or later blot out small-pox from the face of the earth.

At the present time it is almost the universal practice to use the lymph directly from the cow (animal lymph), although that from another person having vaccine disease (humanized lymph) can also be successfully used. The chief reason for using animal lymph is the fact that all danger of communicating other affections, especially syphilis, is thus avoided, although there is reason to believe also that protection is more certainly assured. For securing the cow's lymph numerous farms exist in this country and in Europe, where, under the most perfect sanitary precautions, inoculation is practised on the udder of heifers, whence the lymph is gathered and distributed. In Belgium the heifers are slaughtered after the lymph is taken, and if they are found diseased the lymph is not used. In this country the more usual method is to allow the lymph to dry on ivory points or quills, although it is also collected in capillary tubes. Before the use of animal lymph became general the crusts or scabs from vaccinated arms were preserved and moistened to the consistency of pus before inoculation.

The inoculating principle of vaccine virus has not been isolated. Analogy leads us to expect some vegetable organism will ultimately be found in the fluid of the pock. Quist has cultivated micrococci which, he claims, produced in the child a typical vesicle, while Harold, Ernst, and Martin, of Boston, have isolated from bovine lymph a germ which grows on culture media and produces, when inoculated in the heifer or children, characteristic vesicles.

The operation of vaccination is variously performed. I prefer after thorough cleansing to scrape the skin of the arm or forearm with a lancet until the cuticle is removed and a moist surface results, due to the transuded liquor sanguinis, and on this is rubbed, slightly moistened, the lymph-covered ivory point or quill. Prolonged friction is desirable to secure success. It is a disadvantage to have the surface bleed much, as it interferes with absorption. Another method, handed down by the late Prof. Geo. B. Wood from his predecessors, and available only with liquid lymph, is to make three slight punctures obliquely under the cuticle and work the lymph into each. The punctures should be about a line apart and at the angles of an equilateral triangle. Very convenient instruments of various kinds are made to scratch the surface, which are especially useful where a large number of vaccinations is to be made and celerity is desirable. At the present day it is quite the fashion to inoculate on the leg, especially in the case of girls, in order to avoid an unsightly scar. In infants there is no objection to this, but I have known young girls very seriously disabled for a time by vaccination upon the leg. Another advantage of inoculation on the leg of infants is that there is less liability to injure the affected limb in nursing or carrying. The same

thing is nearly as well accomplished by vaccinating on the forearm, but this makes the resulting scar needlessly conspicuous. Another favorite situation is the region of the insertion of the deltoid muscle.

The Phenomena of Vaccination.—Immediately succeeding the operation a slight inflammatory redness appears, which, however, usually subsides rapidly, and before the third day, as a rule, although it is often delayed two or three days and sometimes longer, a slight elevation, red in color, makes its appearance. By the fifth or sixth day this has already become an umbilicated vesicle filled with a transparent viscid fluid, surrounded by a delicate red areola. The vesicle presents a shiny, silvery appearance; by the eighth day the former takes on a lustrous silver-grey appearance, and by the tenth day both have reached their maximum, the pock being by this time one-third of an inch in diameter (*circa* one cm.), one to two lines in height, umbilicated at its centre, and presenting frequently a minute brown spot or scab in the same situation. The areola is now quite angry looking, often two inches (five cm.) or more in diameter, and shows under a magnifying glass numerous minute vesicles on its surface. At this stage, too, it itches and burns to a degree which causes in adults an almost irresistible desire to scratch, while in the child it gives rise to fretfulness and peevishness and to slight fever. Even in the adult there is slight rise of temperature. On the eleventh or twelfth day the disease begins to decline. The areola narrows and becomes less bright, the lymph more turbid and begins to dry. By the end of two weeks the vesicle has been converted into a dry, brown scab, which generally drops off on the twenty-first to twenty-fifth day, leaving a scar which afterward becomes pitted, very distinct at first, but gradually assuming even a whiter hue than the surrounding integument.

The course described is the typical one in a healthy vaccinated child. In other cases the amount of local irritation is much greater, and with it the degree of constitutional disturbance. There is often adenitis in adjacent glands. Sometimes, in unhealthy children, deep, unhealthy ulcers remain which are very slow to heal, while erysipelas and gangrenous ulcerations have even supervened and been followed by death.

Should a considerable time elapse after vaccination, a revaccination will generally be more or less successful. Usually the entire set of phenomena is less characteristic, although it sometimes happens that the same typical course is repeated. Such successful vaccination is regarded as evidence that immunity from small-pox is no longer present, and the person if exposed to small-pox before vaccination would have taken it. Such an attack is almost invariably less severe and presents the modified symptomatology known as that of varioloid. The period of exemption after vaccination varies greatly. It is often perpetual. More frequently it lasts from ten to twelve years, and every person should be revaccinated at ten to fifteen years, and thereafter whenever an epidemic of small-pox is raging, unless he happens to have been successfully vaccinated within a few years.

At times even in first vaccinations an abortive result obtains, the vesicle drying and dropping off much too early. Should this occur the operation should be repeated.

It has already been said that the possibility of transmitting disease

by vaccinating with humanized lymph has been a potent influence in stimulating the employment of animal lymph. Syphilis seems the only disease which can be thus transmitted, although it is claimed also for tuberculosis. It is, nevertheless, important that every precaution should be taken against such accidents. If humanized lymph is used, as it sometimes must be, only that from children of healthy parents, free of syphilis or tuberculosis, should be selected, and under all circumstances lymph admixed with blood should be rejected. It should be taken from fully matured and perfect vesicles on the eighth day.

It is exceedingly important that the physician should have at hand the data of discriminating between the ulcer of vaccino-syphilis and of vaccination and secondary vaccino-syphilis, and secondary syphilis occurring about the time of vaccination. Such data are found in the following table compiled by C. S. Shelly from Fournier, in Fowler's "Dictionary of Medicine:"—

Vaccino-Syphilis.	*Vaccination Ulcers.*
Chancre developed on the site of usually one or two only of the vaccination punctures.	Ulceration affects all the punctures as a rule.
Inflammation is slight.	Inflammation and ulceration severe.
Loss of substance superficial only.	Ulcer deeply excavated.
Suppuration scanty or absent, scabs or crusts.	Much suppuration.
Border of chancre smooth, slightly elevated, gradually merging into floor.	Margin of ulcer irregular, as in "soft chancre."
Surface of floor smooth.	Floor of ulcer uneven, suppurating.
Induration "parchment like" and specific, not merely inflammatory.	Induration inflammatory only.
Inflammatory areola very slight.	Areola inflammatory and erysipelatous.
Gland swelling constant, indolent [syphilitic] bubo.	Gland swelling often absent; if present, merely inflammatory.
Complications rare.	Complications—sloughing, erysipelas, etc.,—often present.
Chancre never developed before the fifteenth day after vaccination; usually not until after three to five weeks; it is still in its earlier stage twenty days after vaccination.	Ulceration is present twelve to fifteen days after vaccination and is fully developed the twelfth day after vaccination.
Secondary Syphilitic Eruption.	*Vaccination Rashes.*
Due to true vaccino-syphilis.	[Including roseola vaccinalis, miliaria vaccinalis, vaccinia bullosa, vaccinia hæmorrhagica; also accidental eruptions—rubeola, scarlatina, lichen, urticaria, etc.]
Appears, at the earliest, nine or ten weeks after vaccination.	A true vaccinal rash appears between the ninth and fifteenth day after vaccination.
Requires, in every case, the pre-existence of a specific ulcer [chancre] at the site of vaccination.	Absence of inoculation chancre.
Exhibits the character of a true specific eruption.	Eruption does not exhibit specific characters.
Fever often slight.	Fever always present.
Lasts for a long time. Usually accompanied by specific appearances on mucous membranes.	Evanescent.
Vaccino-Syphilis.	*Hereditary Syphilis, showing itself about the time of vaccination.*
Begins with local infection chancre and indolent bubo.	No chancre; begins with general phenomena.
Typical development in four stages, viz.: Incubation, chancre, second incubation, generalization [secondary eruption], etc.	Has no typical development in connection with vaccination.
Never appears earlier than the ninth or tenth week after vaccination.	Time of development quite independent of vaccination. Is attended by the characteristic syphilitic bodily aspects. Other manifestations of hereditary syphilis may be present. The history may indicate syphilis.

Some idea of the efficiency of vaccination may be obtained from the fact that through it small-pox has been blotted out from the German army. Further, it was early shown by Marson that of those who have acquired small-pox after vaccination, the disease is vastly less severe than in those who have primary small-pox. This is confirmed also by the statistics of W. M. Welch, Physician-in-charge of the Municipal Hospital of Philadelphia. From a study of 5000 cases, he showed that where there were good cicatrices, only eight per cent. died ; with fair cicatrices, 14 per cent. ; with poor cicatrices, 27 per cent. ; unvaccinated cases, 58 per cent.

CHICKEN-POX.

SYNONYM.—*Varicella.*

Definition.—Varicella is an acute contagious disease characterized by an eruption of vesicles with pearly contents and attended with little or no constitutional disturbance.

Etiology.—The disease is eminently contagious, but no specific causal organism has been isolated. It is almost purely a disease of childhood, occurring most frequently between the second and sixth year. It is a distinct and separate disease from small-pox, an attack bringing no exemption from that disease.

Symptoms.—The period of incubation is from ten to fifteen days. So slight is the constitutional disturbance that very commonly the appearance of *the eruption* is the first notification of the child's illness. At other times there are *slight peevishness*, *restlessness*, and *feverishness*. At others there is a *slight chill* followed by *fever*. Some muscular pain may be present.

A prodromal scarlatinal rash may rarely present itself, but for the most part the suddenness of the eruption is distinctive. It presents itself in the shape of isolated pimples scattered over the body within the first twenty-four hours after constitutional disturbance. They may then appear first on the trunk, but are apt to be seen first on the face. In another twenty-four hours they are pearly pustules without umbilication, and by the end of the third day they begin to dry up, and in another day are converted into dark-brownish crusts, which drop off, leaving, as a rule, no scar. Sometimes, however, a distinct pit is left, especially if the pock be scratched by the child, as it sometimes is, because of the irritation it excites. The pustules appear in crops, so that on the fourth day they can be seen in all stages, but at the end of a week again all have disappeared. Rarely are there more than half a dozen on the face, yet they may be quite numerous, and the victim well dotted over. They occur also on the scalp.

I have never seen any complications with varicella, and in most cases under my observation the disease would have been overlooked but for the eruption. It is said, however, that hemorrhage pocks sometimes occur accompanied by hemorrhage from the mucous membranes, that nephritis and even gangrene have occurred, and infantile paralysis has developed during an attack of the disease.

Diagnosis.—The diagnosis should not detain one long. The trifling

constitutional disturbances, the rapid, almost sudden, development of the pustules, the absence of umbilication and of areolæ, all distinguish the disease from small-pox.

The **prognosis** is invariably favorable.

The **treatment** calls for no special measures, save the application of a simple lotion or ointment to allay the itching. The principal need of the physician is to make the diagnosis.

WHOOPING-COUGH.

SYNONYM.—*Pertussis.*

Definition.—Whooping-cough is an infectious disease, characterized by spells of coughing accompanied by a long-drawn inspiration producing the "whoop," whence the disease is named.

Historical.—While the writings of Hippocrates, Galen, and Avicenna contain expressions which point to the existence of a specific disease like whooping-cough, it is still disputed as to whether this disease was known to the Greeks. It did, however, appear as a distinct disease in 1600 and thereafter.

Etiology.—While the organism of whooping-cough has not been isolated with absolute certainty, Afanassiew, in 1887, found in sputum from the disease a short bacillus of which he has succeeded in making cultures, and inoculations from these into the tracheæ of animals have produced catarrhal conditions. The contagium bearer is likely to be the mucous secretions, from which, when dried, the contagium may disseminate itself through the air.

Whooping-cough attacks children of all ages not rendered immune by previous attacks, while it is not a very rare affection in adults. In older persons it becomes a serious affection. Its epidemics are more common in the spring and winter and often precede or follow those of scarlet fever and measles. The disease is generally communicated from one child to another, and few escape who are exposed. The delicate and those suffering with bronchial and nasal catarrh are more vulnerable. The disease is more serious in the negro race.

Morbid Anatomy.—There is no morbid anatomy peculiar to whooping-cough. The morbid states found after death are those of the complications, viz., bronchitis, broncho-pneumonia, and collapse of the lung. Vesicular and interstitial emphysema are sometimes present, the former from over-distention of the air vessels and the latter from their rupture.

Symptoms.—Whooping-cough has a period of incubation of from seven to ten days. The *cough* in the beginning of the attack is in no way peculiar, being that of an ordinary cold with slight fever and without expectoration. There may be *coryza* and *injection of the conjunctiva.* This cough may go on for a couple of weeks and, if there be nothing in the history to suggest the nature of the disease, may occasion no suspicion. Toward the end of this period, however, the observing mother will have noted that the cough is gradually growing worse and becoming paroxysmal, that it occurs "in spells." Then suddenly a "whoop" is noted and the nature of the disease is suspected.

The paroxysmal stage has replaced the catarrhal, and soon the diagnosis is plain. The paroxysms become more frequent and more severe. Each one begins in a succession of short expiratory coughs which grow in intensity. All efforts lie in the direction of expiration, and all the expiratory muscles are brought into play to this end. The chest is compressed laterally, and bulges in the sternal region. As the result of such efforts the face is flushed, the eyes are injected and bulging, the tears start, and the nose discharges. Finally, the paroxysm terminates or is interrupted by a loud, whooping inspiration—that is, it may end for the time or be immediately succeeded by another similarly concluded paroxysm. Severe paroxysms commonly terminate in an act of vomiting which brings up considerable mucus. The number of paroxysms in the twenty-four hours varies greatly. They may be as often as every half hour or only four or five times in the day. Emotion will precipitate a paroxysm, as will the inhalation of irritant matters. The little patient resists the paroxysms as long as possible, and when the inevitable comes it will run to the basin or bowl, knowing full well what is to happen. The demure method pursued by little children under these circumstances is often at once touching and amusing. I have known each one of a family of half-a-dozen children to have its own cup ready for seizure at a moment's notice. Rupture of a conjunctival or nasal blood-vessel sometimes occurs and occasionally an involuntary urination. An ulcer may form at the frenum of the tongue, said to be due to the pressure of that part of the organ against the incisor teeth. The termination of the paroxysm is followed by temporary relief.

The paroxysmal stage if uncomplicated is unattended by fever, and physical examination of the chest is barren of results as compared with the intensity of the cough. The percussion note is clear, clearer during inspiration. Auscultation may discover a few moist râles soon after a paroxysm, but during it nothing. Even during the whoop the vesicular murmur may be absent, because of the slowness with which the air enters the chest.

The length of the paroxysmal stage is usually from four to six weeks, although in mild cases it may be shorter. Indeed, there are mild cases of whooping-cough in which the paroxysms are scarcely noticeable and would not be noted except for an occasional "whoop." Toward the end of this period the paroxysms become less severe and less frequent, and soon the stage of decline or convalescence is established. In the course of it the paroxysms become still milder and less frequent and finally subside altogether. They are, however, liable to be renewed for a time if the patient takes cold, and even digestive disturbances are said to have a similar effect. The other phenomena of the stage of convalescence are return of appetite, weight, and strength. The period of convalescence occupies another four weeks, so that the entire length of an ordinary attack of whooping-cough is ten to twelve weeks.

Complications and Sequelæ.—The complications which attend whooping-cough are bronchitis, broncho-pneumonia, and collapse of the lungs, pleurisy, and interstitial emphysema. The broncho-pneumonia is apt to be of the insufflation kind. Collapse of the lungs may succeed it. Interstitial emphysema and even pneumothorax may result from rupture

of the air vesicles, and it is apt to become general and serious. In a case of this kind under the care of my friend, Horace Williams, which terminated fatally, an abscess formed at each point at which the emphysema approached the surface. Cerebral palsy and death from subdural hemorrhage are said to have occurred in whooping-cough. Among the sequelæ may be mentioned, as a rare event, tubercular consumption; also permanent changes in the shape of the chest and the so-called pigeon breast, sometimes the result of a prolonged attack of whooping-cough.

Diagnosis.—The diagnosis cannot be delayed after the appearance of the whoop, and it is scarcely possible without it. Spasmodic cough may occur from other causes, but it is not whooping-cough without the whoop.

Prognosis.—Notwithstanding the enormous number of children who have whooping-cough and get well of it, many of them without any treatment whatever, it is not so harmless a disease as many suppose. At the same time I cannot think that the position assigned to whooping-cough by Thomas M. Dolan,* of being third among the fatal diseases of children in England, is true of this country. The chief danger is from the complication of broncho-pneumonia. The younger the child the greater the danger. As already stated, cases in which interstitial emphysema occurs from rupture of the air vesicles are apt to terminate fatally.

Treatment.—The treatment of whooping-cough is one of the opprobria of medicine. Notwithstanding the claims of many to the contrary, it remains a fact that we possess no means of cutting it short. We may, however, palliate the symptoms by diminishing both the frequency and the severity of the paroxysms. The remedies to this end are the opiates, chloral, and antispasmodics. The former two, as a rule, should be reserved for night. Of the latter, the most efficient are belladonna, the bromides, and asafœtida. Belladonna should be given in full doses. It is difficult to name them, and they must for the most part be arrived at by trial. We may begin with one minim (0.066 gm.) of the tincture every two hours to a child of six months, or $\frac{1}{12}$ grain (0.0055 gm.) of the extract, and increase the dose until the characteristic redness of the skin is produced. The bromides, preferably of sodium, should be given as often, in doses of one grain or two (0.066 to 0.132 gm.) for every year of age. I am confident, too, asafœtida is useful. I use it in the shape of a freshly-spread plaster, large, so as to cover the whole of the front or back, and bandaged to keep in place. It should be renewed often. The odor is soon endured. Antipyrin has acquired some reputation and should be tried. The same efficiency should attach to the other coal-tar derivatives, antifebrin and phenacetin.

The intervals between the paroxysms at night may be prolonged by the judicious use of paregoric or deodorized tincture of opium combined with the antispasmodics. Chloral is often a very useful adjuvant, acting also as an antispasmodic.

The inhalation of germicidal solutions suggested by the probable germ origin of the disease has not as yet produced any results. Carbolic acid five parts to 1000, corrosive sublimate one in 4000, and the

* "Whooping-Cough," London, 1882.

peroxide of hydrogen diluted with two parts of Dobell's solution may be used in this way. The remedies are better used with the steam atomizer, as the steam itself has a soothing effect.

Parents should be cautioned to protect their children from undue exposure, because it is from the results of this that complications arise, and it is the complications which are dangerous. Such complications, and other symptoms which arise in the course of the disease, should be treated by remedies appropriate to them.

MUMPS.

SYNONYM.—*Epidemic Parotitis.*

Definition.—Mumps is an acute infectious disease characterized by inflammation of the parotid gland, sometimes of the submaxillary.

Etiology.—Although a *bacillus parotidis* has been described, it is generally conceded that the real contagium of mumps has not been isolated. Children and adolescents are its favorite subjects, the very young as well as adults being equally exempt. More boys are attacked than girls. The disease is more common in the spring and fall. It is more commonly epidemic, but may be sporadic. It may be associated with measles and whooping-cough. One attack protects against a second.

Morbid Anatomy.—The swollen and hardened salivary gland is the sole morbid product. The swelling is mainly due to serous infiltration.

Symptoms.—From seven to fourteen days intervene between exposure and the invasion, which is ushered in by *moderate fever*, rarely exceeding 101° F. (38.33° C.), although 103° and 104° F. (39.44° and 40° C.) have been noted. The first symptom is usually *pain below* and *in front of the ear*, but pain in swallowing may be first experienced. Simultaneously there may be *swelling* about the ear, which extends rapidly in front of the ear and below it until the entire neck in this vicinity is involved.

The maximum swelling is reached in about forty-eight hours, after which the involvement of the other side begins and extends with equal rapidity. The most prominent point is in front of the ear. The swelling does not, however, subside as fast as it comes on, but persists from seven to ten days.

At the height of the disease the pain and difficulty in swallowing are extreme, the former extending often to the interior of the ear, producing earache, and the hearing may be affected. The parts are so tense and swollen as to make opening of the mouth almost impossible, mastication equally difficult. Suppuration is an exceedingly rare event. In cases of great severity delirium is sometimes present for a short time.

Complications.—The most frequent complication is orchitis, and occurring, as it commonly does, after inflammation of the salivary glands has subsided, it has been regarded as a metastasis ; but this is probably not the case, both conditions being probably the result of the same cause, as originally held by Niemeyer. The swelling may affect one or both tes-

ticles, the duration being longer in the bilateral form. The organs are heavy and painful, but not as much so as in gonorrhœal orchitis. The inflammation lasts for three or four days and then subsides gradually. Usually the gland itself is involved, but occasionally there occurs acute epididymitis with acute hydrocele and œdema of the scrotum. Atrophy is said to have occurred.

Inflammation of the mammary glands and of the vulva sometimes occurs in girls and more rarely of the ovaries.

Diagnosis.—The diagnosis usually presents no difficulties, and any doubt is usually cut short by the acuteness of the attack. Certain enlargements of the cervical lymphatic glands resemble contagious parotitis, and in scrofulous children the swelling in the latter is sometimes prolonged, but the physiognomy is different and distinctive. There is more swelling in front of the ear in parotitis and in the first stage a triangular shape is produced with the apex downward, while the lobe of the ear is raised in a characteristic manner.

Prognosis.—The prognosis is favorable, no fatal cases of uncomplicated mumps being recorded.

Treatment.—No means of shortening the duration of the disease exists. The patient should be kept uniformly warm, and to this end the bed is desirable. It is usual to anoint the gland with some simple ointment, as cold cream, and it may be that the feeling of drawing and tension is thus relieved. No commensurate advantage results from leeching. It is thought by some that the so-called metastasis is occasioned by exposure to cold, and if this be true, there is even better reason for keeping the patient warm. Warm applications are generally better borne than cold. Cotton or wool or flannel warmed and greased gives a sense of comfort. Fever should be treated by appropriate remedies and other symptoms met as they arise.

Secondary Parotitis.—This term is applied to parotitis occurring as a complication in acute disease, typhus and typhoid fever being the most frequent. It may be a complication of pyæmia, phthisis, and carcinoma. Except in pyæmia, when it is metastatic, it is probably caused by the bacteria of decomposing matters in the mouth, which reach the gland through the duct of Steno.

It is a much more serious affection than mumps and often terminates in suppuration. Facial paralysis may result from destruction of the facial nerve, or there may be deafness from invasion of the middle ear.

The **treatment** of secondary parotitis is that of phlegmonous inflammation elsewhere.

INFLUENZA.

Synonyms.—*Catarrhal Fever; Grip; Fr., La Grippe.*

Definition.—Influenza is an acute infectious disease characterized by fever, by catarrhal irritation of any or all of the mucous tracts, especially the respiratory, by muscular pain, and by great prostration. It is commonly epidemic.

Historical.—Although influenza appears to have prevailed as early as 1173 in Italy, Germany, and England, it was not until 1510 that it was

recognized in its true light as an epidemic or pandemic disease. Since that date it has recurred at intervals of from four years to one hundred years. Up to 1870 more than a hundred epidemics had been described. It first appeared in the United States in 1627, in Massachusetts and Connecticut, and extended thence over South America as far as Chili. Since 1889 there has been an epidemic extending almost around the world. It usually begins in the east and travels westward. The last epidemic started in Bokhara in May, 1889, reached St. Petersburg in October, Berlin in November, London in December, and the eastern cities of the United States by the middle of December. While its rate of travel is rapid, it is not more so than travel itself. Its spread is not influenced by the direction of prevailing winds. It travels as rapidly against the wind as with it, some observers say more rapidly. A district invaded in the fall of the year is apt to be infected more or less for several months.

Etiology.—In 1892 Pfeiffer discovered in the pus cells of tracheal mucus an organism which he regarded as that of influenza 0.8 to 0.1 μ long and 0.1 to 0.2 μ broad, *i. e.*, about the same width as the bacillus of mouse septicæmia and half as long. It forms colonies on glycerin agar visible under the microscope twenty-four hours after inoculation as clear, water-like drops. These drops do not coalesce, but remain separate. The bacilli are best stained in dilute Ziehl-Neelsen solution of carbol fuchsin or hot Loeffler methylene blue solution. P. Canon has even found them in the blood.

The origin of the bacillus is not settled. But evidence is accumulating to indicate that the disease is contagio-infectious, rather than miasmatic-infectious, the bacillus coming from another person having the same disease. I thoroughly believe that influenza is contagious, though I thought differently at one time. The fact that it travels only as fast as people travel, and that it does this contrary to the direction of prevailing winds, are strong points in favor of this opinion. The fact that inoculations have thus far been unsuccessful in transmitting the disease is, however, against its contagious nature. The *complications* and *sequelæ* of the disease, pneumonia, pleurisy, endocarditis, and the like, may be the result of the action of a toxin, or the bacillus may be transmitted in the blood to the seat of secondary infection.

Morbid Anatomy.—The anatomical changes are those of the complications. Whatever alterations are the direct result of the disease itself for the most part promptly disappear after death.

Symptoms.—Influenza has a period of incubation of from two to three days, which may be longer. It attacks all ages, infancy less commonly, more frequently those from twenty to forty years old. The mode of onset is by no means constant. The attack may be ushered in by a *chill* or continued chilliness. This is, however, often absent. Most frequently, perhaps, there is *coryza* and *sneezing* with or without watering of the eyes. To this succeeds *cough*, to which is commonly added, very soon, copious expectoration. The cough may be paroxysmal and be attended with prostration at the end of the spell. It is often persistent, while the bronchitis may pass into *broncho-pneumonia* or a *croupous pneumonia* may supervene. Less frequently there may be faucitis, simple, however, and not accompanied by ulceration or white patches. These

symptoms are more or less associated with *muscular pain*, although not invariably. At other times the attack begins with severe *pain* in the back or back of the head, the chest walls, the extremities, or throughout the muscular system. Such pain is sometimes severe and sudden.

Another mode of onset is by an extreme and *sudden prostration.* I have known a man to step from a railroad station, apparently well, and in the course of a few hundred yards become so weak as to have to take a car to reach his home, though not distant. This prostration is apt to be prolonged even in mild cases far beyond what seems reasonable. *Mental depression* is a frequent symptom, and suicide and even manslaughter have been its terminal acts.

There is always more or less *fever.* Commonly it is slight at first, but sometimes very high. I have known it to be 106.2° F. (41.2° C.) at the first observation of a patient. More frequently it does not exceed 103° F. (39.4° C.), and it is often but slightly above normal. During convalescence the temperature may become subnormal, and in the patient alluded to there was a fall from 106° F. (41.1° C.) to 96° F. (35.6° C.) in a very short space of time. Further, the temperature chart may exhibit fantastic changes. *Delirium* is sometimes associated with the fever and may come on suddenly and actively.

While pulmonary catarrh is perhaps the most frequent catarrhal manifestation, it is by no means always present, even when there are pulmonary symptoms. I recall an obstinate case of bronchial spasm without any secretion whatever. In the epidemic, especially of 1893–94, in Philadelphia and vicinity *gastric catarrh* was frequent, producing distressing nausea with vomiting, and adding greatly to the physical weakness. Severe vomiting may even usher in the attack, especially in children. More rarely there is diarrhœa.

Complications.—The most serious complication is pneumonia. It is often invited by exposure during convalescence or in the attempt of a patient to fight out the disease without giving up. In these events it is usually ushered in by a chill and extends rapidly through the whole of one lung or even both lungs. When a part of the primary attack, the pneumonia is more apt to be catarrhal and circumscribed, creeping from the bronchi into the air-vesicles, and is less serious, although it may also be fatal, especially in old persons. At other times the inflammation is confined to the minute bronchioles, and we have the physical signs of a capillary bronchitis. Endocarditis, pericarditis, irregularity of the heart without evident endocarditis or pericarditis, are also complications and sequelæ. Sudden heart failure is to be remembered as a possible and not very infrequent cause of death, as I have reason to know from experience.

A most important fact to be remembered in this connection is the tendency of influenza to develop latent disease into acute disease, and to make slight grades of organic affections more serious. This is particularly seen in connection with heart disease and kidney disease. A small albuminuria with no other symptoms becomes, after an attack of influenza, an incurable and rapidly increasing Bright's disease.* A mild cardiac

* See a paper by G. Baumgarten on "Renal Affections Following Influenza," in Transactions of the Association of American Physicians, Vol. x, 1895.

affection scarcely noticeable by its symptoms may become a grave affection with degeneration of muscular substance and dilatation of the cavities.

Diagnosis.—The diagnosis is ordinarily easy, although doubtless during an epidemic many cases are called influenza which are cases of simple bronchitis, angina, or nasal catarrh. The diagnostic features in addition to the catarrhal factor are the fever of short duration, extreme prostration, and the muscular rheumatic pains. I have more than once thought of a case in its incipiency that it was going to be one of typhoid fever, but the absence of the typical temperature of typhoid, of epistaxis, of diarrhœa, and the shorter duration of the illness turned the scale in favor of influenza.

Prognosis.—The prognosis is generally favorable, especially if the patient goes to bed at once, or at least houses himself. Such a one is almost sure to be well in three, four, or five days. It is possible, however, for one attacked to fight through the disease without losing a day's time. But especially unfortunate is he if he fails in this attempt because of taking cold or inability to hold out longer against the debilitating effect of the disease. In the former he is apt to have pneumonia, in the latter he has to contend with extreme prostration. The prostration of the epidemic variety is something peculiar. The weakness is extreme, and the slightest effort, physical or mental, promptly convinces the patient of this. The duration of the weakness may be greatly prolonged, months being sometimes necessary to overcome it.

Treatment.—The treatment in the majority of cases is very simple. Rest in bed, without medicine, answers for a large number. Beyond this the treatment is mainly symptomatic, phenacetin or antipyrin being generally sufficient to subdue the pains when present. Quinine is necessary in almost every case to keep up the strength. In ordinary cases requiring such treatment, I am in the habit of giving five grains (0.324 gm.) of phenacetin every four hours, alternating with two grains (0.120 gm.) of quinine as often, omitting the former when the pain has disappeared but continuing the quinine. When the pains are very severe, the phenacetin may be given more frequently and even in larger doses. Larger doses of quinine may be needed. The cough may be treated with turpentine stupes and sinapisms to the chest, and when there are positive laryngeal symptoms, "Dobell's solution" sprayed into the larynx is very useful. It may also be sprayed into the nasal passages, or cocaine may be applied locally. Internally, the officinal solution of citrate of potassium in f ʒss (15 c. c.) doses every two or three hours. Where the cough is disturbing, small doses of morphine may be necessary, and if secretion has set in, ammonium chloride in five to ten grain (0.324 to 0.648 gm.) doses, with 15 minims (one gm.) of syrup of squills and two drachms (7.4 c. c.) of compound licorice mixture are sufficient to answer the purpose. If more stimulating effect is required on the secretion, the aromatic spirit of ammonia in half-drachm doses (two gm.), or carbonate of ammonium in doses of five to ten grains (0.324 to 0.648 gm.). Opium may be given in large doses, or morphine in corresponding doses, to relieve pain if required. For the prostration, supporting measures are necessary, and even stimulants may be called for. Whiskey and milk are efficient. The entire absence of appetite

and the complaint that all things taste alike are to be ignored, and the patient must be encouraged to take food, which should be made as attractive as possible. Strychnine is an admirable heart tonic and may be given, $\frac{1}{30}$ grain (0.00216 gm.), every six hours.

Treatment for the pneumonia, which becomes often so grave a complication, is often extremely difficult. In a few cases it is a true "pneumonia fulminans"—strikes the patient down so suddenly and violently as to make all treatment unavailing. Referring the reader for details to the section on pneumonia, it may be said that, as a rule, in the pneumonia of influenza, stimulating and restorative measures of a very positive character are indicated rather than depressing agents. The free use of alcohol and ammonia is especially necessary. Dry cupping and blistering are, in my judgment, never out of place in pneumonia, for they can do no harm, if they do no good. They should be followed by a jacket of wool, to maintain warmth and a uniform temperature.

One need not wait for the physical signs of a pneumonia to present themselves before beginning the treatment. Given a chill after exposure, with no other cause to explain it, and you may consider a pneumonia as almost inevitable. Oftentimes a pneumonic focus in the centre of a lung does not furnish any physical signs, while to wait until it approaches the surface causes a delay in the treatment which may be fatal.

Other complications of influenza are treated as when they are simple diseases. Over-medication should be avoided.

CEREBRO-SPINAL FEVER.

SYNONYMS.—*Epidemic Cerebro-spinal Meningitis; Spotted Fever; Petechial Fever.*

Definition.—An infectious disease of sporadic and epidemic occurrence, probably microbic in origin and especially characterized by inflammation of the membranes of the brain and spinal cord.

Historical.—Cerebro-spinal fever is a disease of modern recognition, for a long time confounded with typhus, and even with our present knowledge at times difficult to distinguish from it. Its distinct recognition dates back no farther than 1805, when Vieusseux pointed it out as a separate disease in Geneva, under the name *fièvre cérébrale ataxique*. It appeared the next year in this country as an epidemic at Medfield, Mass.; again at Grenoble, France, in 1814. It disappeared on both sides of the Atlantic in 1816. It reappeared at Vesoul, France, in 1822–3, prevailing more particularly in barracks, whence it extended to other places more or less until 1849. It prevailed in Italy from 1839 to 1845, and in Algiers from 1839 to 1847. In 1844 a short epidemic visited Gibraltar, a longer one Denmark in 1845–48. It occurred in a mild form in Great Britain in 1846 and malignantly in Sweden in 1854; in Norway in 1859–60; in Holland briefly in 1860–61. Northern Germany was again invaded in 1863, Southern Germany in 1864, and Baden and Hesse in the same year; Austria and Russia mildly a year or two later, and in 1868 Turkey and its adjacent possessions mildly. Canada appears not to have been invaded until 1870.

In the United States another visitation occurred in 1822–23 in Middletown, Conn., and again in 1842, since which it has prevailed more or less in all the States, being especially severe in 1863–65 in Philadelphia and throughout the State of Pennsylvania. It has lingered in Philadelphia ever since, isolated cases being annually reported. The number of deaths in that city from 1863 to 1891 inclusive, as collected by Alfred Stillé and completed by William Pepper, was 2575.

Etiology.—The direct cause of cerebro-spinal fever is believed to be a microörganism not as yet isolated, possibly of more than one variety, including a lancet-shaped bacillus like the pneumococcus. That the infectious agent originates in the person affected is at least doubtful, the disease not being as a rule traceable to another having it, but appearing to arise rather in certain houses or localities where the necessary conditions prevail.

Predisposing causes are cold, moisture, exposure, defective sanitation. Crowded buildings, barracks, and tenements have been favorite localities, especially in Europe. The fatigue of long marches and depressing influences favor it. During the civil war in America both armies were largely affected, but the mortality was not large. Sometimes the disease prevails in the country rather than in the city. It is more common in the young, attacking even infants of less than a year old. Sex and race seem to have no influence.

Morbid Anatomy.—The external appearance of the body after death is not peculiar. Most characteristic are the remnants of the eruption, petechial or herpetic, but they are not constant. The *brain* and *spinal cord* are naturally the seats to which we look for morbid changes, and we find every degree of inflammatory condition, from slight hyperæmia, such as may be found in any form of infectious disease, to a stage in which pus and fibrinous deposits, more particularly in connection with the pia mater, are abundantly present. Higher degrees of hyperæmia involve even the calvarium as well as the dura. The arachnoid spaces may contain serum and pus, but it is under the pia mater that we look for the inflammatory products, serous, fibrinous, or purulent, especially at the bottom of the sulci in the longitudinal and Sylvian fissures and at the base over the pons, the chiasm, and cerebellum. To a less degree the convexity of the brain is also involved, and even the brain substance may share in the hyperæmia, while actual softening has been noted. Adhesions between the pia and the cortex are common, withdrawal of the former carrying the substance of the cortex with it. More rarely there is an effusion into the ventricles and the choroid plexus is congested. The walls of the ventricles may be softened, and in cases of long standing there is even hydrocephalus.

The *cranial* nerves, especially the auditory and optic, may be the seat of a neuritis or bathed in pus infiltrating the lymph-sheaths. The muscular and trophic phenomena resulting from such involvement may be permanent.

The *spinal membranes* are similarly hyperæmic, even to the extent of extravasation of blood at times. The same inflammatory products are found upon them as on the meninges of the brain. They are more frequently seen on the posterior aspect of the cord, but may be general.

Ounces of pus have been removed from the spinal canal. Even the central spinal canal has been found dilated and filled with pus. There may be likewise inflammation of the substance of the cord. The roots of the spinal nerves may be compressed by exudate, producing localized paralysis, or may be themselves the seat of a neuritis, whence the characteristic clonic muscular contractions often present, while the irritation of the sensory roots gives rise to more or less intense pain. Certain malignant cases are of so short duration that there is no time for morbid changes to occur. In such the results of necropsy are negative.

As to *other organs*, there is no characteristic involvement. The spleen may be normal in size or if the illness has lasted some time it may be slightly enlarged. There may be congestion of the liver, kidney, stomach, and intestines, and even extravasation of blood. The same is true of the lungs, in which there may be bronchitis or pneumonia, the latter not very rarely. Endocarditis and pleurisy are sometimes found.

Symptoms.—Cerebro-spinal fever does not present an unvarying picture in its symptomatology, and to attempt to portray every unusual symptom would occupy undue space. Only the most characteristic symptoms will be given, first of the ordinary form and then of the most important modifications of it.

I. *The Ordinary Form.*—No definite time of incubation is known. A *prodromal period* of short duration with *headache* and *pain in the back* or headache and *vertigo* may precede, but the onset is often sudden and associated with a decided *chill*. Headache and pain in the back of the neck and back are, however, more characteristic. Though usually severe, this pain is sometimes so slight as to cause the real condition to be overlooked. It is sometimes so sudden and severe as to be compared to the sting of a bee.

Vomiting is also a frequent early symptom. There is *fever*, but the temperature does not usually exceed 102° F. (38.9° C.). There is nothing characteristic in the fever, and no regular curve of evening rise and morning fall. On the other hand it is extremely irregular. The pulse is full and strong. *Hyperæsthesia* of the skin is a characteristic symptom. It is sometimes extreme, and as the disease increases in severity, *rigidity* of the muscles of the neck and back sets in. This muscular contraction may cause backward curvature of the head and even opisthotonos. Clonic spasm may also occur, though less frequent than tonic contraction. It is more common in children, in whom it may amount to convulsion and take the place of the chill. Spasm of the muscles of the face may occur and of the eye muscles, causing strabismus. On the other hand, there may be *paralysis* of the face muscles and of the eye, more rarely of the trunk. The auditory nerves may be involved, affecting the *hearing*.

Delirium is constant; occurs early in the disease, and rapidly passes into stupor or coma. The delirium is often maniacal, considerable effort being necessary to control the patient.

It has been stated of the *temperature* in this disease that it is rarely high. In some of the earliest descriptions of the disease—and there have been most interesting ones written almost a century ago—the

writers speak of the skin as being cool. This was before the days of the clinical thermometer and the accurate measurement of temperature growing out of it. High temperatures do occur, though rarely, 105° F. and 103° F. (40.5° and 41.1° C.) being noted and others even higher just before death.

The *pulse* goes hand in hand with the temperature, that is, it is not very frequent, at least in adults, at first. As the disease advances it grows in frequency rather as the result of an increasing debility of the patient. So, too, the *breathing* is not apt to be markedly influenced unless there be a lung complication. *Jaundice* has been met with.

The *urine*, as in other infectious fevers, may be scanty and albuminous; but it may also be increased, doubtless because of the involvement of the nervous system. For a like reason there is sometimes glycosuria, and occasionally in severe cases Cheyne-Stokes breathing.

Another characteristic symptom is the *eruption*, although it is not present in more than one-half the cases. It is of at least two kinds, herpetic and petechial. Herpes labialis, although not always present, is nevertheless more constant than in pneumonia. The herpes may be noted elsewhere than on the face, viz., on the trunk and extremities, extending exceptionally even to the ends of the fingers. The contents of the vesicles may be purulent; they may coalesce and break and dry, forming crusts. The petechial eruption is more general. It is an extravasation, and like the similar eruption in typhus does not disappear on pressure. The number of spots varies greatly. There may be only a few, or they may be very numerous, fully justifying one of the names of the disease—spotted fever. It will not do, however, to exclude the disease by reason of the absence of these skin symptoms.

Other eruptions, as urticaria, sudamina, rose-colored spots like those of typhoid fever, pemphigus, and ecthyma, have been noted. Gangrene of the skin has occurred as the result of pressure. Some trophic influence may, however, also be responsible for it.

Sometimes the disease sets in with *diarrhœa*, though more commonly there is *constipation*. The tongue is less apt to be dry than in typhus, probably because the patient is less disposed to breathe through his mouth.

II. *Malignant Form.*—The malignant form of cerebro-spinal fever is characterized by the suddenness of its onset and severity of its cardinal symptoms,—the chill, headache, coma, collapse,—followed by early fatal termination. There is little or no fever; indeed, the temperature may be sub-normal. The pulse is feeble and slow, falling to 50 to 60 a minute, increasing, however, in frequency if the patient lives. The breathing is labored. The urine is scanty and albuminous. But for the prevalence of the epidemic such fulminating cases would not be distinguished from like attacks of other infectious diseases. Such cases may, however, occur even sporadically. They may last but a few hours. They are more frequent in the beginning of an epidemic.

III. The *mild form* presents a corresponding mildness of symptoms and only the presence of an epidemic leads to its recognition.

IV. The *abortive form* terminates abruptly after a sharp development of characteristic symptoms.

V. The *intermittent form* is characterized by remissions and exacerbations in the fever every day or second day, without, however, the regularity of intermittent fever. The fever resembles rather that of pyæmia.

VI. Finally, the term *chronic form* is applied to cases prolonged beyond the usual duration, in which the headache, gastric irritability, and vague neuritic pains reduce the patient to such an extremity of exhaustion and emaciation that he welcomes death as a relief to his suffering; or partial recovery may take place with crippled motion, defective senses, and devouring pains which are a constant source of discomfort.

Diagnosis.—The diagnosis in epidemic cases is usually easy, although it is more than probable that under such circumstances some cases are classified as cerebro-spinal fever when they are really something else. During epidemics typhus fever is the disease with which it is most frequently confounded, especially as epidemics of typhus and cerebro-spinal fever sometimes prevail jointly. The difficulty is greatest at the beginning of the attack, for as time passes the diseases diverge in symptoms. Typhus fever is not characterized by the severe pains in the head and back of the neck, nor by opisthotonos, both of which may, however, be absent in cerebro-spinal fever, or be so slight as not to attract attention. In typhus fever the spots are more constant and numerous than in cerebro-spinal fever. Herpes does not occur in typhus. The typhoid state may be equally pronounced in both, but in general it may be said to be more marked in typhus. The two diseases differ in their duration, typhus having a pretty definite duration of about two weeks, whereas cerebro-spinal fever is either shorter or longer.

Among the diseases which embarrass is muscular rheumatism. More frequently than with typhus fever, perhaps, is it confounded at the onset with this disease. The muscular pains are similar, but the headache in cerebro-spinal fever is a point of difference. Hence, too, as the disease advances the diagnosis becomes plainer.

The isolated cases give most trouble. Typhoid fever, especially the meningeal form of typhoid, in which there is extreme headache and active delirium, simulates cerebro-spinal fever in the beginning not a little, and I have known consultants to hold different views for some days. With the lapse of time, however, the diagnosis may generally be made. The onset of typhoid is also slow; there is not apt to be vomiting or severe muscular pain.

Pneumonia is another source of confusion, especially as the two diseases are sometimes associated, and it is almost impossible to say which is primary. The bacteriological examination is of no assistance, because the *bacillus lanceolatus* is found in both. Further, there are meningeal complications in pneumonia which increase the difficulty, but as these are confined to the convexity there is muscular contraction and tremor, but not retraction of the head.

Tubercular meningitis presents some resemblance to cerebro-spinal fever. While usually less sudden in its development, it is not always so. Delirium and stiffness of the neck, retraction, and even opisthotonos occur. There are, however, no skin symptoms. The termination of tubercular meningitis is invariably fatal. The presence of a focus of tuberculosis is of great value in the diagnosis.

Influenza, too, in one of its many forms occasionally simulates cerebro-spinal fever at times very closely. Extreme muscular pain is characteristic of both, and when influenza is associated with actual cerebro-spinal meningitis, with delirium and stupor, as it sometimes is, one may be excused for being in doubt. Although both are diseases of short duration, influenza spends its fury earlier, and is thus a shorter disease unless prolonged by one of its complications. This feature, if other characteristic symptoms are wanting, may help us to a decision.

Complications and Sequelæ.—Of the complications of cerebro-spinal fever, croupous pneumonia has already been mentioned as not infrequent as well as that it is sometimes difficult to say which disease is primary. The initial chill and herpes are characteristic of both affections, and close attention to other conditions must be given, such as the presence or absence of an epidemic, the order of appearance of the symptoms, the nervous and muscular preceding in cerebro-spinal fever and coming on later in pneumonia. Other complications are those which not infrequently accompany infectious diseases, including endocarditis, pericarditis, polyarthritis, with possible suppuration, and others.

Of sequelæ the most important are blindness due to optic neuritis and more rarely keratitis, deafness from disease of the labyrinth, paralysis in circumscribed areas, as the face, aphasia, defective articulation, persistent headache, shooting muscular pains, mental weakness. Next to scarlet fever cerebro-spinal meningitis is the most frequent cause of deafness. Even chronic hydrocephalus and abscess of the brain are included among sequelæ. Von Ziemssen says the former is indicated by "paroxysms of severe headache, pain in the neck and extremities, without vomiting, loss of consciousness, convulsions, and involuntary discharges of fæces and urine."

Prognosis.—Cerebro-spinal fever is a grave disease, but the mortality varies greatly in different epidemics, ranging from 20 to 75 per cent. according to Hirsch, while v. Ziemssen places it for mild epidemics at 30 per cent., and for severe ones 70 per cent. The death rate is higher for children, those under two years almost invariably perishing while few over five survive. The old likewise succumb easily.

Of few diseases is the course more variable and uncertain. From a duration of two to three days only it may be prolonged to weeks and even months, and its consequences may be permanent. Usually, however, improvement may be looked for if the patient survives five days, more than half the deaths occurring within this period. A remission of symptoms may take place on the third day, to be followed after a very short time by a relapse. This often misleads and gives the illusive hope of permanent improvement. Convalescence is characteristically slow, the symptoms yielding gradually. If the termination is fatal the cardinal symptoms likewise gradually subside, but are replaced by growing debility and exhaustion.

Relapses are prone to occur, prolonging the case indefinitely, while a chronic or protracted form, sometimes spoken of, is probably due to the presence of one of the persistent or progressive lesions above referred to.

Treatment.—The treatment of cerebro-spinal fever is sympto-

matic and supporting. The symptom demanding the promptest relief is the pain, and for this there is no substitute for opiates, and of these the best preparation is morphine, and the best mode of administering it is by hypodermic injection. Doses sufficient to accomplish their purpose should be given, say $\frac{1}{4}$ grain (0.016 gm.) to $\frac{1}{2}$ grain (0.032 gm.), night and morning for an adult. The tolerance for the drug is great. It may be combined with $\frac{1}{120}$ grain (0.00054 gm.) to $\frac{1}{100}$ grain (0.00064 gm.) of atropine. The same preparation may be given by the mouth if the hypodermic administration is not convenient, but the deodorized tincture of opium may be better borne, and where the more frequent administration of opiates is necessary, as hourly or bi-hourly, this preparation is to be preferred because of the possible harmful effects of the too frequent use of the hypodermic syringe. The action of the drug is, of course, to be carefully watched. Phenacetin, antipyrin, salicylic acid, and this class of drugs are no substitute for opium in this painful malady.

Where there are spasms or convulsions there is no remedy equal to chloral. If it cannot be administered by the mouth, a drachm (4 gm.) dissolved in two ounces (60 c. c.) of water may be given to an adult, without hesitation, per rectum. In extreme cases chloroform or ether may be inhaled for the same purpose. The bromides may be used as adjuvants in mild cases, but of themselves are altogether inefficient.

Cold may be applied to the head for the headache and other meningeal symptoms, and is best used in the shape of an ice cap or ice bladder or Leiter's coil. Cold may also be applied to the back of the neck and spine. Counter-irritation to the back of the neck and spine has long been employed, chiefly by blisters. At the present day it is regarded as of doubtful propriety. The Paquelin cautery, which has been of late much recommended as a substitute for the blister, can do no harm applied to the back of the neck. The inconvenience is less than is commonly supposed, and the ulcer heals rapidly. Cupping and leeching in the same localities followed by warm fomentations may be useful. They relieve the pain for a time at least. General bleeding is not to be recommended.

Quinine may be given in tonic doses of six (3.8 gm.) to eight (5.9 gm.) grains, but not for any specific end, while large doses are harmful, causing irritation. Measures of a very decided character to reduce the temperature are not, as a rule, needed. Simple sponging suffices for the most part. Should this be insufficient, however, tub bathing may be used, as in typhoid fever.

The nourishment should be of the best, including animal broths and milk, and where, as is frequently the case in the early stages, they cannot be tolerated by the stomach, they may be given peptonized per rectum, not more than four ounces (120 c. c.) at one time. I have thus nourished for several days until the stomach became retentive a case despaired of, which ultimately recovered. Forced alimentation by the stomach-tube is recommended by Heubner. Alcohol is contra-indicated in the early stages unless there be unusual adynamia. Later, when exhaustion begins to show itself, it may be used and pushed as under similar conditions in other diseases.

There are no specifics which have sustained their primary reputation.

The bichloride of mercury and iodide of potassium have been most praised, and the former drug may be administered from the onset with some reasonable expectation that it may be useful in doses that need not be harmful if not beneficial. Such doses would be $\frac{1}{24}$ grain (0.0027 gm.) every two or three hours for an adult, suitably reduced for children. Mercurial inunctions, which have been much used, are still recommended by v. Ziemssen, although he admits them to be of doubtful efficacy. The iodides are indicated in the later stages when there are symptoms of exudation.

DENGUE.

SYNONYMS.—*Break-bone Fever; Dandy Fever.*

Definition.—Dengue is an epidemic, infectious, possibly contagious disease, characterized by paroxyms of extreme pain in the joints and muscles, accompanied by fever and sometimes eruptions on the skin.

Historical.—Dengue was recognized as a distinct disease in the latter part of the eighteenth century, first in Spain in 1764–68. In 1780 an epidemic prevailed in Philadelphia, which was described by Rush under the name of bilious intermitting fever. In 1824 it prevailed in Calcutta, in 1827 and 1828 in Charleston, Savannah, and in New Orleans, U. S., and in the West Indies, and was described by the late Prof. S. H. Dickson, then of Charleston. Since then there have been numerous epidemics, for the most part south of the thirty-second parallel of latitude.

Etiology.—McLaughlin, of Texas, claims to have found in the blood of a dengue patient a micrococcus, which he holds responsible for the disease, but the effect of inoculations requires to be studied. Analogy would lead us to suspect such an organism, while experience justifies a like conclusion. It spreads as a disease thus caused spreads, by the routes and means of travel. It attacks both sexes and all ages, regardless of season, although warm climates are its natural habitat. No morbid anatomical changes have been associated with the disease.

Symptoms.—Dengue is usually sudden in its onset, after a period of incubation lasting from three to five days, during which there may be some *sense of discomfort* (more frequently there is not), *headache*, and even *chilliness*. Suddenly, often at night, the patient is struck with *pain* in the muscles and joints, and especially the muscles of the back and loins. The pain is searching, as though extending into the bones themselves. The smaller as well as the large joints are affected, and the pain is aggravated on motion. The suffering is extreme, and it may be said that the patient is literally racked with torture.

Simultaneously there are *headache* and *fever*, the former severe and the latter quite high, rising rapidly to 102°, 103°, 105° F. (38.9°, 39.4°, 40.5° C.), and even 106° or 107° F. (41.1°, 41.6° C.), reaching its maximum the second to the fourth day, then declining, reaching the normal about the fifth day. The face is flushed, the conjunctiva congested, the pulse is frequent, 100 to 120, rising and falling with the fever. Delirium is not a marked feature save in children. The tongue is coated and red at the tip and edges, there are loss of appetite, slight nausea, and extreme

thirst, scanty urine, and constipation; at times, however, the urine is copious and clear. Hemorrhage from the nose and gums has been noted, and both Eugene Foster and D. C. Holliday have seen black vomit similar to that of yellow fever, and in one case copious hemorrhage from the bowels, which persisted three months and terminated in death, was observed.

As intimated, at the end of three or four days the temperature falls, the pain subsides, and a short period of comparative comfort, though one also of great prostration, succeeds that of great suffering. It is during this remission that an erythematous rash makes its appearance on the face, neck, and shoulders, and thence over the whole body in two or three days. At the same time the lymphatic glands at the back of the head and neck, in the axillæ and groins, swell, with some return of fever. The eruption is not constant or always uniform. It lasts from a few hours to a couple of days, when it subsides with the second febrile movement and true convalescence sets in. The eruption may also reappear, though rarely.

Diagnosis.—On account of the joint involvement, associated, as it often is, with redness, dengue has not inexcusably been mistaken for acute rheumatism; but the decided remission in a couple of days, the altogether short duration of the disease, and its epidemic character, should soon extricate the physician from such confusion. The absence of any glandular swelling or eruption in rheumatism and the more close limitation of the pain to the joints aid in the discrimination.

After rheumatism, influenza is perhaps the next disease with which dengue may be confounded. It, too, is epidemic, and is attended often by extreme and sudden muscular pains, but the sudden intermission characteristic of dengue does not occur in influenza, nor does the eruption or gland swelling.

Prognosis.—Notwithstanding the extreme suffering, recovery is the invariable rule.

Treatment.—Nothing can be done to cut short the disease. The most satisfactory method to control the pain is by the hypodermic injection of morphia and belladonna. One-fourth of a grain (0.016 gm.) of the former and $\frac{1}{150}$ (0.00044 gm.) of the latter may be given, supplemented by phenacetin and antipyrin, in doses of ten grains (0.66 gm.) of the former and five (0.33) of the latter, every two hours, when the hypodermic injection may be repeated if relief has not been obtained. The coal tar derivatives are also the best remedies for the fever, but they may be supplemented by sponging with cool water, or the cold bath in extreme cases. Prostration must be met by alcoholic preparations.

ERYSIPELAS.

Synonyms.—*The Rose; St. Anthony's Fire.*

Definition.—An acute, infectious, primarily local disease, characterized by a dermatitis attended with the usual signs of inflammation,—swelling, heat, pain, redness, and a peculiar disposition to spread.

Historical.—Erysipelas is described by Hippocrates (B. C. 480),

who had a remarkably clear conception of the disease. Its parasitic origin was first maintained by Henle (1840). Trousseau first asserted, in 1848, that an abrasion of the skin is an invariable condition of its origin. Hueter, in 1876, was especially conspicuous in claiming that the disease owes its existence to a microörganism residing in the blood. Billroth and Klebs held similar views, but it was reserved for Koch, in 1880, to settle the question by finding the specific streptococcus in the lymph vessels and lymph spaces of the skin, though not in the blood. Fehleisen made the same discovery independently of Koch in 1881, isolating and cultivating the erysipelococcus and inoculating man with it. Orth had previously made experiments of the same kind on animals.

Etiology.—The *streptococcus erysipelatis* of Fehleisen is a minute, cleft fungus, a micrococcus in the narrow sense, three to four μ in diameter, arranged in pairs (dipplococci) or chains (streptococci) of from six to 12 cells. The erysipelococcus resembles very closely the streptococcus pyogenes of Rosenbach,—in fact, cannot be distinguished from it microscopically, while even the cultures of the two organisms resemble each other very closely. The streptococcus pyogenes grows more slowly and less uniformly, according to Hoffa, than that of erysipelas, and presents also a brownish discoloration in the middle of its colony. They behave very similarly when inoculated in animals. Simon asserts that the micrococcus of erysipelas is identical with that of pyæmia. Klebs suggests that more than one organism may be concerned in the causation of erysipelas.

The organism probably operates as a local irritant producing the dermatitis. From this as a focus constitutional infection, as in diphtheria, is set up, probably through the influence of a toxic agent generated by the micrococcus. It is found in the lymph vessels and lymph spaces of the periphery only of the inflamed area, and not in the centre, by which fact the spread of the process by peripheral extension is explained.

The organism is transferred from one person to another by direct contact, or by the intermediation of a second person, or through the atmosphere. It cannot be said, however, that the disease is highly contagious in the absence of surgical injury, for in my early experience as a hospital interne at the Pennsylvania Hospital, and later as a visiting hospital physician in the Philadelphia Hospital, though it was the custom to keep the erysipelas cases in the ordinary medical wards, I cannot recall a single instance where the disease was communicated to another patient in the ward. It was very different, however, in the surgical wards, where the disease would spread rapidly from one patient to another, showing the importance of the open surface as a condition of the spread of the disease. The lying-in woman is very readily inoculated, so that no physician should attend a case of labor while attending one of erysipelas. Certain kinds of wounds, as lacerated wounds, and wounds in certain situations, as the scalp, are especially prone to erysipelas. Clean-cut wounds in other locations suffer less frequently. Leech bites, the wounds of the cupping scarificator and of the subcutaneous syringe, are also favorable starting points. Chronic inflammatory processes and skin diseases may also have erysipelas engrafted upon them.

Erysipelas is prone to occur in the epidemic form, more especially in the spring of the year and in old and unclean hospitals, but such epidemics have become much rarer in the last twenty years. This is doubtless one of the results of antisepsis, now so generally practised. The feeble, the intemperate, and those having Bright's disease, or other affections weakening natural resistance, are more prone to the disease. An interesting case of Bright's disease under my care in the Philadelphia Hospital had frequent attacks of facial erysipelas, always accompanied with hæmaturia. Relapses and recurrences of erysipelas are prone to occur, and a person once attacked by erysipelas, far from being protected, is rather predisposed to a second attack. A family predisposition to erysipelas may exist.

Morbid Anatomy.—Like all acute inflammatory states of the skin, erysipelas fades away after death and leaves little, if anything, to be seen unless it has proceeded to the formation of blebs or abscesses. Swelling and proportionate deformity of the part, especially of the face, when extensive may remain, but even this subsides with the lapse of time after death and may totally disappear.

Minute examination finds the cocci in the lymph vessels and spaces at the periphery of the inflamed area, as already stated, and even in the uninflamed tissue beyond the margin.

Various complications attend erysipelas and add their morbid anatomy to that which is more essentially that of the disease. The most important of these are pyæmic abscesses of internal viscera and hemorrhagic infarcts of the lung, spleen, and kidneys. The kidneys are especially apt to be congested, and the lesions of acute or subacute nephritis are sometimes found.

Symptoms.—The form of erysipelas which more particularly concerns the physician is the so-called *idiopathic erysipelas*, which arises independently of any apparent traumatic lesion, but since all erysipelas implies some lesion, however minute, the term is a misnomer. The fact remains, however, that the physician is most frequently called upon to treat the form of erysipelas in which there is no discoverable local lesion.

There is a period of incubation of from one to eight days, after which this variety of erysipelas begins as a *small, red, burning spot* a few lines in diameter, usually on the face, oftenest on the bridge of the nose or the chin. It spreads rapidly, and as soon as sufficient size is attained there is a very characteristic elevation of the patch above the surrounding tissue, which can be recognized by carrying the finger across it. This is of diagnostic value. The future extension of the process is upward over the forehead and laterally toward the ears until the whole face, and more rarely also the neck, is invaded. The eyes become closed by *swelling*, the features are distorted, and the sum of changes produces an appearance not soon to be forgotten. In other parts of the body, as the arms and legs, the same process may go on, but there is not the unsightly distortion found as in the case of the face and head. In some cases the process proceeds to suppuration, and deep-seated *abscesses* form. These, of course, must result from mixed infection with other pyogenic organisms. *Blebs* form, particularly on the lobes of the ears and eyelids, while little vesicles are always visible to a lens. From these a serum may

exude and dry on the skin. As the dermatitis extends to new areas the earlier spots dry up and desquamate. The disease seldom lasts more than four days in one spot, although it may revisit the same spot during the same attack.

This local invasion is commonly, though by no means always, ushered in by a *chill* or succession of chills associated with general feeling of discomfort and *loss of appetite*. Often the rose-red, burning spot is the first symptom noticed. *Fever*, however, probably always precedes, though not noted in the beginning, and it rapidly becomes higher, mounting as high as 105° F. (40.5° C.). There is a corresponding frequency of pulse, associated with *headache* and sometimes *delirium*. The fever continues as long as the disease continues to spread. Often a sudden drop, a crisis, occurs on the fifth to the seventh day, followed by another rise if the disease takes a fresh start.

In more serious cases fever and delirium may be followed by *drowsiness* and *stupor* and a *coated, dry tongue*, all the symptoms, in fact, of a typhoid state. The *urine* is scanty and a febrile *albuminuria* may be present,—in fact, to a degree, may be said to be constant,*—and nephritis sometimes results, while a pre-existing one may have an acute exacerbation engrafted upon it. Mention has already been made under the head of Etiology of *hæmaturia* occurring in these cases.

Grangrene may be associated with the deep-seated varieties, constituting gangrenous erysipelas.

There may be erysipelas of the mucous membranes, which may extend to the skin, or the reverse may take place,—infection from the skin to the mucous membrane.

Complications and Sequelæ.—The possible complications are numerous, but in practice are really not frequently encountered. The most frequently mentioned is meningitis, the result of extension by continuity through the openings of the cribriform plate of the ethmoid bone or by contiguity from the scalp through emissary veins of the skull, but I have never seen such a case. William Osler, however, traced the extension from the face along the 5th nerve to the meninges, causing an acute meningitis and thrombosis of the lateral sinus.

Œdema of the glottis is the result of extension of the disease to the mucous membrane of the glottis. It is promptly fatal.

Mycotic or malignant ulcerative endocarditis is also with comparative frequency secondary to erysipelas, three cases out of 23 being sequelæ of this disease. Pericarditis, endocarditis and myocarditis, bronchitis, pneumonia, and pleurisy may also be mentioned as possible complications; also jaundice, dysentery, and hemorrhages from the nose and bowels. Nephritis of hemorrhagic variety has already been mentioned, and even glycosuria has been noted, possibly an accidental complication. Septic and pyæmic complications do, however, occur and are among the causes of death.

Among the sequelæ may be mentioned a loss of hair. Cicatricial new formations replace the parts destroyed by gangrene and may pro-

* See paper by J. M. DaCosta on "The Internal Complications of Acute Erysipelas," Amer. Jour. of the Medical Sciences, October, 1877.

duce deformity by their contraction. On the other hand, hyperplastic new formations resembling elephantiasis arabum may result. Hyperæsthesia and neuralgia of the involved areas, anæsthesia with which atrophy of the skin may be associated, symmetrical gangrene of the fingers, and painful affections of the joints have all occurred as sequelæ.

Erysipelas may be associated with other infectious diseases, such as typhoid and typhus fever, diphtheria, scarlet fever, and the like.

Diagnosis.—The diagnosis of erysipelas is usually not difficult, although many conditions are called erysipelas by the ignorant which are not of this nature. The acuteness of the disease, the rapidity of its spread, the constitutional disturbance, and fever distinguish it from other conditions which superficially resemble it.

Prognosis.—The prognosis, in the vast majority of instances, is favorable. Only in the aged, the intemperate, and those of broken health from other causes does it prove fatal as a rule. Complications, especially meningitis and septic states, are causes of death. On the other hand, erysipelas is said to exert a favorable influence on certain acute diseases, such as acute rheumatism, choroiditis, and even morbid growth. It has even been suggested to inoculate erysipelas for the cure of such affections.

Treatment.—It is more than likely that a decided majority of cases of idiopathic erysipelas would get well without any treatment whatever. In other words, the disease is self-limited. As the disease is exhausting, internal treatment should be of a restorative and supporting character. Quinine, iron, nutritious food, and stimulants are indicated, while the patient should be kept at rest. The tincture of the chloride of iron is used throughout North America because of some supposed specific influence over the disease, and doses as large as a drachm every three or four hours have been given. I have always given iron, but never in such doses, and I am doubtful whether it exerts any specific effect of the kind claimed. The natural duration of the disease is short, and the effect claimed from the iron is no prompter than that which nature brings. Ten minims (0.666 gm.) every two or three hours are a sufficient dose, and it is exceedingly doubtful whether larger quantities than this are absorbed.

J. M. Da Costa first suggested the use of pilocarpine in the treatment of erysipelas, more particularly in the early stages. J. L. Salinger,* A. A. Eshner, and S. D. Barr also report favorably on the same treatment, which should, however, be employed cautiously. It is recommended that $\frac{1}{6}$ grain (0.01 gm.) be administered hypodermically every three hours until free sweating ensues. After this the interval is increased to four or six hours.

An infinite variety of local measures has been suggested to arrest the spread of the disease, all of which are useless to this end, although some of them are useful in allaying the burning. For this purpose I know nothing better than the old-fashioned mixture of lead water and laudanum in the proportion of four parts of the liquor plumbi subacetatis dilutus, U. S. P., to two of laudanum. Or a mixture may be made of

* Therapeutic Gazette, March 15, 1894.

acetate of lead, opium, and water. Lead-water alone is an efficient local application for this purpose. So is cold water. In the military hospitals in Philadelphia during the late civil war a cranberry poultice was a favorite application, and it was certainly a pleasant, cooling measure, but a waste of a useful fruit. Dusting the surface with finely levigated oxide of zinc or subnitrate of bismuth also has a soothing effect. A rational measure would be, as has been suggested, the hypodermic injection of a two per cent. solution of carbolic acid, or a weak solution, say one to 2000, of corrosive sublimate, just beyond the edge of the advancing dermatitis, but it has never seemed to me necessary, while it is painful and annoying to the patient.

DYSENTERY.

SYNONYM.—*Bloody Flux.*

Definition.—Dysentery is a term applied to any form of inflammation of the large bowel. It may be acute or chronic. There are three distinct varieties :—

(1) Catarrhal.
(2) Amœbic.
(3) Diphtheritic.

Historical.—Dysentery is a disease known for centuries and is frequently referred to in Biblical history. Its severest forms are found in tropical countries where epidemics are numerous and where it is said to cause more deaths than cholera. Sporadic cases also happen everywhere. Severe epidemics also occur in temperate zones, and it is a disease which attacks armies and ships' crews, although its frequency is gradually diminishing from year to year. It prevails almost invariably in summer and autumn and is especially frequent in malarial localities.

CATARRHAL DYSENTERY.

Catarrhal dysentery is the simplest and most common form in temperate climates. It consists in a simple catarrhal inflammation of the large bowel in a portion or throughout its entire extent.

Etiology.—This form may undoubtedly be caused by simple irritants, of which unripe and indigestible food forms a liberal source in this country. To the country physician attacks of dysentery in children who have eaten of unripe fruit in the early autumn are familiar events. Impurities in drinking water have long been regarded as causes, but in modern times there is a disposition to regard such impurities not of themselves as potential factors, but as favoring the operation of other causes. Changes of temperature act similarly, as also do dyspeptic states and constipation.

Morbid Anatomy.—The morbid anatomy of catarrhal dysentery is simple. The mucous membrane of the large bowel is smeared with bloody mucus, and when this is scraped away the blood-vessels beneath are injected and the solitary follicles are found enlarged. Occasionally the latter suppurate or sphacelate, leaving open ulcers.

Symptoms.—Dysentery is usually ushered in by *diarrhœa*, the first stools being copious and painless. Soon, however, these are replaced by small mucous discharges streaked with blood and accompanied by crampy abdominal pains and straining, technically known as *tormina* (twisting pains) and *tenesmus*. The latter is exceedingly trying, causing a constant feeling of unsatisfied attempt at stool, so that the patient is disposed to sit constantly on the closet or to go back repeatedly many times in a single hour, experiencing at the same time intense burning pain in the rectum. Yet the total quantity discharged in the twenty-four hours is not large. Twenty-eight to 42 ounces are a full estimate.

Sometimes a *chill* is the initial symptom. The *tongue* is furred and at first moist. Later it may become dry. There may be *nausea* and *vomiting*.

There is always more or less *fever*, sometimes very slight at others decided, the temperature seldom exceeding 103° F. (39.4° C.). There are the thirst and acceleration of pulse usually attending fever, and sometimes the former is extreme. The *abdomen* may be tender, but not necessarily so. It may be tumid or flat and hard. In addition to the features of the stools already mentioned, which are characteristic, *scybala* or hard fecal masses may be present at first. Later the stools are frequently green in color, from the presence of bile—bilious dysentery—and increase in their transit the burning feeling in the rectum already mentioned. In addition to blood-corpuscles and leucocytes the microscope recognizes large, round, and oval epithelioid cells containing fat drops and vacuoles; also at times the *cercomonas intestinalis*. No specific organisms are found in the simple catarrhal form of dysentery.

The milder cases of catarrhal dysentery are self-limiting, terminating usually in a week, when the character of the stools changes. Other cases are more intractable and resist even judicious treatment for a long time, becoming even chronic.

Diagnosis.—The diagnosis of acute dysentery is very easy. The tormina and tenesmus with the frequent blood-stained mucous stools occur in no other affection. Malignant disease of the rectum is sometimes mistaken for chronic dysentery. Examination of the rectum should be made in all prolonged cases.

Prognosis.—The prognosis of catarrhal dysentery is generally favorable. As intimated, many mild cases get well without treatment, and when judiciously handled unfavorable termination is rare. Favorable termination is not, however, invariable, and cases sometimes end unfavorably after a prolonged course, or they become permanently chronic and incurable. Emaciation and exhaustion are rapid, and even a mild attack reduces its victim decidedly.

Treatment.—The first treatment of catarrhal dysentery duly recognized should always be a purgative. None is better than castor oil. An ounce of oil (30 c. c.) guarded by ten to 20 drops (0.66 to 1.33 gm.) of laudanum is the proper dose for an adult. The saline treatment, especially where there is high fever and no marked adynamia, is also efficient, working a rapid cure in many cases. Two drachms (8 gm.) of sulphate of magnesium or ½ ounce (16 gm.) of Rochelle salts dissolved in water should be given every hour until copious watery purgation results.

When this end is obtained by either remedy an opiate may be given. Plain opium in doses of a grain (0.066 gm.) every three hours, or ½ grain (0.033 gm.) of the extract, is the favorite. Or the drug may be combined with bismuth subnitrate in ten grain (0.66 gm.) doses, or with one of the astringents, tannic acid in two to five grain (0.132 to 0.33 gm.) doses, or the acetate of lead one to two grains (0.066 to 0.132 gm.). Opium suppositories containing one to two grains (0.066 gm. to 0.132 gm.) of opium or ½ grain to a grain (0.033 gm. to 0.66 gm.) of the extract may be useful, in addition, to quiet the rectal irritation.

Hope's camphor mixture is an old remedy which sometimes acts well, especially in cases disposed to become chronic. Dr. Hope's formula, originally suggested in 1826, is as follows :—

℞. Acidi Nitrosi, ... f℥j (3.7 c. c.)
Mist. Camphoræ, ... f℥ viij (240 c. c.)
M. et adde Tr. Opii, ... gtt. xl (1.2 c. c.).
S.—A fourth part to be taken every three or four hours.

The Hope's camphor mixture of the shops made with nitric acid instead of nitrous should not be substituted.

It goes without saying that the food should be liquid and of the blandest kind,—boiled milk, better peptonized, light animal broths, and beef juice, not beef-teas, are the type. Barley or rice may be added to such broths and should be thoroughly cooked.

Amœbic or Tropical Dysentery.

This form of dysentery, occurring for the most part in tropical and subtropical countries, is ascribed to the *amœba coli or dysenteriæ*, an organism first noted by Lambl in 1859 and later by Lösch, but first claimed to be the specific organism of dysentery by Kartulis, who found it constantly present in the stools of the victims of an epidemic in Egypt in 1883. The last observer cultivated the amœba in decoctions of straw and inoculated it in cats and dogs, producing certain symptoms of dysentery. Since then it has been constantly found, not only in the stools, but also in the tissue of the bowel and in the pus of the hepatic abscess secondary to dysentery. It is an active amœba, from 15 to 30 μ in diameter, and is easily recognized by moderate powers of the microscope. It is held by Lutz of Honolulu to be the cause of certain individual cases of dysentery and not of epidemic dysentery. It has been found in the stools of healthy men, and probably enters the organism in drinking water. Cases are not rarely met in the hospitals of Baltimore and Philadelphia, but their subjects probably acquired the disease in the South.

Morbid Anatomy.—The lesions are of a more decided character than those of simple acute catarrhal dysentery, consisting in ulceration, more or less extensive and œdematous, and cellular infiltration of the submucous coat of the bowel, which appears first as hemispherical swellings above the mucous membrane. The latter sloughs away, leaving ulcers at the bottom of which is the infiltrated submucosa appearing as a greyish, gelatinous mass, which subsequently exfoliates, forming a still deeper ulcer with the muscular or even sometimes the serous coat as its floor. The ulcers are round, oval, or irregular, with undermined

edges, so that the visible aperture is often small compared with the extent of destroyed tissue to which it is the entrance. The sloughing away of this infiltrated though sound-looking mucosa produces extensive irregular ulcers, which may be met with in any part of the large intestine or cover almost the whole of it. Nor is the process limited to this gut, but even may invade the lower part of the ileum. The most striking histological character of the infiltrate is the absence of pus-cells in large numbers. The fixed connective-tissue cell is, however, described proliferated.

If the patient dies at a more advanced stage efforts at healing may be found more or less extensive, the contraction of the scar-tissue producing irregularities and inequalities ; also sometimes polypous elevations of the mucous membrane, known as pseudopolyposis. Stricture of the rectum is also a possible result.

Symptoms.—The symptoms of amœbic dysentery are similar to those of catarrhal dysentery, but much more irregular and prolonged. The onset is usually less sudden, but may be equally so. The stools are less numerous, and are apt to be more liquid and more copious. They abound in the *amœbæ coli.* The straining is less severe and persistent, while there may be several days of relief, to be followed by the usual train of symptoms. The fever may be severe or mild.

Complications.—The most common and serious complication is abscess of the liver, which is now believed to be due to the wandering amœba dysenterica, which reaches the liver through the blood-vessels. The abscess may be single or multiple. In the former case it may be of large size, involving fully half of the bulk of the liver. The multiple abscesses are smaller in size and superficial. The abscess walls are peculiar, being ragged from the presence of necrotic projections. Only occasionally in the older abscesses are there firm, smooth fibrous walls. Next to the inner necrotic zone is a zone of cellular infiltration encroaching upon and destroying the liver-cells, and external to this again a zone of intense hyperæmia. The contents of the abscess are not pure pus. In fact, the paucity of the pus-cells here is as significant as in the inflammatory infiltration of the mucosa, indicating a similarity in the etiology. The pyoid material consists rather of fatty and granular debris, and the amœbæ which are also found in the walls of the abscess. These abscesses sometimes break into the lungs, carrying the amœbæ with them, which are sometimes under these circumstances found in the expectoration.

In addition the abscesses described there are found also in the liver in amœbic dysentery patches of circumscribed necrosis, scattered through the liver as the result of the action of the amœbæ.

Diagnosis.—The diagnosis is rendered easy by the recognition of the amœba coli in the stools, which should be examined by the microscope in every case of dysentery.

Prognosis.—The prognosis is much more serious than in the catarrhal variety. The course of the disease is always prolonged, and a fatal issue is much more frequent. It would seem that the patient must outlive the organism before he can recover, and even then recovery is delayed by the exhausted condition into which he has fallen in the

struggle with his microscopic host. When the termination is most favorable, cases of amœbic dysentery last from six to twelve weeks.

Treatment.—The same indication as to diet exists in this as in the other forms of dysentery. It is apparently in this form of dysentery, of which only isolated cases are met in temperate climates, that the ipecacuanha treatment of the East Indian physicians has been so successful. It is claimed to act as a muscular sedative and secretory stimulant, the former by allaying the exaggerated peristaltic activity so characteristic of the disease, the latter by augmenting the secretion of mucus as well as stimulating the activity of the liver-cells in bile formation, a function which in dysentery is in abeyance. Great stress is laid on the mode of administration. A preliminary dose of laudanum is given, and in half an hour afterward from 20 to 60 grains (1.332 to 4 gm.) of ipecac. For three hours after the first dose only a little ice should be sucked, and after that a little iced soda-water and milk administered. Beef-tea or bread or light foods are fatal to the favorable action of ipecacuanha, and to the use of such foods failures are ascribed by the advocates of the treatment. On the second day the drug is administered in reduced quantity, supplemented by salicylate of bismuth, quinine, naphthol, and opium, while milk should form the staple food. Later, farinaceous foods and soups may be carefully given, but no solids should be permitted for a long time.

Warm injections of quinine, one to 5000, one to 2500, and one to 1000, have been employed at the Johns Hopkins Hospital with great benefit, the amœba being rapidly destroyed by them. Perhaps ipecacuanha acts similarly. For the relief of pains opiates must also be administered, preferably by the rectum in suppository or small starch-water enemata. Or morphine may be given hypodermically if the stomach is sensitive.

Diphtheritic Dysentery.

Diphtheritic dysentery is by far the most serious of all the forms of dysentery.

Etiology.—Diphtheritic dysentery is commonly conceded to be due to the action of a vegetable organism which enters the system as a constituent of foul drinking water. Ogata has described a bacillus which he regards as the cause of an epidemic in Japan. It has been isolated, cultivated, and introduced with food or injected into the lower bowel of animals, where it has produced ulcers and hemorrhages in the colon, swelling of the mesenteric glands, and nodular masses in the liver and spleen. It appears not to be the Klebs-Lœffler bacillus.

There is also a milder variety of diphtheritic dysentery characterized as secondary, because it occurs as a complication in numerous acute and chronic diseases. It is said to be not infrequent in chronic Bright's disease, but in a comparatively large experience with this affection I have never met it. Chronic heart disease and chronic cachectic states generally are said to be prone to it. Of acute diseases with which it is associated pneumonia is the most frequent. Diphtheritic dysentery is the variety which prevails in epidemic form, especially where large num-

bers of persons are congregated,—as in armies, on ships, in insane asylums, and almshouses.

Morbid Anatomy.—The characteristic anatomical feature of diphtheritic dysentery is the false membrane and its consequences. In the milder form it appears in the shape of a thin, yellowish membrane capping the folds of the colon. In the severe forms it covers extensive areas of the large bowel and is thick and adherent. It is a coagulation-necrosis of a deep-seated infiltration of the mucosa including the follicular glands in its embrace, and when shed leaves extensive, deep ulcerated surfaces. These also make an attempt at healing, and the cicatrices in their contraction produce irregularities and deformities on the surface of the bowel, including also pseudopolyposis and rarely stricture.

Symptoms.—The symptoms are those of the simple catarrhal form greatly intensified. The *fever* is higher, the *pain* is greater, the *tormina* and *tenesmus* are more severe, the *stools* more bloody, and the *adynamia* more profound. *Delirium* is often present. The abdomen is tender and swollen, and typhoid fever may be simulated. The symptoms in the secondary form are less severe than in the primary.

Complications and Sequelæ.—The complications in this form of dysentery are more numerous. Abscess of the liver is also one of them, and is ascribed to thrombotic extension from the seat of inflammation along the vessels of the portal system into the liver, or to emboli carried from the primary focus to the liver. Perforation of the bowel is not a very rare complication, having been found by Woodward in a study of the statistics of the late civil war in America 11 times in 108 autopsies. This accident is followed by a peritonitis, which is usually fatal, the local symptoms of which vary with its exact seat. If in the neighborhood of the cæcum perityphlitis ensues, if lower down in the rectum a proctitis. A peritonitis may also arise by extension of the inflammation from the mucous lining of the bowel.

The same opportunities have enabled Woodward to show the undoubted association of malaria with dysentery, though it is likely that the "chills" referred to in older reports were sometimes septic and due to the dysentery. The same is true of the joint swelling described by the older authors, among whom was Sydenham. They may be a part of pyæmic processes. Paralysis, commonly paraplegia, as a sequel is attested by Woodward and Weir Mitchell. Pleurisy, pericarditis, endocarditis, and Bright's disease are among sequelæ reported.

Diagnosis.—The same diagnostic symptoms which enable us to recognize the other varieties of dysentery attend this in severe degree, but it is the occurrence of successive cases that give the stamp by which we recognize the diphtheritic type.

Prognosis.—The prognosis of this form of dysentery is the most unfavorable of all the varieties. Most cases perish, death being preceded by extreme adynamia and other symptoms of the typhoid state, including dry tongue, stupor, emaciation, and the cadaveric countenance. Consciousness is sometimes painfully persistent to the end.

Treatment.—The first consideration in the treatment of diphtheritic dysentery is a bland and non-irritating but nourishing diet, one that leaves as little residue as possible. The peptonized foods, such as pep-

tonized milk and beef peptonoids and beef juice, are the types. To these stimulants should be freely added. Opiates are needed to relieve the pain, and their hypodermic use is sometimes especially useful for this purpose. When the necrotic membrane is removed an extensive ulcerated surface remains to be healed. Such healing is favored by the restrained peristalsis which opium produces. The same purpose may be served by the use of ipecacuanha if the effect claimed for it by the East Indian surgeons is produced. On the other hand, it is doubtful whether soluble remedies intended for the direct healing of the ulcers ever reach these surfaces in an active state when administered by the mouth. Bismuth, being largely insoluble when administered in large doses, undoubtedly reaches the bowel, and may doubtless produce some healing effect. More promising is the use of iodoform, which can also be expected to reach the lower bowel in a similar manner, and which is not only more healing in its action, but is also antiseptic. It may be given in a pill or capsule in doses of ½ to three grains (0.0324 to 0.194 gm.).

Chronic Dysentery.

Any one of the forms of dysentery described may become chronic.

Morbid Anatomy.—All the lesions described as occurring in the described varieties of dysentery may be present. The most common is ulceration, which is variously extensive and exhibits also efforts at healing. On the other hand, cases of chronic dysentery are met with in which there are no ulcers whatever. The coats of the bowel are thickened, especially the submucosa and the muscularis, while patches of black and slate-grey discoloration are scattered through it, the result of blood extravasation and disintegration. Puckering, pseudopolyposis, and cystic degeneration due to occlusion of the follicular glands may be present.

Treatment.—The patient should be put to bed on a diet easy of assimilation and furnishing a minimum of waste. Its quantity should be just what is needed and no more. From what has been said it may be inferred that I have little confidence in methods of treatment the object of which is to get remedies to the diseased bowel by way of the mouth. Bismuth in large doses and iodoform may, however, be tried for the purpose; half a drachm to a drachm (two gm. to four gm.) of bismuth should be given at a dose, so that from 12 to 15 drachms are reached in the course of a day. Iodoform may be given as above directed.

The topical treatment of chronic dysentery by way of the rectum is that on which most reliance is placed at the present day. Its object is to get remedies to the diseased part. To this end they are dissolved and their solutions are introduced into the lower bowel. Nitrate of silver is the favorite remedy, but alum, sulphate of zinc, sulphate of copper, and acetate of lead are also used in the same doses. Twenty to 30 grains (1.32 to 1.98 gm.) are dissolved in a pint (liter) of water, and from three to six pints (liters) are introduced at one time through a long tube introduced gently well up into the bowel, but at the onset weaker solutions and smaller quantities are injected. The patient should be placed on his back with the hips elevated by a pillow, so that there may

be the coöperation of gravity. I have recently had a good opportunity to use this treatment in the wards of the Hospital of the University of Pennsylvania and confess to disappointment in the results. My cases improved to a certain point, but none got well. The treatment is sometimes painful.

Very decided counter-irritation to the abdomen by iodine and even by blisters is sometimes of decided benefit. At least these measures seemed to mark the turning point in the disease.

SEPTICÆMIA AND PYÆMIA.

Definition.—Septicæmia and pyæmia are general febrile infections caused by the entrance into the blood of toxic substances generated by pathogenic bacteria. The former term is used when the condition is unattended by suppuration, pyæmia when abscesses are present. The bacteria usually responsible are the pus-producing organisms, viz., the *streptococcus pyogenes* and *staphylococcus pyogenes*. The two conditions are essentially the same, both being septic states induced by a like cause, the presence or absence of pus foci as a consequent symptom constituting an anatomical difference. Both present different degrees commensurate with the amount of septic product absorbed.

Septicæmia.

While the pus organisms are most frequently responsible for the *intoxication* of the blood by their pathogenic products or toxines, from the medical standpoint the term septicæmia may be applied to the toxic condition produced by any of the pathogenic bacteria which invade the blood and tissues with or without a visible site of infection. Milder degrees of septicæmia have been described as fermentation fever and *sapræmia*, but the latter is now regarded as equivalent to septicæmia.

Symptoms.—Illustrative cases of the more usual form of septicæmia are puerperal fever following retained placenta, infection by scarlet fever or erysipelas, or difficult labor involving laceration, and the poisoning by a dissecting wound. The symptoms set in in from three to four hours to three or four days, more frequently within twenty-four hours.

The usual initial symptom is a *chill* varying in severity. It may, however, be preceded by malaise, headache, and slight feverishness. It is succeeded always by high *fever*. In bad cases the chill is repeated daily, perhaps more than once a day, and the temperature rises to 105° F. (40.5° C.) or higher. The patient is restless; the tongue, at first red and glazed, becomes dry and leathery; the pulse, at first full and bounding, becomes weak and compressible, with a rate of 130 or more. Prostration soon becomes marked. The fever is subject to decided remissions, which give rise in the inexperienced to delusive hope. The chill is commonly followed by profuse *sweating* and further *prostration*. The mind early begins to wander and *delirium* is soon decided, becoming low and muttering as the disease is established, and is ultimately replaced by unconsciousness.

There is tenderness of the abdomen in puerperal cases; in all a tendency to *enlargement* of the *spleen*, with hypostatic *congestion* of the *lungs* and possible broncho-pneumonia.

Diagnosis.—The diagnosis is usually easy, and even if at first obscure may be arrived at by a careful study of cases. The resemblance of the more serious form to intermittent fever has been referred to when treating of the latter disease, but in septicæmia the remissions are not so total, and the patient remains very ill notwithstanding their occurrence. More difficult is it to distinguish the milder degrees which produce a continued fever, from typhoid. (See "Simple Continued Fever" at end of Section I.)

Prognosis.—The prognosis in the milder degrees is favorable; in true septicæmia very unfavorable, the patient sometimes dying within twenty-four hours, while recovery is scarcely known except in the milder grades resulting from dissecting wounds.

Treatment.—In treatment all possible infecting foci should be removed by surgical measures; after this the strength should be supported by quinine, strychnine, and stimulants. The food should be liquid and of the most nourishing kind. Antipyretics may be used to reduce temperature, but it is better to accomplish the same thing by hydro-therapy. (See also Treatment of Pyæmia.)

Prophylaxis is much more efficient than treatment, and with modern aseptic surgery and aseptic obstetrics, septicæmia is becoming much more infrequent.

Pyæmia.

Definition.—Pyæmia is a general febrile affection due to sepsis, characterized by recurring chills and profuse sweats, with remissions or intermissions in the fever; also by abscesses variously disseminated in different tissues and organs of the body.

Etiology.—The same essential cause lies at the bottom of pyæmia as of septicæmia, but associated with the former as important etiological factors are thrombosis and embolism. To this association Virchow first drew attention, and it is to these factors that the pyæmic abscesses are due. Fragments of a venous thrombus due to a phlebitis at the seat of putrid inflammation are broken off and carried in the circulation until a lodgment is effected. These fragments swarm with bacteria, and the embolus excites intense inflammation which goes on to abscess formation—the metastatic or embolic abscess. Emboli may be multiple and there will be as many abscesses as lodged emboli. It will be remembered that the non-infectious embolus produces simple hemorrhagic infarct.

The seats of election in their order of frequency are as follows: The lungs, liver, spleen, kidneys, brain, and joints, the subcutaneous connective tissue, pelvic connective tissue and subperitoneal connective tissue; the marrow of the long bones and the parts about the cavity of the middle ear. A frequent source of multiple abscesses is the interesting disease, mycotic endocarditis, better known as ulcerative endocarditis, itself a specific inflammation caused by some pathogenic septic organism floating in the blood and lodging on the heart valves, where it excites a septic valvulitis. The vegetations produced by this are broken off and

become emboli. These are carried through the arterial system to points of lodgment and constitute the arterial pyæmia of Wilks. The term idiopathic pyæmia is applied to that form in which multiple abscesses exist with the other symptoms of pyæmia, but no infective focus is discoverable.

Abscesses occur in the lungs when the septic emboli originate in osteomyelitis or in inflammatory affections of the periphery ; in the liver, where they arise from septic foci in the portal area, especially in the intestines ; the pelvic connective tissue, when they start in the uterus and its appendages ; in the spleen, kidneys, and brain, if the emboli arise in the left heart, or are so small that they can pass from the right heart through the lungs to the left heart.

Symptoms.—A rapidly rising *fever* is the first symptom of pyæmia, often so closely followed by a *chill* that its previous existence is not suspected. The severity of the chill is proportionate to the intensity of the infection and the degree of inflammation which results from it. The temperature during the chill reaches 103° to 104° and 105° F. (39.4° to 40° and 40.5° C.) and is followed by a *sweat* and fall of temperature, after which the latter again rises to a point even higher than that first attained. Then follows another sweat and fall and thereafter a succession of intermissions variable but quite characteristic. The rise is generally toward evening, and thus there is a certain resemblance to typhoid fever, while the rigors and sweats suggest malaria. There are other symptoms of fever, viz., *thirst*, *loss of appetite*, and *nausea*. The *strength* of the patient rapidly wanes, he soon sinks into a condition of exhaustion and semi-consciousness, from which, however, he may be aroused to take medicine and nourishment.

The various local involvements cause *localized symptoms*. Emboli in the lungs cause cough and hurried breathing, but there may be no distinctive physical signs. In the liver they may cause tenderness and enlargement with jaundice ; if in the kidney, there is probably no sign, though there may be albuminuria and hæmaturia ; if the intestines, diarrhœa ; if in the skin, superficial abscesses ; if in the joints, swelling, tenderness, and fluctuation ; if in the brain, little is added to the existing nervous symptoms. There may also be secondary abscesses of the parotid gland and pancreas, the former producing hard, painful swelling and the latter deep-seated pain in the epigastric and umbilical regions.

The *abscesses* contain the pyogenic bacteria which are responsible for them. Not all cases are promptly fatal. There is a form of chronic pyæmia which lasts for months, in which the symptoms are less distinctive and in which the history of an infected wound may be the only cue to its real nature. One such case came under my observation, that of a young physician who received a dissecting wound from which the symptoms started and which terminated fatally with meningitis after many months' illness.

Diagnosis.—The diagnosis is not usually difficult, though sometimes the disease is overlooked and the symptoms ascribed to some other cause. Reference has already been made to the resemblance of the disease to typhoid fever and malarial fever, but the physician should not be

long in doubt; a careful study of the case will show marked differences in history, while the status præsens exhibits only a superficial resemblance. There are no rigors followed by sweats in typhoid fever, as a rule, and the temperature chart in pyæmia is much more irregular. The suddenness of the pyæmia is characteristic, though it is by no means invariable. In remittent fever the chill and fever and sweat are more regular, the prostration is not so extreme, and, above all, it is promptly cured with quinine. The plasmodium, if found, definitely settles the question as to the malarial fever. There should be no confounding of pyæmia with simple intermittent fever. The complete absence of symptoms between paroxysms is in no way comparable to the evident desperate illness despite the temporary absence of fever in pyæmia.

Among the causes of pyæmia which have been overlooked is osteomyelitis. Gunshot injuries of bones and compound fractures, if followed by suspicious symptoms, should lead to investigation. Mycotic or ulcerative endocarditis is often overlooked, and not without reason, as it is so often overshadowed by other symptoms. A cardiac murmur with irregular temperature and sweating, with unusual prostration should excite suspicion.

Gonorrhœa and prostatic abscess are occasionally causes, as are also tuberculosis of the kidney and calculous pyelitis, the last two, perhaps, more frequently than the first two.

Prognosis.—The prognosis is very grave. Even when recovery takes place in comparatively mild cases, it is with shattered health. More fortunate are the rarer instances of recovery after puerperal pyæmia, which, when they do occur, are more apt to be complete. Where calculous pyelitis and even tuberculous pyelitis are causes, operation often furnishes prompt relief, which is more or less complete.

Treatment.—The treatment is like that for septicæmia. First remove, if possible, the primary surgical focus and relieve secondary foci as they appear. After that the symptoms are to be combated and the strength supported to the utmost. To the latter end the most nutritious and easily assimilable food, quinine in liberal doses, alcohol freely, and strychnine are the sheet anchors. To these may be added sponging to lower the temperature; atropine, oil of erigeron in doses of 10 to 30 ♏ (0.65 to two gm.) in a capsule or on sugar; ergot, 15 to 30 minims (one to two gm.); agaricin, one to two grains (0.05 to one gm.); the dilute mineral acids, 15 to 30 minims (one to two gm.), to check the sweating, which adds to the exhaustion.

Among the more favorable cases, in which operative treatment is followed by prompt and sometimes more than temporary relief, are cases of septicæmia originating in vesical and prostatic disease and calculous and tuberculous pyelitis. In tuberculosis of the kidney, as tuberculosis elsewhere, especially illustrated in the peritoneum, exposure to the air seems to have a destructive influence upon the bacillus.

HYDROPHOBIA.

SYNONYM.—*Rabies.*

Definition.—Hydrophobia is an acute infectious disease of animals, communicable to man, and characterized by intense tonic spasm beginning in the larynx.

Historical.—The disease was known to the ancients, including the East Indians, Egyptians, and Israëlites, but is not mentioned by Hippocrates (B. C. 460–357), though Democritus, living about the same time (B. C. 470–362), is said to have considered it an affection of the nerves allied to tetanus. Aristotle (B. C. 384–322), however, recognized it in dogs. According to Celsus, who wrote about the date of the Christian era, it was named *ὑδροφοβια* by the Greeks. The Latin poets, Virgil, Horace, and Ovid, mentioned it, as did also the historian, Plutarch (*circa* A. D. 50–106), and Galen (A. D. 130–200) and Cælius Aurelianus (fourth century A. D.) discussed it. William Youatt first described it with accuracy in the lower animals and man.* Pasteur first showed its infectious character, and ascribed it to a toxine developed by a microorganism as yet undiscovered.

Etiology.—The dog is the most frequent subject of the disease, and it is from him that it is almost invariably communicated to man. The wolf and cat are also subjects and communicate the disease to human beings by their bites. In such cases, whatever the contagium may be, the contagium bearer is conceded to be the saliva of the animal. The contagium is a fixed and not a volatile one. The researches of Pasteur go to show that it is also contained in the central nervous system, especially the medulla and brain. Klebs suggested that the disease is caused by a bacterium found in the salivary glands of those affected with hydrophobia. Gibier, Fol, and Babès claim to have found micrococci in the brain substance, but these claims have not been confirmed by others, though their experiments have been repeated. There can scarcely be a doubt, though, that an organism is the agent of infection.

The period of incubation is extremely variable, ranging from one week to two months or longer. Even two years have elapsed before symptoms set in. The average is said to be from six weeks to two months; but by no means all persons bitten take the disease, a most important point to be remembered in estimating the efficacy of supposed curative measures. Not more than 15 per cent. of those bitten, according to Horsley, become affected. Various causes contribute to this. Thus the saliva may be wiped off in the transit of the tooth through the clothing, and such removal of virus may reduce the danger of the second bite of the same animal, even though it be on the unprotected skin. Again, the young seem to be more susceptible. Bites on the face and hands are more frequently followed by infection than those on the lower extremities and remainder of the body. Statistics by Watson, in America, and by Bollinger, in Germany, show more cases to have resulted from bites in the upper extremities, while according to

* "Canine Madness," being a series of papers published in the Veterinarian, 1828, 1829, 1830. London, 1830.

Horsley wounds about the face and head are more apt to cause the disease than on the hands, which are second in order, and after these other parts of the body.

To a very important practical question, How long after a bite may the dreaded suspense of an expected outbreak last? accurate answer seems scarcely possible. Yet notwithstanding the fact that cases are recorded of an outbreak after an interval of two years, it may be said with confidence that if three months have elapsed after the bite, the victim may feel assured that he is safe.

Morbid Anatomy.—The morbid anatomy of rabies, as far as recognized, is limited to the upper spinal cord, medulla, pons, and cortex of the brain, and is revealed only to the microscope. The blood-vessels are dilated and congested, the perivascular sheaths are invaded with leucocytes, and there are even small hemorrhages. Often there are no discoverable changes.

Symptoms.—Rabies is usually divided, from its clinical standpoint, into two varieties, *furious* or *convulsive*, and *dumb* or *paralytic* rabies. W. H. Welch, of Johns Hopkins University, suggests, with good reason, a third form of *mixed* rabies, representing a combination of convulsive and dumb rabies. The variety common to human beings is the furious or convulsive, though paralytic rabies also occurs in man, especially after bites on the lower extremities, and would seem to be increasing in proportion to the convulsive form. So, too, in dogs furious rabies is the more usual, while in rabbits the paralytic form is more common.

It is true, also, that a sharp distinction cannot always be made between the two forms, while a stage of excitation and stage of paralysis may be made out in the same case, and it amounts largely to this, that in the furious form the stage of paralysis may be short or wanting, while in the paralytic form the stage of excitement may be short and may be manifested only by acceleration of breathing, elevation of temperature, and symptoms referable to irritation of the vagus nerve. The most reliable observations go to show that there is no difference in the quality of the virus producing the two forms, but that the latter are due rather to differences in the individual, the seat of inoculation, or perhaps the quantity of the virus.

The *first* or *premonitory stage* succeeds upon the period of incubation, and lasts about twenty-four hours. The *cicatrix* of the bite, which has been for some time healed, may become painful or the seat of irradiating pain, or become livid, or even break out again. The patient is *morbidly depressed* or irritable, is *feverish*, *loses appetite*, and is *sleepless;* there is hoarseness or huskiness of voice. A feeling of intense anxiety and a moodiness are very characteristic, his probable fate being the sole subject of contemplation. There is an increased excitability, as a result of which the banging of a door or flash of light causes the patient to start.

The *second* or *spasmodic* stage is the true hydrophobic stage, setting in usually after the first twenty-four hours. It is also called the furious stage. The sum of its symptomatology depends upon an *exalted irritability* of the *muscles of the larynx*, as the result of which they contract upon the slightest irritation in their vicinity, the act of swallowing being

the most frequent exciting cause. Attempt at swallowing is followed by the most *powerful contraction* associated with *dyspnœa*, even when the glottis is open or tracheotomy has been performed; whence the *fear of water*, the contact of which with the throat is followed by such frightful spasm of the muscles of the larynx and elevators of the hyoid bone. Even the saliva, which is secreted in increased quantity, cannot be swallowed without paroxysms. Hence it is discharged from the mouth, sometimes forcibly, giving rise to the popular idea that the patient is frothing at the mouth. The paroxysm may be associated with maniacal excitement, in which the patient is sometimes uncontrollable, rolling his eyes, striking about with his arms, and making snapping noises with the mouth, which are compared to the biting of dogs. They are altogether due to uncontrollable spasmodic shutting of the mouth. On the other hand, between the paroxysms, when the mind is clear and the reason sound, there is often found a touching concern on the part of the patient lest he does some harm to those whom he loves. There is more decided feverishness in this stage, the temperature rising as high as 103° F. (39.4° C.), while the pulse is frequent and sometimes irregular. Albuminuria and glycosuria have both been found in this stage. The second stage lasts from one to three days, sometimes a little longer.

In the *third* or *paralytic* stage the patient has become exhausted. There are no more paroxysms and he is quiet. His heart gradually fails, and he dies by syncope, although he may die in a convulsion or in asphyxia. This stage usually lasts from six to eighteen hours. Happily the disease is one of short duration, ranging from two to six days, notwithstanding its long period of incubation.

Diagnosis.—Hydrophobia most resembles tetanus. Yet the diseases are very different. Hydrophobia has a long period of incubation, while tetanus has a short one, from three to ten days. Tetanus begins with trismus and is associated with opisthotonos. Neither of these symptoms are present in hydrophobia. Tetanus has no laryngeal symptoms, no spasms in swallowing. The mental depression so characteristic of hydrophobia is wanting in tetanus.

More difficult is it to distinguish hydrophobia from the imaginary condition known as pseudophobia or lyssophobia, numerous cases of which have been reported, and the occurrence of which doubtless furnished the foundation for the belief by some that there is no such disease as hydrophobia, and that all cases are lyssophobia. The resemblance is often very close, especially the depression and mania, and it is even said that strong men have been so overcome by this fear as to die in consequence. The condition, however, generally passes away. Especially is this the case when it transpires that the biting dog was not rabid. Hence the usual practice of immediately killing the dog supposed to be rabid is not altogether a wise one, since it makes it impossible to settle the question conclusively as to its madness. It is better, if possible, to confine the animal until the possibility of recovery is settled. The certain knowledge that the biting dog is not rabid is generally a cure to the lyssophobia.

Prognosis.—The prognosis of hydrophobia, once established, is, unfortunately, totally unfavorable. The possibility of spontaneous recov-

ery cannot be denied, but it is certainly exceptional. The preventive treatment is more successful. The claims of Pasteur will be considered under treatment. Youatt's success by cauterization with nitrate of silver is also there referred to. Bollinger's statistics go to show that out of 134 cases in which the bite was cauterized, 92, or 69 per cent., were attacked, while 42, or 31 per cent., died of the disease; of 66 not cauterized 83 per cent. died of the disease.

Treatment.—The curative treatment consists first in prompt measures to eliminate the poison. Suction is the promptest measure available and should be practised, if possible, by the victim himself, as it is not without danger to a second person. An abrasion in the mouth or a carious tooth may be the medium of inoculating such person with the dreaded virus. If suction is practised the mouth should be promptly rinsed. It is doubtful if the cupping glass is as efficient even if at hand.

Next in availability is cauterization, which should be practised by a glowing hot poker or other instrument of the kind, or a galvano-cautery or Paquelin's cautery. In the absence of such means silver nitrate or caustic soda should be used and thoroughly applied. Youatt thought nitrate of silver amply sufficient, having failed once only out of 400 times, and in this instance he declared that the patient died of fright. He himself was bitten seven times and in each instance used this agent. In the absence of these caustics pure carbolic acid may be used or corrosive sublimate one to 500 or 1000. When the symptoms once set in palliation alone is possible.

The paroxysms should be controlled by inhalations of chloroform and averted as far as possible by full doses of opium, preferably, as a rule, morphine hypodermically. Chloral may at first suffice. As light and noise excite paroxysms the patient should be kept quiet and secluded and even in a dark room.

Pasteur's Treatment by the Attenuated Virus.—This is of the nature of preventive treatment. Pasteur discovered that the virus of hydrophobia is located in the nervous system, especially in the brain, medulla, and spinal cord. He then ascertained that inoculations by virus from this source in rabbits produced a virus of such increased virulence that after 25 successive inoculations there resulted a virus which acted after a period of incubation of eight days, and after 25 successive inoculations more in seven days. The virus from the medulla of rabbits, with this short period of incubation, is called "fixed" virus as contrasted with the "street" virus. Now, although the spinal cords of such animals contain the virus in a state of great intensity, Pasteur ascertained that its intensity was greatly reduced by preserving the cords in dry air, and that it disappeared altogether in two weeks. The desiccation is practised in sterilized glass vessels in which are placed pieces of caustic potash. If, now, dogs are inoculated with an emulsion of such medullas of reduced virulence, say cords preserved from twelve to fifteen days, and then with the cord preserved for a shorter period, that is, with progressively stronger virus, they acquire immunity from inoculation by the fresh cord of the rabid rabbit.

Pasteur availed himself of these facts to inoculate human subjects who had been bitten. He used an emulsion of rabbits' cord which had been

kept fourteen days, and on successive days made 12 more inoculations from cords preserved a shorter time, until those only one day old were used, after which immunity was secured. Later, that is, in 1886, Pasteur reported to the Academy of Sciences the so-called "intensive method," consisting in inoculations from cords of increasing virulence in more rapid succession, which is the method now commonly adopted in the various institutes. A careful examination of the results of this treatment by the most exact and conscientious observers, such as Victor Horsley, of London, or William H. Welch, of Johns Hopkins University, as well as the records of the numerous Pasteur institutes throughout the world, goes to show that the treatment is a powerful agent in saving life. In illustration it may be said that the treatment was commenced at the Pasteur Institute in Paris in 1886, at the end of which year the percentage of deaths was 0.94 per cent., while by the end of 1891 it had been reduced to 0.25 per cent., and for 1895 it was 0.13 per cent., or a mortality of two out of 1520 inoculations. In consequence of the difficulties in the way of carrying out the treatment it is practically available only at the Pasteur institutes referred to, of which there is one in New York City, in this country, and in almost all the large cities in Europe.

Tizzoni and Centanni, in 1892, reported success in the treatment of infected rabbits by blood-serum of animals of the same species rendered immune to the disease. The animals were rendered immune by the "Italian method," that is, by inoculating rabbits with attenuated virus obtained by subjecting virulent material to the action of an artificial gastric juice. After digestion for less than twelve hours the virus still kills rabbits when inoculated beneath the dura mater, but the period of incubation is prolonged. After from twelve to twenty hours' digestion it no longer kills rabbits, but causes an infection from which they recover and after which they are immune. From these animals the serum was injected in doses of three to five c. c. into the peritoneal cavity, or circulation of animals inoculated with "street" virus, with success. Later a *dry antitoxin* was used with equal success. Tizzoni and Schwartz, who extended these studies, are engaged in preparation for its application to human beings.

TETANUS.

Synonym.—*Lockjaw.*

Definition.—Tetanus is an infectious disease characterized by paroxysms of tonic spasm, repeating themselves with increasing severity.

Etiology.—The specific cause of tetanus is a bacillus, which was isolated by Nicolaier in 1884 and obtained in pure culture by Kitasato in 1889. It is a slender rod with rounded ends, develops at ordinary temperatures, and is found in the soil, in pus, and putrefying fluids of wounds, sometimes forming threads, sometimes irregular masses. It is non-mobile, anærobic, refusing utterly to grow in the presence of oxygen; develops spores within itself, though when studied early in pus is often sporeless. It is one of the most indestructible of bacilli, its spores resisting a temperature of 176° F. (80° C.), while the bacilli retain their vitality in the dried condition for months. According to G. M. Sternberg

they resist a five per cent. carbolic solution for ten hours, but will not grow after fifteen hours' immersion. If five per cent. hydrochloric acid is added they are destroyed in two hours. They are destroyed in three hours by a one to 1000 bichloride solution, but when five per cent. hydrochloric acid is added the *spores* are destroyed in thirty minutes. Exposure to passing steam for from five to eight minutes kills the spores. The toxin, on the other hand, is rapidly destroyed by heat and light, being unable to bear a temperature above 140° to 149° F. (60° to 65° C.). In the dark in a refrigerator it can be kept indefinitely. Cultures of the tetanus bacillus in all media give off a peculiar characteristic odor,—a burnt-onion smell with a suggestion of putrefaction.

The bacilli do not, however, pass into the blood, but manufacture with great rapidity a ptomaine or toxine, which is absorbed and excites the disease.* This was first shown in 1890 by Kitasato, who observed that cultures of the tetanus bacillus which had been sterilized by filtration through porcelain produce the same symptoms including ultimate death as inoculation with cultures containing the bacillus. Indeed, Brieger in 1886 isolated from impure cultures three ptomaines, which he called *tetanin*, *tetanotoxin*, and *spasmatoxin*. The first of these causes the characteristic symptoms of tetanus, the second tremors, convulsions, and subsequently paralysis, and the third intense tonic and clonic spasms. More recently Kitasato and Weyl obtained Brieger's tetanin and tetanotoxin from pure cultures; while Brieger himself, with Frænkel and Kitasato, has succeeded in isolating from tetanus cultures a far more deadly ptomaine or *toxalbumin*, which was purified by Brieger and Cohn, who have also shown that it is not a pure albuminous body. Brieger has also isolated such poisons from the organs of those dead of tetanus, and Nissen has demonstrated toxine in the blood of those ill of tetanus.

Further, it has been shown by Behring and Kitasato that there exists in the blood of animals immune to tetanus a substance with opposite properties, therefore called antitoxine, and by the gradual introduction of the toxin into animals these observers have been able to produce in their blood a potent antitoxic substance. The method for its production is similar to that for diphtheric antitoxin, but slower. Tizzoni and Cattani have successfully prepared it in a solid form, in which, it is claimed, it can be kept indefinitely and shipped as wanted, and applied it to treatment of cases of traumatic tetanus with success. Six cases have been thus treated up to December 24, 1892. The Lancet for August 10, 1895, contains a review of 35 cases treated at all stages with antitoxin, 23 successfully.

The excitation of tetanus is favored by certain conditions. Wounds, particularly contused and punctured wounds, especially of the hands and feet, are favorite foci, whence the term *traumatic* for such cases of tetanus, and *idiopathic* for cases not thus caused. A similar focus is the badly-cared-for umbilical cord, whence *tetanus neonatorum*, affecting especially the colored race. It is probably because the contused wound affords a more favorable nidus for the growth of the bacilli rather than

* Hochsinger alone claims to have found the bacilli or their spores in the blood of tetanic cases.

that there is any peculiar laceration of nerves, as formerly thought. It is more common, too, in hot countries, and in places and seasons where there are decided alternations of heat and cold. It affects both sexes and all ages, but it is more frequent in men for obvious reasons, the average percentage of cases, according to F. X. Dercum,* being 22.

Idiopathic tetanus is much more rare than traumatic and it constantly happens that close examination, in cases of apparent idiopathic tetanus, results in the discovery of a previous undiscovered trauma. Exposure to cold, especially damp cold, is one of the recognized causes of idiopathic tetanus. It can only produce a condition favorable to the lodgment and multiplication of the bacillus.

That tetanus should occasionally prevail in epidemics is one of the natural results of its mode of causation.

Morbid Anatomy.—There is no essential morbid anatomy in tetanus. There may be congestion, extravasations, and perivascular exudates due to impediment of the movement of the blood during spasm, granular changes in cells from modified nutrition, all results rather than causes of symptoms.

Symptoms.—A period of incubation of from ten to fifteen days is required for the operation of the specific cause of tetanus. Occasionally only does a *chill* precede the other symptoms. There appears first usually a *stiffness in the neck* and *jaws*, and the patient opens his mouth with difficulty, but not with pain. Then the stiffness extends to the back and abdominal muscles and to the legs, which may be fixed in extension, more usually during a paroxysm. The result is that the abdominal muscles feel like a board and the whole trunk is inflexible. If an attempt is made to flex the thighs on the abdomen the whole body comes up in a single piece ; if the body is turned over, it is like turning over a wooden man. There is, in a word, *orthotonos*. Again, as in a striking case of my own, the symptoms may begin in the abdomen and by the intermittent character simulate cramp.†

These symptoms are present in various degrees, less marked in the mild cases, more so in the severe ones. Thus the jaws may partly yield to forcible extension or they may be rigidly locked. The eyebrows may be raised and the angle of the mouth drawn out, producing the *risus sardonicus*, or tetanic grin. There may be *paralysis* of the facial muscles and difficulty of swallowing in the so-called head tetanus or hydrophobic tetanus described by E. Rose, characterized also by violent *spasm* of the *pharynx* and *œsophagus* associated more particularly with injuries to the 5th nerve.

All of the symptoms are further increased during the *paroxysm* which is excited by various sensory impressions, sometimes exceedingly trifling, as a breath of air or the contact of a dress, a footfall, or the slamming of a door. The muscles of the trunk contract more strongly, and if the patient be on his back the body may be so bowed that only the back of the head and heels touch the bed—opisthotonos ; or the side of the

* Article "Tetanus," Keating's "Cyclopædia of Diseases of Children," Vol. IV, p. 913, 1890.

† Philadelphia Medical Times, Vol. I, 1871, p. 418.

face and leg, producing pleurosthotonos; or the abdominal muscles may bend the body forward—emprosthotonos. Spasmodic closure of the jaws sometimes causes the tongue to be bitten. The paroxysm may then relax, and during its relaxation the patient will be able to walk about. In severe cases the spasm may involve also the muscles surrounding cavities, as the thorax, compressing as in a vise their contents, causing extreme pain. Indeed, *pain* is almost everywhere an accompaniment of these spasmodic contractions, and the perspiration stands out in great drops on the face and covers the body. An attempt to speak is transformed into a fit of crying. The frequency of the spasms varies greatly; they may occur every couple of minutes or almost incessantly, or once in several hours. The temperature is generally, but slightly, if at all, elevated, rising to 101° F. (38.3° C.) and more rarely to 102° F. (38.9° C.). At times, however, it rises higher, to 105° to 106° F. (40.5° to 41.1° C.), and it is said also in fatal cases to reach 108° to 110° F. (42.2° to 43.3° C.). In a case reported by Joseph P. Tunis * it fell as low as 96.6° F. (35.5° C.), reaching a maximum of only 101° F. (38.3° C.). The *pulse* is generally frequent, 130 to 150, *respirations* 30 to 45. There is often *constipation*, which is a more serious symptom in severe cases, because the efforts to relieve it are apt to bring on spasm. Among the rare events have been the rupture of muscles and spasmodic closure of the glottis, producing fatal asphyxia. Generally, death is produced by exhaustion, the mind remaining unclouded throughout.

Diagnosis.—Tetanus is liable to be confounded with strychnine poisoning and hydrophobia. Strychnine poisoning differs from tetanus in the absence of rigidity between the paroxysms and of trismus, and the more marked involvement of the extremities, as well as the history of the case. In hydrophobia there is no trismus, and while convulsive dysphagia occurs sometimes in tetanus, it is very rare. As in strychnine poisoning, too, the individual paroxysms are more distinct.

Cerebro-spinal meningitis produces a rigidity similar to that of tetanus, but the cerebral symptoms give it its stamp, and fever is a much earlier symptom than in tetanus. The stiffness of the jaws in parotitis and severe tonsillitis is similar to that of tetanus, but there the resemblance ends.

The interesting and rare condition known as *tetany*, or intermittent tetanus, characterized by the paroxysmal tonic contraction in groups of muscles, more frequently in the extremities, is hardly likely to be confounded with tetanus.

Prognosis.—The prognosis of traumatic tetanus is exceedingly unfavorable, not less than 80 per cent. perishing, while in the idiopathic form less than one-half die.

In children the prognosis is more favorable than in adults, but some very severe cases get well. The case of Joseph P. Tunis, already referred to, is a truly remarkable one of recovery in a boy six years of age after seventy days' duration. Most cases die within the first six days, and cases living to the sixth day are very much more apt to get well, and the aphorism of Hippocrates, that "such persons as are seized

* Archives of Gynæcology and Pædiatry, April, 1892.

with tetanus die within four days, or if they pass these they recover," is frequently substantiated. Localization of the spasm to the muscles of the face, neck, and jaw is characterized by a tendency to recover, and the so-called Rose's head tetanus most commonly gets well. The cases in which there is very little elevation of temperature are apt to do well. Convalescence is likely to be protracted even in mild cases.

Treatment.—There are many difficulties in the way of the antitoxin treatment of tetanus in human beings, and it has perhaps been a disappointment up to the present time. The amount needed in proportion to the body weight increases enormously with the stage of the disease, in consequence of its extremely rapid production by the bacilli —millions of times, where diphtheria antitoxin increases but ten fold.

In the absence of antitoxin the treatment of tetanus must be the treatment of the symptoms. Morphine is indispensable to control the pain and defer the paroxysms or diminish their severity, although anæsthesia by ether or chloroform may be required in addition during the paroxysm. The milder sedatives, as chloral, may suffice in mild cases, but it is insufficient in severe ones. Chloral may be used as an adjuvant in not less than 15 grain (one gm.) doses for adults where the quantity of morphia otherwise required is excessive. Its efficiency is increased when combined with double the dose of bromide of potassium. To a less degree phenacetin, antipyrin, and antifebrin may be useful. Salicylic acid in large doses has been thought to be useful.

Reasoning from its physiological action on the nerve centres, calabar bean ought to be a useful remedy, and it is commonly used in doses of ¼ to ½ grain (0.0165 to 0.033 gm.) three to five times a day. Curare should also be useful for its sedative effect on the terminal nerves, but experience has not confirmed expectation as yet. One-twenty-fifth grain (0.0026 gm.) may be given hypodermically and cautiously increased. The strength of curare varies greatly. Warm baths are serviceable in relaxing spasm and often very comforting to the patient.

The most nourishing food in liquid form is necessary, and usually, also, stimulants are freely administered in tetanus, with a view to sustaining the patient against the exhaustion which sooner or later causes death unless the disease is arrested.

ANTHRAX.

SYNONYMS.—*Malignant Pustule; Contagious Carbuncle; Splenic Fever.*

Definition.—An acute infectious disease of animals, especially affecting cattle and sheep, but transmissible also to man; caused by the implantation and multiplication of the bacillus of anthrax.

Historical and Etiology.—Anthrax was known to the ancients as a destructive disease of animals. The true poison was not, however, discovered until Pollander found it in 1855. Two years later Brauell also found it independently. Davaine, in 1863, greatly extended our knowledge of the whole subject by discovering the bacillus and inoculating many animals, including mice, rats, guinea pigs, cows, sheep, goats, and birds, and more recently Louis Pasteur and Robert Koch,

1878–81, have studied the bacillus exhaustively from the biological standpoint. It was the first microörganism recognized as the cause of an infectious disease. Mention should be made of Toussaint, who was the first to produce immunity by the use of sterilized cultures. His paper on "Immunity from Anthrax acquired as a Result of Protective Inoculations" was published in the Proceedings of the French Academy of Sciences, July 12, 1880.

The bacillus of anthrax is a minute cylinder five to 20 micromillimeters in length and one to 1.25 micromillimeters in breadth. It is found in enormous numbers in the blood and tissues of the animal infected with anthrax, where it multiplies rapidly by division. In artificial cultures it grows in long threads, in the interior of which appear minute ovoid spores, which are loosed by disintegration of the bacilli, which have but a transient existence, while the spores are very tenacious of life. Their vitality may remain in abeyance for long periods of time and with the return of favorable conditions of heat and moisture revive. Introduced into the blood of animals they develop into bacilli. The medium of their transfer is the blood, secretions, flesh, hair, and the like, from those infected to others, including human beings. Here, as in glanders and hydrophobia, an abraded surface is necessary for successful inoculation, although the possibility of absorption through intact mucous membrane and skin is asserted. Those most frequently infected are herdsmen, stable-hands, butchers, wool-sorters, and the like.

It is thought that anthrax bacilli may exist elsewhere than in animals, as in marshes and on the banks of streams, whence they may be carried by freshets into pastures and so infect grazing animals with the disease. Commonly, however, the affection spreads from other animals having the disease. Pasteur has found the bacilli in the herbage over the buried bodies of animals dead from the disease. It is primarily a disease of herbivora, from which it is transmitted to carnivora and man.

Hoffa has isolated a toxin which he calls *anthracin.*

Morbid Anatomy.—The body after death is cyanotic. The blood is dark and viscid, coagulating slowly; the spleen is enlarged and soft. On the skin are carbuncular and gangrenous changes, the subcutaneous tissue is infiltrated with bloody serum, the blood is thin and uncoagulated, all the tissues and organs are more or less infiltrated with blood. The gastro-intestinal mucous membrane is œdematous and ecchymotic, there are enlarged follicles and gangrenous patches infiltrated with bacilli, constituting the so-called carbuncle of mucous membrane. Even the nervous tissues are the seat of analogous lesions.

Symptoms.—Anthrax has a period of incubation of about seven days, after which there are a number of modes of manifestation of the disease, of which the chief are *internal anthrax*, or malignant pustule, and *external anthrax.*

External Anthrax manifests itself as *malignant pustule* or *malignant anthrax œdema.*

Malignant pustule occurs most frequently on exposed surfaces of the skin,—the arms, hands, or face,—at the seat of inoculation. It begins as an *itching* and a *burning, smarting pain*, resembling often that from the bite of an insect. The spot becomes red and develops rapidly into

a papule, in the centre of which a vesicle soon appears, which is filled with clear, or at times bloody, serum. The vesicle bursts, the papule enlarges and becomes indurated, surrounded by a number of small vesicles. The induration extends, while the centre becomes dark and discolored. Within thirty-six hours a brown eschar makes its appearance and rapidly undergoes disintegration. The vicinity becomes œdematous, the lymphatics inflamed, swollen, and painful.

To these local symptoms are added those of general infection, with its *thirst, high temperature*, and *frequent pulse*. The tongue becomes dry, the liver and spleen enlarged, the breathing rapid, and death supervenes in from three to five days.

Occasionally recovery takes place, but it is only in mild cases, in which all the symptoms, local and general, are less severe, that the vesicles dry up into a crust or scab, and the induration dies away.

Malignant *anthrax œdema* begins in the eyelids and passes thence to the head, hand, and arms. The skin reddens and becomes œdematous, vesicles may arise, but there are no papules, although the œdema may proceed to extensive gangrene. The local symptoms in this form follow rather than precede the constitutional disturbances, as in the papular form, and the termination is even more invariably fatal than the latter.

Anthrax presents an interesting contrast to hydrophobia in the absence of the anxious mental condition so characteristic of the latter.

Internal Anthrax manifests itself as *mycosis intestinalis*, or intestinal anthrax, and *wool-sorter's* and *rag-picker's* disease. Intestinal anthrax, or mycosis intestinalis, is often ushered in by chill followed by nausea, vomiting, bloody diarrhœa, and abdominal pains and tenderness. In addition to these symptoms pustules may form on the skin. It arises from the ingestion of meat infected with anthrax.

Wool-sorter's disease is a form of internal anthrax acquired by inhaling the bacilli into the lungs by those engaged in sorting wool, especially that imported from Russia and South America. It begins with chill, fever with high temperature, pain, dyspnœa, bronchitis, and cough. There are rarely premonitory symptoms and often no external lesion. It is rapidly fatal, the patient dying often in twenty-four hours in collapse. Other cases are more protracted, and there may be vomiting, diarrhœa, delirium, and unconsciousness, while the brain may be the chief seat of involvement, the capillaries being filled with bacilli. *Rag-picker's* disease is a special etiological variety.

Diagnosis.—The diagnosis of external anthrax is usually easy from the symptoms, in connection with the history of exposure to the cause. The fluid of the pustule may be examined for the bacilli, which are large and easily recognized. Cultures may be made and a mouse or guinea pig inoculated.

Internal anthrax is more difficult of recognition and may escape it altogether unless a knowledge of the occupation of the patient suggests it.

Prognosis.—The prognosis is unfavorable. Yet not all cases perish. The intestinal form and "wool-sorter's disease" are especially fatal.

Treatment.—Prophylaxis is exceedingly important. Animals dead of the disease should be deeply buried or, better, cremated; their hides

should not be used; infected pastures should be shut off; disinfectants should be freely used in the wake of the disease.

The curative treatment consists in a vigorous attack on the seat of the infection. Deep crucial incisions should be made, and to these the actual cautery or caustic potash or strong carbolic acid should be applied and the wound dressed with a strong solution of carbolic acid, one to 20, or powdered bichloride of mercury diluted with calomel powder four to 15 per cent. may be thrown into the bottom of the incisions. As the sublimate dissolves it deepens the cauterization. The treatment is very severe and etherization may be necessary. Cocaine at least should be freely used. In the œdematous form numerous free incisions should be used and treated as the cuts into the carbuncle.

With the local treatment should be associated stimulating and restorative measures, including alcohol, highly nutritious food, quinine, and strychnine. Five to ten grains of ipecacuanha powder every three or four hours are recommended by Davies-Colley.

Internal anthrax must be treated by the general measures just alluded to, but is generally hopeless. Free purgation is advised at the outset, with a view to removing the infecting material.

GLANDERS AND FARCY.

SYNONYMS.—*Farcy; Malleus humidus.*

Definition.—Glanders is an infectious disease more especially of the horse, communicable to man and certain domesticated animals, but not to cattle; characterized by nodular growths in the nares, when it is known as glanders, and under the skin, when it is called farcy. Among animals to which it is communicable are the lion, sheep, rabbit, guinea pig, cat, dog, and mouse.

Historical.—Glanders was apparently described by Aristotle (B. C. 384 to 322) as occurring in the ass. It is first mentioned under the name *μάλις* and *malleus* by Apsyrtus, a veterinary surgeon under Constantine the Great (A. D. 272 to 337), who, however, included under this name a number of affections of a more or less similar nature, and we are indebted for our first accurate knowledge of the disease to Rayer, whose monograph appeared in 1837.

The contagious nature of glanders was recognized by Soleysal (1664), Garsault (1741), and by the two writers Lafosse (1754–72); its inoculability and fatal character were demonstrated by Abeldgaard in 1795, while its contagiousness was experimentally established by Viborg (1797). Notwithstanding these facts, its contagiousness was long contested in France, and it was not until the middle of the present century that finally it became generally acknowledged in that country, chiefly through the labors of St. Cyr. Lorin, in 1812, Waldinger, in 1816, and Veith, in 1822, made researches on the injurious effects of the virus on man. Schelling, of Berlin, was the first to give an exhaustive description, in 1821, of the disease as it occurs in man.

Etiology.—Glanders is the direct result of a bacillus—the *bacillus mallei*—described by Löffler and Schütz in 1882. It is a short, non-

motile bacillus not unlike that of tubercle and leprosy, but shorter than either. It is commonly seen among the cells of the growth, but has also been found in the blood. The disease is communicated through the discharge from the infected animal to an abraded skin surface or intact mucous membrane. The human victims are usually hostlers or others working about horses.

Morbid Anatomy.—Glanders presents itself in the shape of nodules ranging in size from that of a lentil to a fist, or it may infiltrate more diffusely. It is composed of round cells which invade the skin and muscles. A few of these develop into epithelioid cells, but all soon break down, leaving ulcers on the mucous membranes and skin, and abscesses under the latter. Internal organs, as the lungs, liver, spleen, kidneys, and even stomach, and nervous system, bone, and cartilage may be invaded. The ulcers on the skin are often serpiginous, whence the name *Wurm* among Germans.

Symptoms.—Glanders has a period of incubation of from three to five days, rarely a week. There is an *acute* and *chronic form*. The acute terminates within three weeks, while the chronic may last months and even years.

In Acute Farcy after the period of incubation a *feverish state* develops. At the point of infection on the skin there appears a *nodular swelling*, or an ulcer which tends to spread and discharge a fetid hemorrhagic pus. The adjacent tissue becomes red and œdematous and the lymph-vessels and lymphatic glands are inflamed. Papules that become pustules may also develop in the neighborhood. Such an eruption has been mistaken for that of small-pox, but is soon replaced by open ulcers. The so-called *farcy buds* are nodular, subcutaneous enlargements along the course of the lymphatics and may suppurate. The nose is not involved.

In Acute Glanders of the nasal mucous membrane there is first *redness* and *swelling* at the point of inoculation with *burning* and *dryness* of the nasal mucous membrane. *Intense pain* in the forehead from involvement of the frontal sinuses may also be present. This is promptly followed by nodule formation and the rapid breaking down of the nodules and discharge of fetid hemorrhagic or muco-pus. The destructive process extends to the nasal septum, the mouth and pharynx, and even the larynx, lung, and other organs. The submaxillary glands swell and suppurate. From these lesions result the usual symptoms of *painful deglutition*, *cough*, and *hoarseness*, with *fetid expectoration*.

To these symptoms in both forms are added *intense prostration* and depression, muscular and joint *pain* and soreness, *abscess* formation, *high temperature* with chilliness, and finally *typhoid symptoms* and death.

The *spleen* and *liver* may be enlarged, *albuminuria* present, and it is said even *leucin* and *tyrosin* are found in the urine.

In Chronic Farcy the localized tumors form under the skin, especially of the extremities, and break down, but the process is more slow and there is no special involvement of the lymphatic glands.

Chronic Glanders is less easy of recognition. The symptoms are more like those of incurable coryza and sometimes of chronic laryngitis. It may be necessary to make cultures and inoculate an animal, preferably the guinea pig, which perishes in thirty days and presents already testicles swollen and suppurating.

Diagnosis.—The diagnosis in the acute form is usually easy. It has, however, been confounded with pyæmia and small-pox. Chronic glanders is to be distinguished from syphilis and tuberculosis. In doubtful cases cultures should be made. Especially characteristic is that on the cooked potato, which by the third day furnishes an amber-hued film, which on the sixth to eighth day is red and turbid, surrounded with a pale green area.

Prognosis.—The prognosis in the acute variety is invariably fatal; in the chronic form 50 per cent. recover.

Treatment.—In the cutaneous form excision and cauterization should be practised as early as possible, followed by antiseptic dressings.

In the nasal variety sprays of carbolic acid and bichloride of mercury and peroxide of hydrogen should be introduced into the nose and throat.

FOOT AND MOUTH DISEASE.

SYNONYM.—*Aphthæ epizooticæ.*

Definition.—An acute infectious disease of the lower animals, especially of cattle, sheep, swine, more rarely the goat and horse, and still more rarely of fowls, dogs, and cats. It is communicable to man and characterized by the appearance of vesicles in the mucous membrane of the mouth and between the toes, and on the teats of cows. The disease in cattle spreads rapidly and entails often serious loss.

Historical and Etiology.—The early confusion of foot and mouth disease with anthrax and actinomycosis makes it difficult to date its first recognition. Hertwig, however, established its contagiousness as early as 1834 by experiments upon himself and two other men. The experiments consisted in the drinking of infected milk. Local and constitutional symptoms of the disease resulted.

The contagium bearer is especially the contents of the vesicle alluded to, but milk, blood, and urine and feces are also media. It is communicated to man through the ingestion of unboiled milk, butter, and cheese, or through contact with the fluid of the vesicles on the teats by milkers. It is said to be communicable even by the saliva from the affected animal.

A streptococcus has been isolated from the fluid of the vesicle by Klein, and a micrococcus from milk by Cnyrim and Libberitz; the specific power of neither has as yet been determined.

A certain relation is believed to exist between the aphthous sore mouth of children and the foot and mouth disease, chiefly because it has been observed that aphthæ is apt to prevail in children at the same time with the foot and mouth disease in cattle.

Morbid Anatomy.—As recovery invariably takes place, no lesions other than those to be noted under symptoms have as yet been observed.

Symptoms.—The disease has a period of incubation of from three to five days. At this time there is a *febrile movement* with *malaise* and *loss of appetite.* On the mucous membrane of the lips and tongue, and sometimes on the hard palate and pharynx, come *vesicles* containing a yellowish serum. There is a sensation of *heat* and *burning* throughout the mouth, and the *swelling* may be so great as to make speech difficult and

swallowing painful. There is *copious salivation.* Almost simultaneously appear vesicles between the fingers and toes and around the nails. Vesicles have also been noted on the nipples of women. Indeed, they have been found scattered all over the body, so that the case resembles smallpox. The hands, especially, may be extensively involved. Gastro-intestinal symptoms are sometimes present.

Prognosis.—The prognosis is favorable in man, recovery being the rule. Very young children may perish. The suckling young of animals perish in large numbers, because of the infected milk on which they subsist.

Treatment.—The disease can be easily avoided by simple prophylactic measures by those in contact with animals, of which the use of boiled milk is the most important. Cleanliness of man and beast conduce to the same end.

Curative measures of a simple kind generally suffice. Mouth washes of a saturated solution of chlorate of potassium should be frequently used. Powdered borax and alum may be directly applied. The separate ulcers or vesicles should be touched with the solid silver nitrate. The skin lesions should be washed in corrosive sublimate solution and dressed in sublimate cotton or salicylated cotton. The fever should be combated with suitable antifebrile measures.

THE BUBONIC PLAGUE.

SYNONYMS.—*Oriental Plague; Black Death.*

Definition.—The plague is a febrile infectious disease, characterized by a tendency to buboes or carbuncles, in addition to the usual phenomena of the typhoid state.

Historical.—It has already been said that the historical plague described by Thucydides corresponded rather with the typhus of to-day than the Oriental plague, which still occurs rarely in Asia, and of which the last epidemic occurred in Hong-Kong, China, in May, 1894. The disease which is known to-day as "the plague" has been recognized as a separate disease since the middle of the fourteenth century, when it was called "black death." With the improvement of hygienic conditions it is becoming rare with the progress of time. In addition to the recent epidemic in China the region of the lower Volga was visited as recently as 1878 and 1879.

Etiology.—The recent epidemic gave the opportunity of isolating its specific germ, which was done by Kitasato. It is a short rod with rounded ends, and resembles the bacillus of chicken cholera. It is found in the blood, glands, and other viscera, and in no other disease excepting the plague. Obtained in pure cultures it can produce in inoculated animals the same effects as in human beings. It obtains entrance through the respiratory and digestive tracts and excoriations.

Morbid Anatomy.—There is no morbid anatomy to the plague beyond the buboes, which seem to be an essential symptom, and the various tissue alterations which attend high fevers generally.

Symptoms.—A period of incubation of two to seven days usually

precedes the appearance of the *intense weakness*, which is one of the characteristic symptoms of the plague. The usual *fever* of the infectious diseases then sets in with great severity and with its usual accompaniments, among which the typhoid state is conspicuous. *Petechiæ*, *vibices*, *hæmaturia*, and even *hæmatemesis* may be included.

Preëminently characteristic is the *bubo* or suppurating gland. It appears on the second or third day, if the patient live to it. It is to be found in the groin, affecting the vertical rather than the horizontal chain of glands, in the arm pit, or in the neck. It commonly reaches the size of a walnut or egg, when it ruptures if not opened with the lance. It may, however, subside without discharging. It is painful and tender, as buboes are commonly. Coincident with the appearance of the bubo the fever subsides, a profuse sweat breaks out, and the pulse falls to 90 or 100. In addition to the bubo, carbuncles may also be present in the lower extremities, the buttocks, or in the neck.

Diagnosis.—In its fever, its intense prostration, its petechiæ and vibices of the early stages the plague resembles typhus. The bubo and the carbuncle seem to be the distinctive signs, although they are said to be sometimes absent in the milder cases of a declining epidemic.

Prognosis.—The plague is said to be the most fatal of all diseases, 70 to 90 per cent. perishing, districts and towns being half depopulated, while whole families have been annihilated. It is likely, however, with the improved sanitation of modern times, an epidemic to-day would be attended by less serious results. Death occurs very early on the second or third day, and if recovery takes place it is delayed by the slowly healing buboes and carbuncles.

Treatment.—Free stimulation, nutritious food, as in the most adynamic forms of typhus and typhoid fever, together with cool baths to combat the fever, are the measures indicated. Antiseptic treatment of the abscesses should be practised, and may shorten the duration of these plagues of the skin as compared with the older treatment. The following are Kitasato's general directions :—

"The disease prevails under faulty hygienic conditions ; it is, therefore, urged that general hygienic conditions be carried out. Proper receptacles for sewerage should be provided, a pure water supply afforded, and streams cleansed ; all persons sick of the disease isolated ; the furniture of the sick-room washed with a two per cent. carbolic solution in milk of lime ; old clothes and bedding are to be steamed at 100° C. (212° F.) for at least one hour, or exposed for a few hours to sunlight. If feasible, all infected articles should be burned. The evacuations of the sick are to be mixed with milk of lime, and those who die of the disease are to be buried at a depth of three meters (*circa* 12 feet) or, preferably, cremated. After recovery the patient is to be kept in isolation at least one month. All contact with the sick is to be avoided, and great care exercised with reference to food and drink."

SYPHILIS.

SYNONYMS.—*Lues venerea; The Pox.*

Definition.—Syphilis is a specific constitutional disease due to inoculation by a special virus or to hereditary transmission. Under the former condition it is known as acquired syphilis, under the latter as hereditary syphilis. It is further characterized by a tendency to a localized deposit of a great variety of inflammatory new formations. It is apparently confined to the human race.

Historical.—Syphilis was first described as a separate form of venereal disease in 1494, when it prevailed as an epidemic among the troops of Charles VIII before Naples. From here it spread over Italy into France, Germany, and the rest of Europe. As this was immediately after the discovery of America it has been alleged that the disease was introduced into Europe from America, but it has also been claimed that it was introduced from Africa. In fact, we have very little definite knowledge on the subject, but there seems good reason to think that the disease is much older than the dates given.

Etiology.—In common with all infectious diseases, syphilis is ascribed to the operation of a bacillus, and two or three have been described as ætiological factors and then abandoned. The latest is that described by Lustgarten in 1884. It resembles the tubercle bacillus and leprosy bacillus, and is from three to four μ in length, slightly clubbed at the ends. Matterstock, Travel, and Alvarez claim that it is not differentiable from a similar bacillus found in præputial and vulvar smegma. Cultures of the bacillus of syphilis have not as yet been successful, and it must be admitted that there is still much doubt on the subject.

Syphilis is one of the most highly contagious diseases. In the first place, the blood of the syphilitic is inoculable and capable of producing the disease. Further, the secretions of all primary and secondary lesions of the skin and mucous membranes are similarly potent. The products of the third or gummatous stage are not so regarded, although opinions are not unanimous on this point. A raw or abraded surface is a necessary condition of inoculation. The physiological secretions, such as the tears, milk, nasal and bronchial mucus, do not communicate the disease when inoculated, although they may become virulent by contamination with the poisonous secretions. Exceptions to this law are the spermatozoid of man and the ovule of woman, each of which if derived from a syphilitic source is capable of infecting the other.

The disease has three stages, a *primary*, *secondary*, and *tertiary*. The *primary* is characterized by a primary sore associated with glandular enlargement in the neighborhood of the seat of inoculation. The *secondary* stage furnishes lesions of the skin and mucous membranes, among which sore throat is especially conspicuous. It is sometimes accompanied by fever. The *tertiary* is characterized by affections of deep-seated structures, the osseous and nervous systems and the viscera, including the liver, spleen, kidney, testicles; also the subcutaneous and submucous tissues.

The initial sore makes its appearance within six weeks after exposure, usually in two to three weeks. The phenomena of the second stage

usually show themselves within three months or from six to twelve weeks. The third stage is more difficult to define by temporal limits. It is by years rather than by months and is characterized, as stated, by the involvement of the deeper-seated organs. Hereditary syphilis, when not present at birth, makes its appearance within the first three months; after six months the child may be regarded as safe.

In the vast majority of cases, acquired syphilis comes from sexual intercourse, but it may be the result of contact in many ways, as by the lips, teeth, infected hands, and other parts of the body. Drinking cups, utensils, and other articles used by the infected in common with others, sometimes convey the infection. Physicians are not infrequently infected in midwifery practice, the initial lesion making its appearance around the nail or in the web between the first and second fingers. Wet-nurses acquire the disease from syphilitic nurslings, the chancre occurring in a fissure or abrasion of the nipple. Vaccination has in rare instances been a means of infection.

Hereditary syphilis may become transmitted through the father or the mother. In the former instance it is called sperm inheritance, in the latter, germ inheritance. Syphilis may be communicated by the father while the subject of the active disease or after all signs of it have disappeared. On the other hand, a syphilitic father may beget healthy children. The question has sometimes to be decided by a physician as to whether a syphilitic, apparently recovered, may marry with safety to offspring. It will be seen from the above that an absolute answer dare not be given, but this much may be said, that the longer the interval since the primary attack the less likely is the offspring to be tainted, and it is generally acknowledged that systematic and continuous treatment may eliminate the disease altogether. An interval of not less than three years should be insisted upon between the disappearance of the last symptom and the patient's marriage. It is to be remembered also that each successive child of syphilitic parents shows less signs of the disease, until finally healthy offspring results.

A syphilitic mother may, of course, bear syphilitic children from germ infection, producing thus true hereditary syphilis; but a child may also be infected at the moment of its birth, when the syphilis is congenital but not inherited. On the other hand, a woman may bear a syphilitic child, and, though herself without signs of the disease, will not, according to Colles's law, be infected by her child should she suckle it while it has syphilitic ulcers of the lips and tongue. Yet a healthy nurse who suckles this same child or merely handles it and dresses it may be infected. Such a woman is supposed to have received protective inoculation without evident signs of the disease; and we may have here an example of protection through a natural antitoxine absorbed from the syphilitic fœtus by its non-syphilitic mother.

A woman may be infected after conception when the child may be born non-syphilitic or syphilitic by placental transmission.

Of course, when both father and mother are infected, the chances of the offspring being infected are doubled.

Morbid Anatomy.—I. *Of Acquired Syphilis.—At least five sets of lesions* may be traced to acquired syphilis. The *first* is the initial lesion,

the chancre or primary sore at the point of inoculation; this constitutes primary syphilis. Beginning as a wounded or abraded spot, a vesicle or papule develops, which subsequently softens in the centre and forms an ulcer with a hard, gristly base and edge, constituting the hard or indurated chancre. It is found to consist in a dense infiltration of small cells, some of which develop into large formative (epithelioid) cells and others even into giant cells, but no further differentiation takes place; for the most part the infiltration breaks down and is absorbed, a few of the cells going to form the cicatrix. In the broken-down tissue is found the Lustgarten bacillus. Along with the chancre there is a *second* lesion, an adenitis of the adjacent lymph glands, which may suppurate, forming a bubo, or there may be a hyperplasia of connective tissue, terminating in persistent induration of the gland. Buboes may be long stationary and are then said to be indolent. They may be multiple.

The *third* lesion is the mucous patch, or *condyloma latum*, which is one of the events of the secondary stage of syphilis. It has its seat on mucous membrane or on soft, moist skin, as in the perinæum, groins, between the toes, at the junction between the skin and mucous membrane at the angle of the mouth and about the anus. It consists of an inflammatory infiltration of the epidermis and corium with small cells. A more hypertrophic infiltration of the papillæ of the mucous membrane is the acuminate condyloma, or venereal wart, especially common about the vulva and anus.

The *fourth* lesion is the cutaneous affection, or syphilide, of which there is a macular, a papular, a pustular, and a squamous variety. All are characterized by a copper-colored hue, especially permanent after the other features have subsided, and a tendency to symmetrical distribution. The macular or roseolar syphilide affects more particularly the abdomen, the chest, and the front of the arms, while the face is exempt. This syphilide persists a week or two. The papular eruption is in groups on the face and trunk. The pustular eruption often closely resembles that of small-pox. The squamous syphilide resembles other squamæ, but it is especially distinguished by its coppery hue. It involves preferably the backs of the arms and the front of the thighs—the extensor surfaces.

The *fifth* or remaining set of lesions constitutes the tertiary manifestation, and involves the deeper tissues, such as the osseous and the nervous systems, the liver, lungs, kidney, and subcutaneous tissues. The most general of these is the *fibroid induration*, consisting in a development of fibroid tissue like that of chronic inflammation. The new tissue thus formed arises around the blood-vessels, and consists, at first, of a small-celled infiltration, which later is converted into fibroid tissue. It occupies, for the most part, small areas surrounded by normal, unaffected structures. It is found in the periosteum, the sheaths of the nerve trunks, the capsules, and interstitial tissue of organs and muscles. When in the capsules of organs it sends prolongations into their interior, which partition off the organ and by their subsequent contraction give rise to irregular thickening and cicatricial puckering.

A differentiation of this fibroid change, the most characteristic lesion of syphilis, is the *gumma*, a yellowish-white fibrous nodule closely con-

tinuous by its outer layer with the connective tissue of the organ in which it is imbedded. It varies in size from a pin-point to a diameter of three to five centimeters ($\frac{1}{8}$ to $\frac{1}{5}$ inch). Histologically it is with tolerable ease separated into three layers, the oldest or central in a state of atrophied cheesy degeneration, an intermediate layer of imperfect fibrous tissue, and an external layer of vascular granulation tissue rich in cells. It is frequently associated with the fibroid change above described. In the degenerative changes to which the gumma is subject it may produce extreme destruction of the organ in which it is imbedded.

The seats of the gummy tumor are the skin, subcutaneous and submucous tissue, muscles, fasciæ, bone, where it forms the syphilitic node, the connective tissue of organs, especially the liver, brain, testicle, and kidney, less commonly the lungs. When in submucous tissues, it may give rise to deep-seated ulceration and suppurative processes, leading to destruction not only of soft tissues but also of bone. Especially frequent and repulsive in its result is the destruction of the nasal bones with perforation of the palate.

The bacillus of Lustgarten is said to be found in the gummy tumor.

Another lesion of syphilis, although probably not peculiar to it, is *syphilitic arteritis*, which consists in a cellular thickening of the vessel-walls, beginning in the intima and intruding thence on the lumen of the vessels. The outer coat is abnormally vascular and infiltrated with small cells, which also invade the muscular coat. These are the phenomena of obliterative endarteritis, which have thus far been studied only in the vessels of the brain by Greenfield and Huebner.

Symptoms.—The symptoms of acquired syphilis are so largely the morbid states described under the head of morbid anatomy that most of them need only be enumerated in connection with the date of their appearance.

The *chancre* or initial sore presents itself from two to three weeks after inoculation, usually on some part of the penis, especially in the prepuce, and in females the labia and vaginal part of the cervix. It may be so small as to escape notice, especially when within the urethra. The sore lasts from three to four weeks to as many months. Its peculiar induration is easily recognized by taking it up and pinching it between the fingers, though it is often not characteristic on mucous membranes, as in women. The glandular enlargement in adjacent glands is simultaneous or as soon as the induration is well established.

The secondary symptoms manifest themselves usually from the sixth to the twelfth week, but may be as late as three months. *Sore throat* is one of the first of these symptoms, and is commonly associated with *fever*, which rarely exceeds 101° F. (38.3° C.). It may be remittent and even strikingly intermittent, and in rare instances rises much higher than 101° F. (38.3° C.), reaching 104° F. (40° C.), and even 105° F. (40.5° C.). The sore throat alluded to is associated with hyperæmia of the fauces, often with intractable, gray-based ulcers, and less frequently with mucous patches and syphilitic warts. The inflammation may extend from the throat into the Eustachian tube and middle ear, producing impaired hearing. The larynx is especially liable to become the seat of ulceration, which may heal and produce marked deformity.

Then there are the syphilides named. Along with these, a very common symptom is the *falling out of the hair*, and especially from the eyebrows, giving rise to a striking change in the facial expression. An inflammatory condition at the root of the nails, *syphilitic onychia*, causes them to become brittle and distorted. Other secondary symptoms not mentioned are *iritis*, and more rarely *choroiditis* and *retinitis*. The former presents itself in from three to six months after the primary chancre, and is one of the most painful and trying of symptoms, requiring prompt and energetic treatment. Involvement of the *ear* ossicles is rare but possible, producing deafness.

In addition to the tertiary symptoms already named, viz., fibroid induration, nodes, and gummy tumors, are *deep-seated syphilides*, which destroy the skin in its deepest layers and produce loathsome sores. These on healing leave ugly scars. Syphilitic rupia is the commonest. It consists of large pustules, which dry, and crust over with laminated scabs, while beneath is a deep ulcer. This may subsequently heal, leaving a scab. Large pustular lesions, tubercular syphiloderms, occur especially in the neighborhood of the sacrum.

Joint affections are sometimes associated with tertiary syphilis. These may, of course, result from the invasion of the joint ends of the bones by the gummatous syphilitic disease, to which they are subject, but there may also be direct involvement of the *serous* tissues themselves by inflammatory and gummatous processes which give rise to pain and interfere with motion. The *bone affections* of syphilis are characterized by nocturnal pains, said to be due to pressure from distended veins.

The involvement of internal *glandular* organs occurs later, ten or more years after the primary lesion, though precocious tertiary lesions of this kind have been reported much earlier. *Amyloid disease* is a very common tertiary affection, involving liver and spleen and producing some of the most striking enlargements of the former. But cirrhosis and cicatricial markings are also common. Syphilitic lesions of the liver are of such a degree and importance as to demand separate consideration under the diseases of that organ.

A sarcocele involving the whole testicle is among the tertiary affections often mistaken for tuberculosis, from which it may be distinguished by the fact that the latter is accompanied by tuberculosis elsewhere, and involves the proper structure of the testicle instead of the whole organ. Sclerosis of the spinal cord is frequently associated with the syphilitic history, and it is often ascribed to it. A special condition is an involvement of the nervous system of such importance as to require a separate section. Gummy tumors of the brain occur, producing pressure symptoms; a similar association is true of *arterio-sclerosis* as well as the *arteritis obliterans* alluded to.

II. *Of Hereditary Syphilis.*—Except the primary chancre all the symptoms described as occurring in acquired syphilis may be present in the congenital form. It may be said, in a word, that visceral alterations are more prominent, especially those involving abdominal organs. It is necessary, therefore, to mention here only those which may be regarded as additional.

Among the most important of these is repeated *abortion*. It is very

common to have four or five and even more abortions, while each successive one usually takes place longer after conception until finally a living child is born. Such aborted products are shriveled, the skin exfoliates, and there is often reason to believe they have been some time dead. Syphilitic children born at term have evidently been arrested in development, are shriveled and wizen-faced, and may suffer from cutaneous syphilides. The so-called *pemphigus neonatorum*, with blebs occurring about the wrists, hands, ankles, and feet, is characteristic. There is also apt to be enlarged liver and spleen. Or a child may be born apparently healthy and take on these symptoms after three or four weeks. Lesions of hereditary syphilis are reported as beginning even later than this, up to the sixth month. This is, however, unusual. Rhinitis, or nasal catarrh with *snuffles*, is one of the earliest, to be followed by cutaneous lesions, particularly about the nates. *Fissures* about the lips and ulcerations on the muco-cutaneous surface are present, from which the discharges are inoculable.

Disease of the epiphyseal *cartilages* of long bones and of the cartilages of the ribs is a very common characteristic of tertiary syphilis. The zone of the cartilage adjacent to the bone exhibits proliferated cartilage cells and prolongations over the end into the diaphysis instead of being sharply separated. There is tendency to hemorrhage. A syphilitic cry, high pitched and harsh, is described. To these may be added any of the symptoms already mentioned under acquired syphilis.

A later symptom is "notched teeth," first described by Jonathan Hutchinson as characteristic and distinctive of hereditary syphilis. The teeth affected are the permanent central incisors of the upper jaw. The appearances are not uniform, and are better appreciated by examining the accompanying drawing than from descriptions. Other late symptoms are keratitis, iritis, impaired hearing from ear affections, periostitis, and splenic and hepatic enlargement.

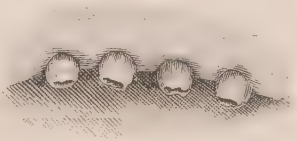


FIG. 14.—SYPHILITIC TEETH.

If it survives the earlier lesions or escapes them, the syphilitic child remains undeveloped and stunted in its growth, and in consequence of arrest of development a singular reversal of the appearance of premature age, described as characteristic of the syphilitic child at birth, takes place. The new-born syphilitic child looks prematurely old. A popular novelist has aptly described the appearance of the syphilitic child in the terse phrase, "A little old man with a cold in its head." The syphilitic who outlives his childhood remains, however, younger looking than he actually is, insomuch that a young man of twenty may appear as though he were but twelve, a condition to which Fournier applies the name *infantilism*. In such the forehead is prominent, the frontal bases are marked, the bridge of the nose depressed, the tip turned up. The head may be asymmetrical.

Diagnosis.—The recognition of general syphilis is not difficult. The symptoms described are of themselves distinctive, and if there be added the history of exposure or heredity they are unmistakable. When there is doubt the administration of specific remedies will soon

clear it up. In consequence of the fever and frequently associated splenic enlargement with roseola, the second stage of syphilis has been confounded with typhoid fever. The pustular syphilide has sometimes caused its subject to be taken to a small-pox hospital, where, however, time soon dissolves all doubt.

Prognosis.—The prognosis of acquired syphilis depends wholly on the treatment. With properly conducted treatment it is favorable, without treatment or with defective treatment the most serious consequences result, while the physical inconvenience and suffering scarcely exceeds the mental misery which the knowledge of the presence of so loathsome a disease entails. In congenital syphilis treatment is less satisfactory for the severer manifestations, and it is perhaps fortunate that so many perish in infancy or early childhood. Even those most fortunate remain delicate and vulnerable to disease through life, and too often fall victims to causes which but slightly affect the healthy man and woman.

Treatment.—*Prophylaxis.*—Against sexual syphilis the only prophylactic measure to be relied upon is sexual purity. The duty of the physician is plain in respect to this, and the medical man who advises illicit sexual intercourse for any reason degrades his calling. Medical men should be exceedingly cautious in their necessary professional contact with all suspected of having syphilis and protect themselves against accidental infection. It is to be remembered that the secretions of all primary and secondary lesions, as well as the blood of syphilitics, may transmit the disease.

Treatment of the Primary Sore.—With the present view that the hard chancre, which makes its appearance after a period of incubation of at least two weeks, is simply the local expression of a general disease, nothing is gained by "burning it out." The indication is simply to heal the ulcer as thoroughly and as soon as possible. Fortunately, there is little difficulty in accomplishing this. A simple dressing, as lint wet with bichloride solution one to 2000, or mercurial ointment smeared on adhesive plaster, with an uncovered edge so as to secure adhesion, will accomplish the healing in a short time. Or iodoform, bismuth, or calomel may be dusted over it.

The constitutional treatment should begin at once. Mercury and iodine are the two remedies and if properly used will eradicate the second stage and hold the third in abeyance, even after it has manifested itself. Mercury is the remedy par excellence of the second stage, iodide of potassium of the third. The best method of administration for mercury is undoubtedly by inunction. The following is the plan to be pursued: A warm bath is taken, if possible, each day, and immediately thereafter a drachm (four gm.) of mercurial ointment or a piece half an inch in diameter is spread between the hands and rubbed one day on the inside of one thigh, the next on the inside of the other; again, under the arm, on the chest, and so on until each part of the body covered by softer skin is treated, after which the same course can be repeated. The friction is to be kept up until the skin is thoroughly dry, half an hour being usually necessary. The part rubbed should be washed off the following day. Parts covered with hair are to be avoided, because mercurial eczema characterized by pustules starting from the hair follicles is

more apt to be produced in these localities. During this time the patient should not smoke and the teeth should be frequently and carefully cleansed and the mouth washed with solution of chlorate of potash with a view of averting mercurial sore mouth. Sooner or later, however, sore mouth may manifest itself by a fetid odor, swollen gums, and a sensation as though the teeth were loose, when the treatment should be suspended for a week or ten days.

The daily friction should be kept up for thirty days, if possible, after which, if no symptoms are present, it may be discontinued. The inunctions should be followed up by the use of protiodide of mercury, ¼ grain (0.016 gm.) three times a day, or the biniodide, $\frac{1}{16}$ grain (0.004 gm.) three times a day. The former is usually preferred because less irritating. This last addition to the treatment should be kept up indefinitely. By such means as these tertiary symptoms can be averted if the patient is but willing to keep them up. The great difficulty is to secure this. He tires of the monotony and the trouble involved in a faithful adherence to the directions, and symptoms sooner or later return. Should secondary symptoms recur a course of inunctions may be repeated.

In lieu of the inunction the *hydrargyrum cum cretâ*, or gray powder, may be used. It is the favorite of Jonathan Hutchinson, who gives it in form of a pill, one grain (0.066 gm.), with one grain (0.066 gm.) of Dover's powder, from four to six times a day, and is commended by my colleague, Louis A. Duhring, who has used it with great success.

Or the mercury may be administered by fumigation. For this the patient sits on a chair, wrapped in blankets to the chin, as in a tent. Under the chair is placed a spirit lamp and over this a tin plate on which calomel is spread. It is volatilized by the heat and deposited with the vapor on the patient's skin. The exposure should last twenty minutes, after which the patient should be put to bed, wrapped in blankets, without washing or drying.

Most recently mercury has been administered by direct injection into the muscles. One-third of a grain (0.0216 gm.) of bichloride dissolved in 20 minims (1.333 gm.) of water is injected once a week, or from one to two grains (0.066 to 0.132 gm.) of calomel in 20 minims (1.333 gm.) of glycerine and water. The injection is made deep into the muscles, and not in the subcutaneous tissue, through silver canulæ. The points selected are the sides of the thorax and back, where abscesses are said to be less likely to occur. Great care should be taken in sterilizing instruments. The nicest attention to these points is, however, still followed at times by abscesses.

In the treatment of the third stage the iodides are especially useful. It is here that the massive doses of iodide of potassium are indicated and often produce such magical results. The most convenient mode of administration is the saturated solution, of which one drop contains a grain (0.066 gm.). Starting with ten drops, a drop may be added each day to the dose until the symptoms yield, that is, until the gummy tumors melt away. Pressure symptoms and head and bone pains are relieved. The iodide is well exhibited in milk. The indication for its discontinuance or reduction in the dose are the erythematous rash, coryza, and salivation and constriction about the salivary glands.

TUBERCULOSIS.

Definition.—Tuberculosis is a general or local infectious inflammatory disease, the result of the implantation and proliferation of the tubercle bacillus. The specific inflammatory product is the miliary tubercle, a nodule of new formation around an irritated point, the focus of which is the tubercle bacillus.

The tubercle bacillus is a short rod-bacterium three to four μ in length, equal to about $\frac{1}{3}$ the diameter of a red blood disc, and $\frac{1}{6}$ to $\frac{1}{5}$ as broad as it is long. When successfully stained and viewed with high power it presents at times a beaded appearance once ascribed to the presence of spores, but now, I believe, regarded as the result of unequal staining. It can be studied satisfactorily only when stained by one of the aniline dyes.*

---

* Of the various methods of staining tubercle bacilli that by the carbol-fuchsin solution of Ziehl-Neelsen, with or without Gabbet's counter-stain of methyl blue, appears to be, on the whole, the most satisfactory. By this method the bacillus takes a bright red color from the fuchsin, the mordant being carbolic acid.

The carbol-fuchsin solution is made as follows:—

Powdered fuchsin,	1 part
Alcohol,	10 parts
Five per cent. solution carbolic acid,	100 parts.

Mix and filter.

The older the solution the better.

A rapid and a slow method are practised with this staining fluid, the former being more commonly used for diagnostic purposes.

(1) *The Rapid Method with Carbol-fuchsin, with or without Counter-stain by Methylene Blue.*—A very small caseated clump of the sputum (care being taken that a bit of food is not taken by mistake) is selected with forceps or a platinum loop and laid on a clean cover glass. Another cover glass is superimposed and the two are rubbed together until the specimen is thoroughly smeared over both. They are then separated, two specimens being thus obtained. When dry, one of them is passed, sputum side up, three times over the flame of a spirit lamp or Bunsen-burner, by which the albumin is coagulated and the specimen is fixed. The cover glass is then completely covered with the staining fluid and held over the flame until the solution begins to vaporize, care being taken to keep all parts of the glass thoroughly covered. At the end of one minute it is washed in water. It is then decolorized in acidulated alcohol, eight to ten gtt. of HCl or five gtt. of HNO_3 to a watch crystal of alcohol, and examined. For this a $\frac{1}{12}$ oil immersion lens and Abbé's condenser are best suited, but after a little experience an ordinary dry system of 350 diameters' amplification, or higher, will easily reveal the bacilli, which are stained a handsome red.

The preparation is more brilliant and its study rather less trying to the eyes if counter-stained by a Gabbet's acid blue, composed of

Methylene blue,	2 parts
Twenty-five per cent. solution sulphuric acid,	100 parts.

After being washed in water the specimen is immersed for one-half to two minutes in the acid blue, washed off in water, dried between folds of filter-paper, and examined in water.

(2) *Slower Method with Carbol-fuchsin, Counter-stained by Gabbet's Acid Blue.*—This slower method is always more satisfactory, if time permits, and should alone be used for permanent preparations.

The steps are the same until the staining stage is reached, when the cover glasses containing the specimen are placed in the carbol-fuchsin solution, say at five or six o'clock in the evening, and allowed to remain until next morning. They are then washed in water, counter-stained by Gabbet's acid blue solution, washed in water, dried between folds of filter-paper, and studied in water, or, if it is desired to mount the specimen permanently, it is passed through alcohol, mylol, or oil of cloves into Canada balsam. Specimens stained in aniline colors should not be mounted in glycerine, as this gradually withdraws the stain.

When bacilli are very few, in viscid sputum, the centrifugator may be used, or Biedert's method pursued. Fifteen c. c. of the sputum are mixed with 75 to 100 c. c. (about two tea-

Etiology.—Although the evidence in favor of the bacterial origin of tuberculosis may be regarded as conclusive, the readiness with which it lodges and grows varies greatly ; indeed, the number of instances in which it fails to take root doubtless vastly exceeds that in which it does. Hence the contagiousness of tuberculosis is slight, and, although there appears to be no difficulty in transmitting the disease from one domestic animal to another, it is with extreme rarity that a case of tuberculosis in a human being can be traced to another. In a practice of thirty years, including large general hospital service, I can recall but a single instance of probable communication of the disease, and this was from a husband to the wife, who was his faithful nurse for years. It would seem, however, that the contagium is more active than such experience would lead one to suppose. Thus Cornet studied the records of certain institutions whose inmates are devoted to nursing, and discovered the fact that a large proportion of these (62.8 per cent. in twenty-five years) died of phthisis ; also that of 100 nurses, 63 died of this disease. It is to be remembered, however, that the life of the Sisters in convents is unwholesome from too close confinement. On the other hand, the statistics of the Brompton Hospital for Consumptives in London is decidedly against any conclusion that contact with patients peculiarly endangers the lives of doctors, nurses, or attendants. This, too, though they cover a period when no precautions were taken to destroy the bacillus.

Flick's studies also point to a greater activity of the contagium than is usually admitted. He examined all of the houses in a ward in Philadelphia where there had been deaths from consumption, and found that 33 per cent. of such houses had more than one case, that 25 per cent. of these houses had been infected prior to 1888, and that more than 33 per cent. of the deaths which occurred since 1888 took place in them. These observations accord with the results of Cornet's experiments, which demonstrated that the scraping from the walls of phthisical wards inoculated into the lower animals produced tuberculosis.

A truly remarkable experience of Reich is related by Eichhorst.* In the town of Neuenburg, containing 1300 inhabitants, the midwifery cases were about equally divided between two midwives. One of these contracted consumption. She was in the habit of blowing from her mouth into the air passages of the new-born children with a view to clear away the mucus. Within two years ten of the children delivered by this woman died of tubercular meningitis, while of the children delivered by the healthy midwife none showed any sign of tuberculosis.

The conditions which favor the growth and multiplication of bacilli have been carefully studied, but have been only partially determined. One of the best recognized of these,

Heredity, is much more influential when both parents have the disease

---

spoonfuls) of water, four to eight gtt., according to the density of the fluid, of liquor potassæ are added, and the whole boiled, four to six teaspoonfuls of water being gradually added, if necessary, until a thin fluid results. This is allowed to stand in a conical glass for two days, when the supernatant fluid is removed and the sediment is examined as before. It is to be remembered that bacilli treated with alkalies stain slowly, and longer immersion in the staining fluid may be necessary on this account.

* "*Pathologie und Therapie*," Vol. I, p. 559.

than when one is affected. It seems to be true that the child resembling a tuberculous parent is more liable to the disease than one who resembles the healthy parent.

A second favoring condition is *scrofulosis*, or the "delicate constitution." The peculiar enlargement of the lymphatic glands, formerly known as scrofula, is now regarded as true tuberculosis of those glands. There remains, however, a condition called scrofulosis, characterized by paleness, softness, and translucency of the skin of its subject, in whom inflammations run a slow course, and tend to resolve slowly and to terminate in cheesy products. To this some, and notably Rindfleisch, would still apply the name scrofulosis or the tuberculous diathesis. In these the tubercle bacillus finds a favorable soil. On the other hand, there is a tradition not altogether unfounded that persons affected with tuberculosis of the lymphatic glands are less prone to tuberculosis of the lungs than others.

Defective and *insufficient food*, especially when associated with imperfect ventilation, privation, grief, and over-work, are also conditions which favor the growth of the bacillus.

Frequently recurring bronchial catarrh by lowering the vitality of the mucous membrane engenders a soil favorable to the growth and multiplication of the tubercle bacillus. Any of the causes which produce such catarrh may be included among predisposing factors. Particles of dust inhaled in the pursuit of various trades and avocations, as in coal mining, stone cutting, and steel grinding, are well-known to have this effect. Measles, whooping cough, and typhoid fever with bronchial complications are sometimes followed by it.

Damp localities favor the development of tuberculosis, and the very interesting observations of H. P. Bowditch, made a number of years ago, show that in houses thus situated case after case occurs, and whole families have been swept away. It is more than likely these results are dependent on a vulnerability engendered by the "colds" and catarrhs which such localities induce.

No race is exempt, but the colored race is especially predisposed, as is also the American Indian when brought under the influence of civilization. Tuberculosis appears to be spreading among the Indians, even in districts in the Rocky Mountains where the disease is rare among the whites.

Climates characterized especially by frequent, rapid changes of temperature favor the development of tuberculosis. Such are the temperate zones. Tuberculosis is less common in the frigid and torrid zones, but these climates are not exempt.

Age is doubtless a predisposing cause, the susceptible period for pulmonary tuberculosis being between twenty and thirty-five, for meningeal tuberculosis between two and seven, while the lymphatic glands, including the mesenteric and bronchial, are prone to involvement in the first ten years of life. The mesenteric glands are more commonly infected during the first five years of life, including the nursing period, and that during which the child is nourished on milk.

The *shape of the chest* has long been regarded as influencing the development of tuberculosis, and a form of body peculiar to phthisical sub-

jects was described by Hippocrates (b. B. C. 460–357); Galen (b. A. D. 130–200) described the same type of chest. At the present day two varieties of chests are described as phthisical, the *alar* and the *flat*. The first is narrow, shallow, and long, the angles of the scapulæ projecting like wings behind, the proper ratio between the antero-posterior and transverse diameters being, however, preserved. The ribs droop or are unduly oblique. The throat is prominent, the neck long, and the head bent forward. In the flat chest the antero-posterior diameter is disproportionately short, owing to the absence of convexity in the cartilages, which are sometimes even turned in, depressing the sternum and producing a form of chest which, on section, is kidney-shaped. In this form there is not the increased obliquity of the ribs characteristic of the alar chest.

Traumatism is also an agency of acknowledged importance in favoring the lodgement of the tubercle bacillus. This is more particularly seen in the development of tuberculosis of the joints succeeding injury. It is true, however, that contusion of the chest without apparent laceration of the lungs or fracture of a rib has been followed by tuberculosis.

Mode of Invasion and Spread.—The bacillus of tuberculosis is probably omnipresent in the atmosphere, being derived from the drying and pulverization of expectorated sputum. The entrance into the body in the vast majority of instances is by the respiratory tract. Hence the great frequency of tuberculosis in the lungs and bronchial glands, which are the first tissues open to its approach. It is possible, however, for it to enter by the skin, causing lupus or skin tuberculosis. It enters more readily by open wounds. Through the alimentary canal we have an undoubted route of infection. This happens most frequently in children from the use of the milk of tuberculous cows,* in adults from the swallowing of sputum. It is not necessary that the cow should have tuberculosis of the udder to render her milk tuberculous. This has been conclusively shown by Bollinger and confirmed by Hirschberger. The boiling of the milk destroys its infective qualities. Tuberculous meat is less frequently the cause of tuberculous infection by the intestine because it is almost invariably cooked before eating.

The tubercle bacillus having once invaded an organ produces localized tuberculosis, which may or may not become generalized in a manner to be presently described. More rarely tuberculosis may become general from the onset without any local initial lesion being discoverable. This constitutes one of the varieties of acute tuberculosis. Once established, tuberculosis spreads by contiguity and through the lymphatic system and blood. In the former the tubercle grows by the addition of miliary tubercles at its periphery. Through the lymphatic system tuberculosis spreads to the lymphatic glands, and thence to the adjacent serous membranes. The barriers of the lymphatic glands once passed, the blood becomes the medium of a general infection. In the vast

* At the recent meeting of the Association of American Physicians, Washington, May, 1896, Theobald Smith, in a noteworthy paper "On Two Varieties of Tubercle Bacilli from Mammals," said he thought infection of the human subject through the milk of cattle decidedly questionable, and that the subject should be reinvestigated from the standpoint of different species in different groups of animals difficult of inoculation upon any other.

majority of cases generalization takes place from a focus of tubercle somewhere in the system, as the lungs, or a tubercular lymphatic gland, from which the bacilli start their migration.

The favorite seats of the tubercle are the lymphatic glands, lungs, liver, kidney, spleen, intestinal canal, urogenital mucous membranes, the brain (especially its membranes and blood-vessels), the bones and joints. In fact, no tissue or organ is exempt, the salivary glands and pancreas being least frequently invaded.

Morbid Anatomy and Histology of Tubercle.—The miliary tubercle is the beginning of all tubercular deposits. It is itself a compound body composed of smaller submiliary tubercles, of which from ten to 50 unite to form a miliary tubercle. It is about the size of a millet seed, hence the name miliary. By actual measurement it ranges from one to five millimeters ($\frac{1}{25}$ to $\frac{1}{5}$ inch) in diameter. In its young state it is a translucent grey granulation, especially characterized by its want of vascularity. The typical submiliary tubercle is about 0.4 mm. ($\frac{1}{60}$ inch) in diameter, and contains a giant cell in its centre, surrounded by a close infiltration of lymphoid cells or a higher tissue of the lymphadenoid connective tissue type, in the meshes of which are lodged loose lymph corpuscles or larger epithelioid cells. The giant cell may be wanting, and the whole tubercle may be a mass of lymphoid cells, among which the tubercle-bacilli are scattered, or the bacilli may be found in the giant cells, in the epithelioid cells, or even in the lymph cells. When isolated the miliary tubercle is found surrounded by a dense connective-tissue network, welding it firmly to the other tissues, in which it is imbedded. The miliary tubercle is further characterized by its want of vascularity. In thin sections of an injected preparation it will be found that the blood-vessels go up to the tubercle and there terminate abruptly. To this lack of vascularity the tubercle owes its tendency to cheesy degeneration, in the course of which it assumes an opaque white color. When this happens, the centre exhibits under the microscope a granular, ground-glass appearance, while macroscopically tubercle in mass assumes a yellow color.

In certain situations, especially in the lungs, the miliary tubercle forms foci, which gradually increase in size and constitute tubercular infiltration, the yellow or crude tubercle of Laennec and Louis as contrasted with the miliary or grey tubercle. According to these and other observers during the first thirty or forty years of the present century, grey and yellow tubercle were simply differing forms of tubercle. Later, however, the influence of Virchow, Buhl, and Niemeyer (1857–70) caused it to be quite generally accepted that there was but one kind of tubercle—the grey granulation or miliary tubercle, while yellow or crude tubercle was nothing but cheesy inflammatory matter. The subjects of this were still regarded as having phthisis, but not tuberculosis, whence the celebrated declaration of Niemeyer (1866), "The greatest danger to most phthisical patients is the development of the tubercle."*

Even before the discovery of the bacillus by Koch, in 1882, a discovery which resulted in the reëstablishment of the unity of tuberculosis,

* "Niemeyer's Lectures on Phthisis," New Sydenham Society Transactions, 1870, p. 11.

the view began to gain ground, especially through the teachings of Buhl and Rindfleisch, that cheesy matter may be metamorphosed true tubercle, or it may have been primarily scrofulous inflammatory deposit, either of which might produce tubercle by an auto-inoculation. In the meantime, in 1865, Villemin announced the inoculability of tubercle. With the discovery of the bacillus by Koch in 1882, however, began the overthrow of the duality of phthisis, the final result of which was the proposition now generally admitted, *that all phthisis is tubercular.*

The *histogenesis* of tubercle is in no way peculiar. We have only to remember that the bacillus is an irritant. The same response occurs to it as to other irritants. The wandering leucocytes flow from the adjacent vessels and form the lymphoid cells which constitute the bulk of the tubercle. The stabile cells of the connective tissue, the endothelial and perithelial cells of the blood- and lymph-vessels, the epithelium of the serous membranes, proliferate and enlarge, forming the epithelioid cells, and, in some instances, the giant cells, in both of which bacilli may be imbedded. The bacilli seem, however, to vary inversely with the giant cells. Thus, in lupus, joint and lymphatic gland tuberculosis, giant cells are numerous and bacilli scanty, while in lung tuberculosis bacilli are numerous and giant cells scanty.

The reticulum of connective tissue, usually more or less present at the periphery of the miliary nodule, is formed just as is connective tissue in ordinary non-specific inflammation, by the fibrillation of the protoplasm of cells and the rarefaction of the resulting matrix.

The origin of giant cells has been much discussed, but it seems likely that any one of the connective-tissue cells named is capable of developing into a giant cell. It may also perhaps arise from the fusion of individual cells. It contains from four to five to 20 nuclei, commonly arranged in the periphery of the cell.

Tubercle is subject to changes, of which the most frequent and characteristic is *caseation.* It is a regressive change, whereby the primarily transparent tubercular tissue is converted into an opaque yellowish substance of various degrees of consistency, resembling certain varieties of cheese, whence the name. The process is a form of coagulation-necrosis, beginning in the centre of the tubercle, by which the cells are first converted into granular masses without definite outline, and in which nuclei are no longer demonstrable by ordinary staining methods. Bacilli are, however, present. At times, on section, a quasi fibrillation appears in the caseated tubercle which is not to be mistaken for a true fibrous matrix. It may be the result of compression.

Most frequently caseation is followed by *softening.* The precise conditions necessary for this are not known, though commonly, as soon as a caseated mass reaches a certain size it breaks down into a pyoid product which is not histologically pus, but consists of a number of fat drops, granular debris, and shrivelled, formless cells. In the broken down material the tubercle bacilli are exceedingly numerous, much more so than in the dry caseated tubercle. From this circumstance it is held by some that the caseation and subsequent softening are the effect of the bacilli, the action of which is compared to that of bacteria of decomposition. It seems much more reasonable to ascribe these degenerative

changes to defective nourishment of the new formation. This is sustained by the fact that softening does not take place until the tubercular mass acquires a certain size, commonly half a centimeter to a centimeter (0.2 to 0.4 m.) in diameter. The more rapid the formation the earlier does softening set in, that is, the smaller the areas which undergo softening.

More rarely caseated tubercle becomes infiltrated with lime salts and undergoes *calcareous* change, by which a sort of healing is accomplished. The calcareous infiltration of tubercle is more especially prone to occur in lymphatic glands, but also happens rarely in the lungs.

Another form in which the tubercle presents itself is the *solitary tubercle*, which is not made up of united miliary nodules, but consists of a single large, cheesy mass varying in size from that of a pea to that of a human fist. It is almost invariably secondary to primary tuberculosis somewhere else, commonly in the lungs. It is made up chiefly of round cells, in which are found also tubercle bacilli. In the peripheral layers a more fibrous tissue prevails, which, in certain tubercles, becomes so abundant as to give rise to the term "fibrous tubercle." In addition to caseation the solitary tubercle is subject to puriform liquefaction, forming the so-called tuberculous abscess, and also to calcification. The two processes last named may be associated in a single solitary tubercle. Sometimes it is encysted. An especially favorite seat for solitary tubercle is the brain in children, especially the cerebellum at the border between the white and grey substance. The nodules are sometimes multiple. It is found also in the spinal cord, the spleen, the liver, and the heart.

Finally a tubercle, and especially the miliary tubercle, may undergo a *fibroid change*, or sclerosis. Under these circumstances the new formation is converted into fibroid tissue. A certain more limited degree of cheesy metamorphosis takes place at the same time, but the product is a firm, tough nodule. This fibroid change is more prone to occur in tuberculosis of the peritoneum.

So much for the change in tubercle itself. It is, however, capable of exciting *retroactive inflammation* in its own neighborhood. Thus in the lungs a catarrhal pneumonia involving adjacent acini is often produced. In other instances an overgrowth of interstitial tissue ensues. Sometimes it is excessive and results in the so-called fibroid phthisis. More frequently this form of consumption is the result of a coincident irritation by another cause, such as the irritant particles encountered in such occupations as steel grinding, stone cutting, and mining. Associated with tubercular processes, especially in the lungs, is constantly found true suppuration,—mixed infection,—whence the admixture of pus in the expectoration of pulmonary consumption. It is held by some, and apparently by Koch himself, that the tubercle bacillus alone is capable of exciting suppuration.

ACUTE TUBERCULOSIS.

SYNONYMS.—*Diffuse General Tuberculosis; Acute Miliary Tuberculosis.*

The most emphatic expression of the infectious nature of tuberculosis is found in the disease known as acute miliary tuberculosis. In it miliary tubercles develop in different parts of the body as the result of an irruption of bacilli into the blood or lymph channels. The infection is in almost every instance an auto-inoculation, of which the source is a nodule of softening tubercle in some part of the body.

In 300 cases of miliary tuberculosis examined by Buhl, such a source was found in all but ten, while Simmonds in 100 cases found the caseating focus in every instance. The most common seat of such a nodule is the lungs, next a tubercular lymphatic gland, especially a tracheo-bronchial gland. After this there is less constancy, but tubercular joints, a tubercular pleurisy, tubercular peritonitis, and even a skin tuberculosis may be held responsible. Such a nodule may break directly into a vein, furnishing an instance of true embolic infection. Acute tuberculosis occurs most frequently in young persons between twelve and twenty years of age, but adults are not exempt.

Any tissue or organ may be involved, but very seldom do we find all the organs of the body affected, though it is quite common to find lesions in more than two, as, for example, the lungs, the pleura, the membranes of the brain, and the peritoneum. The first three are favorite locations.

Three principal *clinical forms* of acute tuberculosis are recognized, one presenting signs of acute general infection without special localization, another exhibiting, in addition, easily recognizable pulmonary symptoms, and the third cerebral and cerebro-spinal symptoms. The last two being characterized by distinctive local symptoms, their consideration is deferred to that of the diseases of organs. Tubercular pleurisy is similarly deferred for a like reason.

Morbid Anatomy.—This is the anatomy of tuberculosis in the different organs and tissues of the body, and so far as not already described will be given when treating disease of these organs.

Symptoms of General Miliary Tuberculosis.—The general or typhoid form of acute tuberculosis has long been recognized as resembling in a startlingly close manner the symptoms of typhoid fever, and many mistakes have been made in diagnosis because of this resemblance. Since the use of the clinical thermometer in diagnosis, however, such mistakes have been less frequent.

As in typhoid fever, several days, and even weeks, of ill-defined sickness often precede the taking to bed. Fever, with its heightened temperature and frequent pulse are present, as are also the dry tongue, hebetude, and delirium of typhoid. Yet fever is not always present, and afebrile cases are reported by Reinhold and Eichhorst. A slight cough may be present, but not enough to suggest a lung involvement beyond what is characteristic of typhoid fever. If differences are sought in the fever of the two diseases, it will be found that the pulse and respiration may be unduly frequent as compared with typhoid fever, but, above all,

the temperature will be found to differ in its course from that of typhoid fever. There is an absence of the characteristic "tidal wave" rise of temperature of typhoid. There is an evening rise and morning fall, and an occasional inversion, with lower evening and higher morning temperature, takes place, which is held to be characteristic. The range is between 101° and 103° F. (38.3° and 39.4° C.), but may reach 104° or 105° F. (40° or 40.5° C.). The countenance is apt to be more dusky than in typhoid. Repeated examinations of the lungs fail to discover physical signs indicating disease of these organs, and thus the conclusion that there is no lung involvement is apparently confirmed. Later, however, pulmonary symptoms may set in, also meningeal symptoms, the duration of which may lead to a suspicion that the disease is not typhoid fever.

Excessive sweating is a symptom more characteristic of acute tuberculosis than of typhoid fever and may result in sudamina, which also characterize the latter disease. *Herpes* is, however, often present, while it is almost a negatively pathognomonic sign of typhoid. These two symptoms, *i. e.*, sweating and herpes, together with the intermitting fever, constitute a resemblance to malarial fever. Waller and Eichhorst have found rose-colored spots on the abdomen and breast, but they are certainly infrequent. *Enlargement of the spleen* is often present and even *hemorrhage from the bowels* has been noted. Small *albuminuria* is a frequent symptom, not due, as might be expected, to a tubercular involvement of the kidney, but to the fever process.

In view of the general possibilities of acute miliary tuberculosis, there may be *pleural* or *pericardial friction* and other symptoms of pericarditis and pleurisy as well as those of *peritonitis* and *meningitis*.

Tuberculosis of the choroid coat of the eye has been frequently met in acute miliary tuberculosis, more particularly in cases where there has been the widest dissemination. Notwithstanding the difficulties which attend the investigation, the instances in which tubercle bacilli have been found in the blood have been so numerous that in suspected cases it should be examined. Rutimeyer suggests that the blood be taken for this purpose from the spleen by means of a hypodermic syringe, since it has been shown that the blood of this organ may be especially rich in bacilli. On the other hand, bacilli are not often found in the sputum in acute general tuberculosis, even if there be involvement of the lungs, because in this form of tuberculosis the tubercles are situated not in the open air passages so much as in the interstitial tissue of the lung and in the blood-vessel walls.

The course is invariably toward an unfavorable issue. Almost never less than four weeks in duration, it is often eight and even longer, although cases are reported to have terminated at the end of two weeks and even twelve days. Such must, however, be extremely rare.

Diagnosis.—As stated, acute miliary tuberculosis resembles especially typhoid fever, but a carefully kept temperature chart will soon exhibit a difference from that of typhoid. If bacilli are found in the blood and tubercles on the choroid the question is settled at once. The duration of the disease, though short, is apt to be longer than typhoid fever, and before the clinical thermometer gave us its valuable information

the first suggestion that something else than typhoid fever was present came about from noting an absence of the usual defervescence.

The resemblance to intermittent fever has been noted. Here, too, a close study of the temperature will soon show the difference, while a search for the hæmatozoon of malaria should be made. The failure of quinine to cure will settle the question against a malarial cause for the fever.

Prognosis.—Acute miliary tuberculosis always terminates fatally sooner or later, although delusive improvements often raise hopes which are not realized.

Treatment.—Treatment for acute tuberculosis can only be symptomatic. To our present knowledge a cure has never been accomplished. Antipyretics may be used in moderate doses, three to five grains of antipyrin, antifebrin, or phenacetin, the last probably the best; also anodynes to quiet cough or otherwise secure comfort. Supporting food and stimulants are indicated.

TUBERCULOSIS OF LYMPHATIC GLANDS.

SYNONYMS.—*Scrofula, or the King's Evil; Tuberculous Lymphadenitis.*

Etiology.—Even before the discovery of the bacillus of tuberculosis by Koch in 1882, it was generally conceded that what has been known as *scrofula*, or the King's Evil, was a true tuberculosis of lymphatic glands. The minute study of these glands showed the presence of miliary tubercles, and since Koch's announcement the bacillus has been found in them. The bacillus may be regarded as the immediate cause of the specific inflammatory process.

Tuberculous lymphadenitis is most common in children and young adults, but may occur at any age.

Symptoms.—The glands most frequently affected are those of the neck, which appear in various degrees swollen and tender, in many instances suppurating and rupturing when not opened by the surgeon's knife. The submaxillary glands are usually the first involved, but those in the posterior cervical triangle are also frequently invaded on one or both sides, though commonly one side more than the other. The cervical and axillary glands may be conjointly involved, forming a continuous chain behind the clavicle and pectoral muscles. The bacillus usually affects the glands nearest its point of entrance, and presumably the cervical glands are infected by bacilli, which enter by the way of the nasal or naso-pharyngeal passages. The vulnerability of these mucous membranes to the bacilli is, of course, increased by any inflammatory state present. As a rule, there is little or no constitutional sympathy in such a degree of invasion. There may, however, be slight fever.

More rarely there is involvement of all the lymphatic glands of the body. Such cases are sometimes met among negroes. In them there is swelling, pain, and tenderness of all the visible glands, including the cervical, submaxillary, and axillary glands, while autopsy discloses the involvement of bronchial, mesenteric, and retroperitoneal glands. In such cases there is more or less continuous fever, but death is usually

the result of some intercurrent disease, or of pressure upon the respiratory passages.

When the mesenteric or retro-peritoneal glands are especially involved the disease is called *tabes mesenterica.* These cases occur among children. The trunk and limbs are puny, wasted, and anæmic, while their little bellies are prominent, partly because of the enlarged glands and partly from tympany, producing a striking picture. The tympanitic distention often predominates, making it difficult to feel the enlarged glands. In these cases, too, there is often diarrhœa, with thin, offensive stools, yet the bowels are not generally the seat of tuberculosis. There may be tuberculosis of the peritoneum, which may also give rise to an uneven, nodular, tender, and painful enlargement easily recognized by palpation. The disease prevails among poorly fed children in the slums and badly-drained and illy-ventilated houses of the poor. There is fever, fretfulness, and a general aspect of abject misery. Death generally takes place through exhaustion, or some acute intercurrent disease, such as enteritis, carries off the little sufferers. More rarely adults may be affected with tabes mesenterica either as a primary disease or as secondary to pulmonary tuberculosis. I well remember a case associated with peritoneal tuberculosis in which the diagnosis between this condition and carcinoma was difficult, the autopsy determining the question in favor of the former.

In addition to the visible pictures described, the bronchial glands are often involved without visible enlargement, the condition being first found at autopsy, when it may or may not be associated with lung tuberculosis. The enlargements may, however, reach such a size as to form a recognizable mediastinal tumor which may or may not produce the signs of pressure. The bacilli which invade these glands filter through the respiratory passages.

While tuberculous glands of the neck, and even of the axilla, tend to suppurate, the retro-peritoneal and mesenteric glands more frequently caseate without suppuration, and especially characteristic is a tendency in the latter to calcify, furnishing a mode of healing of tuberculosis. The bronchial glands are also less prone to suppurate, but caseate and, at times, liquefy.

Diagnosis.—The diagnosis of tuberculous lymphadenitis requires its differentiation from lymphadenoma (Hodgkins' disease), lymphatic leucæmia, and simple lymphoma. The affected glands in tubercular lymphadenitis are usually more tender than those in Hodgkins' disease; they are more closely adherent to each other and the adjacent tissues, and are, therefore, more fixed and immovable than the glands of Hodgkins' disease. Again, tuberculosis rarely invades more than one group of glands, is associated with caseation and suppuration, while the lymphadenoid growths do not suppurate. Notwithstanding this, the tubercular process is slower. Tuberculosis affects the young—those of either sex under twenty—while Hodgkins' disease occurs at any age, is less frequent in the young, and is more common in males.

From lymphatic leucæmia tuberculosis of lymph-glands is easily recognized by the absence of leucocystosis characteristic of the former. Simple lymphoma also affects a single group of glands, and is doubtless

often mistaken for scrofulosis of the glands about the neck. The glands are, however, harder and less tender, less painful than tubercular glands, and there is less constitutional involvement, less anæmia.

Sarcoma involves groups of glands, but spreads rapidly, invading also adjacent tissues, while carcinoma is always secondary to primary cancer somewhere else.

Prognosis.—The prognosis is generally favorable unless systemic infection occur, recovery being sometimes spontaneous. This is favored by favorable conditions to be mentioned under treatment. In former times "scrofula" was regarded as a protective against consumption. At the present day it is looked upon as a menace because of the danger of systemic infection through it, and it is said that three-fourths of the cases of acute tuberculosis owe their existence to it. Under the circumstances we must regard cases of recovery from tubercular lymphadenitis in childhood as instances of a survival of the fittest. Certainly our present knowledge demands a prompter attempt to eradicate the local condition than was formerly practised.

Treatment.—The general management of a case of tuberculosis of the lymphatic glands is similar to that of a case of tuberculosis of the lungs. The patient should be surrounded by the most favorable hygienic conditions, have the best of food, take cod-liver oil and the iodide of iron. The local use of iodine is undoubtedly efficient at times in dispersing these glandular swellings, probably by exciting an inflammatory process destructive to the bacillus, which in a general way is similar to the reactive effect of tuberculin.

When suppuration has set in it is best to open an exposed abscess with the knife, because if allowed to open itself there is apt to result an unhealthy sinuous ulcer, very slow to heal, and when healed causing marked disfiguration by unsightly cicatrices. The access of air permitted by the opening seems also to be antagonistic to the life of the bacillus, for with the healing of the abscess the tubercular process stops in that particular gland. Counter-irritation by any means seems to act similarly, although iodine appears to be the most efficient.

LEPROSY.

Synonym.—*Elephantiasis græcorum.*

Definition.—Leprosy is an infectious disease, due to the *bacillus* lepræ, characterized by a subcutaneous and submucous nodular infiltrate or by similar infiltration of nerve trunks. The former constitutes tubercular leprosy, the latter the anæsthetic variety.

The disease is described in the Books of Moses, who gave many rules for its recognition, the isolation of victims, the test of recovery, and rules to be complied with before the convalescent could mingle with his people, and is identified with the early history of Egypt and India. It prevailed in Europe in the Middle Ages but has become almost extinct there, except in Norway and Sweden, Hungary, and Roumania. In Greece and Turkey, Palestine, Syria, Egypt, India, China, and Siam it is still endemic.

Historical and Etiology.—The bacillus of leprosy was discovered by Hansen in 1880, and subsequently clearly described by Neisser, and is especially characterized by its close resemblance to the tubercle bacillus. The bacilli are delicate rods whose length equals ⅓ to ½ the width of a red blood disc. They are for the most part found in the interior of cells, rarely outside of them. Some of these cells are of large size and known as lepra cells. In the former they often form clumps in their interior. The bacilli are exceedingly numerous in leprous tissue. They stain readily in anilin colors, but not in vesuvin, differing in this respect from tubercle bacilli, and also in that they liquefy coagulated blood serum, while tubercle bacilli do not. In the fresh condition the lepra bacilli exhibit active movement.

While the disease is contagious, its spread, under the most favorable circumstances, is exceedingly slow, the most intimate contact, as that between parent and child, being often unattended by inoculation. Experimental inoculation was, however, successfully performed on a Hawaiian convict by Arning, as well as in rabbits by Melcher and Artmann. According to Morrow, in the majority of cases the disease spreads by sexual intercourse, but cracks and fissures in the skin also favor the lodgement of the bacillus. In certain countries, especially the tropical, its spread is more rapid. Such are India, where there are said to be 250,000 lepers, and the Sandwich Islands, where, in 1889, there were 1100 in the settlement at Molokai. In the West Indies there are also many cases, and some remarkable morbid specimens from Trinidad were exhibited by Dr. Beaven Rake at the Pan-American Medical Congress, held in Washington, U. S. A., in September, 1893.

In this country the cases are for the most part isolated ones which enter by the seaports of the Pacific and Atlantic coasts. In Tracadie, on the Gulf of St. Lawrence, there is, however, a leper settlement, the disease having been brought from Norway in the latter part of the eighteenth century. The number of cases is being gradually reduced, there being now but 18 as compared to 40 a few years ago. This is apparently the result of segregation, which is now generally practised where possible.

All ages and sexes are liable to this disease. Animals are not subject to it, although guinea pigs have been successfully inoculated. A curious impression has arisen that the disease is caused by eating spoiled fish or vegetables. To this view Jonathan Hutchinson has given the weight of his opinion. In view, however, of the acknowledged bacillary origin of the disease, this can only be considered as a predisposing cause that lowers resistive vitality by altering nutrition.

Morbid Anatomy.—Tubercular leprosy is characterized by its nodular outgrowths on the skin, which are made up of a small-celled infiltrate, maintaining itself for a considerable time, after which it breaks down and ulcerates. The ulcers may heal, producing cicatrices. The mucous membrane is also invaded, particularly that of the eyelids, the conjunctiva, cornea, and larynx. Lymphatic glands, cartilage, liver, lungs, and spleen are also at times affected. The lepra nodes are vascular, differing in this respect from tubercles.

The morbid anatomy of the anæsthetic variety is included in the anatomical changes of the skin described in the symptomatology of that type of the disease.

Symptoms.—Nothing is known of a period of incubation. The outbreak of the disease is apt to be preceded by an *intermittent febrile movement*, which has been mistaken for intermittent fever and which may last for one or two years. There is often an erythematous redness of the skin, which in places becomes pale and in others assumes a brownish tinge. From this appearance the name macular leprosy has been applied to certain cases which go no farther. From these spots the pigment may also disappear, leaving perfectly white anæsthetic areas—*lepra alba.*

In the *tubercular* form, which is the more common, an *infiltration of the skin* with *tubercular nodules* takes place. These remain for a long time intact without degenerating, but sooner or later, as a rule, though often only after many years, softening and ulceration take place. Some of them, on the other hand, gradually disappear spontaneously. The number of nodules varies greatly. Some of them are pediculated, others amount to a simple thickening of the skin, which is conspicuous in such portions as the eyelids, nose, and ears, parts of which may disappear by ulceration. Even the cornea and conjunctiva may be the seat of nodules, and blindness may result.

The same development may take place in mucous membranes producing obstruction of the respiratory passages, including the nose and larynx. There may also be leprous deposits in internal organs, including the liver, spleen, lungs, and lymphatic glands.

In the nervous or *anæsthetic* form the peripheral nerves become infiltrated with the leprous growth and are converted into thickened cords which may even be felt under the skin. These are at first painful, but later become anæsthetic. Trophic phenomena of a striking character result, producing dryness, smoothness, and tightness of the skin with a total absence of nodules. Atrophy and wasting ensue from the same cause, and toes, fingers, and even larger limbs drop off. Great vesicles also sometimes form.

Diagnosis.—The diagnosis of the tubercular form is not difficult. The anæsthetic variety resembles closely certain forms of scleroderma, but the trophic changes are more extensive. The resemblance of the early stage to intermittent fever has been referred to. The diagnosis may be made absolute by the detection of the bacilli in portions cut out for the purpose of study.

Prognosis.—The course of the disease is almost always prolonged, and the patient may die from intercurrent causes. In some cases death results from the gradual exhaustion of the system, which is more rapid in the ulcerative forms. It is said that recovery has taken place, although this must be doubtful.

Treatment.—So far as known, treatment is unavailing. Segregation should be practised whenever possible, for such a course is invariably accompanied by a falling off in the number of cases, and the continued practice of this method must ultimately result in its being stamped out.

Among the remedies which have been recommended are mercury and iodine by inunction. Internally, iodide of potassium, creasote, and salicylic acid, chaulmoogra oil and gurjun oil. The first has most reputation, and is regarded by Danielson after forty years' experience as distinctly useful. Chaulmoogra oil is used in doses of two drachms (0.8 gm.) every two hours, and gurjun in doses of ten minims (0.66 gm.). The latter may also be used by inunction.

ACTINOMYCOSIS.

SYNONYMS.—*Big Jaw; Swelled Head; Bone Tumor.*

Definition.—An infectious inflammatory disease of cattle, communicable also to man, and depending for its existence on a peculiar fungus named by Hartz, a Munich botanist, *actinomyces*, or ray-fungus.

Historical and Etiology.—The great German surgeon, Langenbeck, was the first to discover, in 1845, that the disease, "big jaw," previously well known in the slaughter houses of Germany, could be communicated to man. His results were not, however, published until 1878, a year after Bollinger discovered that it was due to a fungus. Bollinger took it to Hartz, who gave it its name. The same year, 1877, James Israel, of Berlin, found the fungus in man, but did not recognize it as identical with the Hartz fungus. It was reserved for Ponfick in 1879 to establish thoroughly the identity of the disease in man and in cattle. Belfield, of Chicago, first recognized the parasite in cattle in this country. Henry F. Formad and George A. Bodamer first studied the disease in Philadelphia, and through them I was able to examine specimens of the swelled head from the slaughter houses of that city.

The fungus belongs to the species *cladothrix*, and is known as the ray fungus. As found in the pus from man and cattle affected with the disease, it appears as a small, yellowish granule from one to two millimeters ($\frac{1}{25}$ to $\frac{1}{12}$ inch) in diameter, detectable by the naked eye. By the microscope the granule is resolvable into conical threads, radiating from a centre to which they are attached by their small ends, the other club-like ends being outward. This gives the external surface a mulberry appearance. The centre is composed of a granular substance, containing numerous bodies resembling micrococci. The disease has been reproduced by inoculation of the fungus from a diseased animal, as well as by the inoculation of cultures. It is thought to arise primarily in animals in the course of their feeding on vegetable matter. This is the more reasonable because the ray fungus has been isolated from vegetables. A similar origin is ascribed to it in man.

The effect of the parasite is to produce granulomatous and fibromatous new formations, which ultimately become the seat of suppuration. The former, like tubercle, is composed of small round cells, epithelioid cells, and giant cells. The fibrous matter consists of proliferated connective tissue about the granulation growth, expanding and enlarging the bone until it resembles an osteo-sarcoma, for which it was for a time mistaken.

The tendency to suppuration is more marked in man than in cattle, where the process is a more localized one, while in man the disease runs

its course with the formation of multiple abscesses and chronic pyæmia. Such course is supposed to be due to an admixture in man of pyogenic organisms with the true ray fungus. Associated with the suppurative process in man is a tendency to fatty degeneration of the cells of the granulation tissue.

Morbid Anatomy.—In addition to the lesions presently to be described about the jaw and head, there are found in the lungs, when the latter are invaded, the miliary nodules alluded to, made up of groups of fungi, surrounded by granulation-tissue. Broncho-pneumonic areas and abscesses large enough to be recognized by their physical signs during life may also be present. Erosion of the vertebræ, ribs, and sternum may also occur.

Symptoms.—The route of infection is generally the mouth, while the special seats seized upon are carious teeth, whence the *jaw* is invaded and becomes swollen. The *swelling* may extend thence to the face and temporal region, and even the neck, producing *discharging sinuses* like those associated with dead bone. Alongside of these are cicatricial marks of healing. More rarely the tongue, fauces, and even the intestines, large and small, and the liver are invaded. The latter organ may also become involved metastatically. The fungus has been found in the stools, first by Ransom, and pericæcal abscess has been found due to it.

The *lungs* are also favorite seats of invasion by actinomycosis, and it was in these organs in man that James Israel recognized the fungus which proved to be the ray fungus also. The symptoms produced are those of bronchitis,—fever, cough, and more or less fetid expectoration, in which the fungus is occasionally found. In the lungs the posterior and lateral parts are affected rather than the apices. They may be invaded simultaneously with the jaws. The course of lung actinomycosis is chronic, and resembles that of pulmonary consumption, the average duration in man being ten months.

Actinomycosis may occur in connection with the *skin* alone, and even in the *brain* abscesses may occur containing the mycelium. Bollinger has reported a case of the primary disease in the brain of man, while Gamgee and Delpine and O. B. Keller have found it in the brain secondary to pleural invasion. The metastatic abscesses are the direct result of the transfer of a portion of the fungus.

Diagnosis.—Sarcoma of the jaw presents a macroscopic picture very like that of actinomycosis, but its course is more rapid and there is less suppuration, yet these signs are of themselves insufficient, and the recognition of the fungus may be necessary to a diagnosis.

More frequent, perhaps, than any other error is that which mistakes the disease for pyæmia, of which, indeed, as it occurs in man, it is a chronic variety. There are the same sort of metastases in the lungs and elsewhere; in man with pus formation, in animals without or with slight suppuration.

Treatment.—The treatment is surgical, consisting in thorough extirpation, the opening of the abscesses and removal of the dead bone, followed by thorough drainage. Iodide of potassium in doses of 40 to 60 grains (3.66 to 4 gm.) has been recommended by Thomassen. Cures are reported from its use.

INFECTIOUS DISEASES OF DOUBTFUL NATURE.

EPHEMERAL FEVER—FEBRICULA.

SYNONYMS.—*Irritative Fever; Gastric Fever; Simple Continued Fever.*

Definition.—A fever of short duration, depending on a variety of irritative causes. A febrile movement, lasting twenty-four hours and disappearing, may for convenience be called *Ephemeral Fever;* if of three or four days' duration, *febricula.*

Etiology.—The most frequent cause of this form of fever is probably the ingestion of foods difficult of digestion, either inherently or by reason of some temporary functional derangement of the stomach. In a word, dyspepsia is perhaps the most frequent cause of such a fever. This is especially the case with children, in whom the condition is often spoken of as gastric fever.

Another cause is probably exposure to cold insufficient to produce a bronchitis, tonsillitis, or other affection, too slight to be recognizable by the usual signs. Undue exposure to the sun, too, may produce it, or even fatigue. The inhalation of noxious gases is a possible cause, though somewhat discredited by recent studies;* also, the absorption from the stomach and intestine of lower toxic albumoses from putrid or decomposing foods—auto-intoxication by ptomaïnes.

It is possible, too, that the germ of an infectious disease or its toxic products may enter the economy in quantity insufficient to develop the specific affection which is its usual result. Possibly the poison of rheumatism or malaria may operate in this way.

Symptoms.—The symptoms of irritative fever are those usual to fever in mild degree, *i. e., moderate elevation of temperature*, rarely above 103° F. (39.4° C.), *frequent pulse*, *headache*, a sense of *lassitude* and *weariness*, *loss of appetite*, *nausea*, and *restlessness;* in children perhaps *delirium.* The fever is apt to terminate suddenly by crisis on the third or fourth day.

Diagnosis.—The diagnosis resolves itself into this: where a careful search fails to reveal the operation of a cause, save one of those referred to, and no symptoms develop characteristic of any of the recognized diseases, the affection is irritative fever.

Prognosis.—Always favorable.

Treatment.—Rest in bed, a simple aperient, a fever mixture consisting of solution of citrate of potash, sweet spirit of nitre, solution of acetate of ammonia or aconite tincture, will suffice to break up the fever and ensure recovery.

* Abbott, A. C., "Effects of the Gaseous Products of Decomposition on the Health, Etc." Trans. of the Asso. of Amer. Physicians, Vol. x, 1895.

PROTRACTED SIMPLE CONTINUED FEVER.

Definition and Etiology.—It seems necessary for the present to continue this term for a feverish process of a longer duration than ephemeral fever or febricula—a fever of long duration that is not typhoid, not influenza,—lasting from two weeks to three months, and without definite lesions. Knowing, however, what we do know, and limited as our knowledge still is of infection, it is more than likely that some day a specific cause will be found for each of a motley group of such fevers, which will give them a definite name, just as cases formerly thus grouped are now relegated to typhoid fever.

Some of these cases, too, may belong to the group covered by the terms *cryptogenetic septicæmias* suggested in 1878 by W. v. Leube, cases of general septicæmia with concealed local infection, undiscoverable even at necropsy, characterized by a fever which persists for weeks. Many of these recover completely, including cases in which the natural doubt as to whether they are of malarial or tubercular origin is settled in the usual way, against malaria by the inefficiency of quinine, and against tuberculosis by reason of recovery. J. M. Da Costa has well described such cases in a recent paper on "Protracted Simple Continued Fever."* Some of the more serious forms have been traced after death, by the aid of the bacteriological examination, to the streptococcus, staphylococcus, and even pneumonococcus infection. Cases of prolonged fever succeeding pneumonia and pleurisy which subsequently recover may well be ascribed to any of these organisms.

Symptoms.—It can scarcely be said of the symptomology of the milder forms of these fevers to which reference is here intended, that it includes more than a mild *fever*, seldom exceeding 103° F. (39.4° C.), with slight morning remission and evening rise, and the usual high colored urine; it may be with mild *gastro-intestinal derangement*, such as a slightly coated tongue, but no diarrhœa, no lung complication, nothing essential but the mild fever. The latter is, however, rarely higher, and there is occasionally enlargement of the spleen.

These fevers admit, moreover, of a certain classification, based on locality and perhaps modifying local cause. This would be the case with the *thermic fever* of the South, described by my colleague, John Guitéras, characterized by *wakefulness*, great *nervous excitement* and *disordered muscular function*, with unimpaired appetite, but without eruption or other symptom of typhoid fever, and lasting for several weeks. This, as suggested by Da Costa, is probably also the *ardent fever* of the older writers, in its severer form the inflammatory fever, described by Copeland. On thermic fever, Guitéras tells me lately, he has changed his views and is forced, in the light of modern studies, to ascribe it to some unknown infectious cause. To this, he says, he has been led by two facts, first, that he finds it farther north than he originally thought it occurred—his original studies were made at Key West—and, second, its occurrence in more than one member of a family.

* Trans. of the Asso. of Amer. Physicians, Vol. xi, 1896.

Such, too, may be the "Asthenic Fever" of Murchison, and the "Starvation Fever," described by Da Costa;* the "Atypical Continued Fever of Nashville," described by Cain;† "Simple Continued Fever," described by Baumgarten,‡ of St. Louis; the "Malta or Rock Fever," to be described; the "Innominate Fever" § of Goodhart, who says in his paper, "There is too great a tendency to label all continued fevers by some definite name;" and the "Inexplicable Fever" of Hale White.‖

Diagnosis.—The cases are to be distinguished above all from irregular and mild forms of typhoid fever, similar forms of remittent fever, miliary tuberculosis, the fever which sometimes attends chlorosis, hysteria at times, and some other nervous disorders. Da Costa emphasizes a feverish state caused by lithæmia, another in rapidly advancing sclerosis, which may be separated by other distinctive signs, usually evident when sought for. In cases where there is enlargement of the spleen the resemblance to typhoid is closer and the diagnosis may have to remain in doubt until settled by time. The tubercle bacillus should always be sought for in doubtful cases; also the plasmodium of malaria.

Prognosis.—This is generally favorable, except in some of the severer cases ultimately traceable to true infection. Some cases of the so-called thermic fever, reported by Guitéras, died and came to autopsy without definite lesions being discovered.

Treatment.—The treatment of simple continued fever of longer duration, as well as of the shorter forms, is symptomatic, and remedies for the relief of symptoms are for the most part alone indicated. With continued fever there is always a tendency to weakness, and supporting measures are indicated, including quinine, strychnine, and small doses of iron. Due attention to the bowels should be given, as the effect of constipation in keeping up fever is well known.

WEIL'S DISEASE.

Synonym.—*Acute Febrile Jaundice.*

Definition.—An acute infectious disease, characterized by jaundice and fever, described by Weil in 1886.

Etiology.—The cause is as yet undetermined, but it affects males in preference to females, especially butchers, laborers, and brewers, and its subjects are from twenty-five to forty years of age. A few cases have occurred in this country, two having been reported from the Philadelphia Hospital by J. H. Musser and John Guitéras.

It occurs commonly in the summer months, and nearly always in groups of cases.

Symptoms.—The disease sets in suddenly, most frequently with a *chill* and without prodrome. There is *fever* with temperature of 102° to

* Trans. of the College of Physicians of Philadelphia. Third Series. Vol. v.
† Southern Practitioner. December, 1891.
‡ Trans. of the Asso. of Amer. Physicians. Vol. viii, 1893.
§ Guy's Hospital Reports. Vol. xxx, 1888.
‖ Br. Med. Jour. Vol. ii, 1886, p. 1096.

104° F. (38.9° to 40° C.), *headache*, muscular and joint *pains*, and *epigastric pain*, which is characteristic. There is especially *tenderness* in the *calf muscles*. *Jaundice* promptly makes its appearance. The fever lasts usually from ten to fourteen days, and is characterized by decided remissions. The *liver* and *spleen* are both *enlarged;* the former may be tender. Associated with the jaundice are the usual *clay-colored stools* of obstructive jaundice. Beyond the epigastric pain, which may be hepatic in origin, gastro-intestinal symptoms are not marked. The tongue is coated, and there may be vomiting and diarrhœa. There may be dizziness, confusion of mind, and even delirium. The *urine* contains biliary coloring matter; sometimes albuminuria with casts and even blood.

After a duration of from eight to fourteen days, convalescence sets in, usually slowly, and it may be much prolonged.

Diagnosis.—The conditions with which Weil's disease might be for a time confounded are bilious remittent fever, acute yellow atrophy of the liver, and phosphorus poisoning. The former would be excluded by the absence of the plasmodium of malaria, while the mildness and favorable termination would exclude the other two diseases.

Prognosis.—Recovery is usual, but a few autopsies have been made with the discovery of no definite morbid anatomy. There is cloudy swelling and even fatty degeneration of the cells of the heart, liver, kidney, stomach, and intestines.

Treatment.—This is symptomatic.

MILK SICKNESS.

Synonyms.—*Trembles; Puking Fever; Slows.*

Definition.—An infectious disease prevailing in the western and southwestern parts of the United States, characterized especially by trembling, vomiting, constipation, and a peculiar fetor of the breath.

Etiology.—A like disease prevails among the cattle of the infested districts, and it is supposed to be communicated to man through the milk and its products, viz., cheese and butter, and also flesh when used as food. It is more common in summer and autumn and in dry seasons. Nothing more definite is known as to its cause.

Morbid Anatomy.—Our knowledge of the morbid anatomy of milk fever is chiefly by inference from that obtained by necropsies on cattle, those on man being few and imperfect. The lesions noted by Grof under these circumstances are as follows: Cerebral sinuses; meningeal vessels of the brain and cord distended with blood; pia mater opaque and overlaid with purulent exudate; brain soft; stomach and intestines contracted and mucous membrane injected; lungs, liver, kidneys, and spleen engorged with blood, the liver and spleen soft, the latter enlarged in cases to twice the normal size, the blood fluid.

Symptoms.—There is usually a *prodrome* of two or three days, manifested by simple *uneasiness* and *discomfort*, after which the disease is usually ushered in suddenly by severe epigastric *pain*, *constipation*, *nausea*, and *vomiting*. Hence the term "puking" sickness. There is also moderate *fever* and disproportionate *thirst*. The *pulse* at first is full,

later small and rapid. There is marked *tremor* or muscular twitching on attempt at motion. The constipation is characteristic. The tongue is swollen and the breath is peculiarly foul. This is said to be diagnostic. A typhoid state may supervene, preceded by restlessness, irritability, coma, and even convulsions.

The duration of the disease is from two to ten days or longer. The short cases are the fatal ones. When recovery takes place convalescence may be protracted three to four weeks.

Treatment.—The treatment is symptomatic, and consists chiefly in combating by alcohol, aromatic spirit of ammonia, and food the tendency to weakness. Happily, the disease appears to be dying out as land is improved.

Prophylaxis may be secured by fencing off cattle affected and carefully guarding against the use of infected food and milk.

MALTA FEVER.

Synonyms.—*Mediterranean Fever; Neapolitan Fever; Rock Fever.*

Definition.—An anomalous fever, characterized by remissions at intervals of from three to six weeks, remissions lasting a few days only at first, but lengthening with each recurrence until they attain a duration of a month or six weeks, with, as a rule, ultimate recovery.

Etiology.—The cause of this peculiar fever has been traced to a bacterium, the *micrococcus melitensis,* by Bruce, whose researches have been confirmed by Hughes. Pure cultures have been obtained and in six cases the disease has been reproduced in monkeys. It has been regarded as a form of typhoid, also malarial typhoid, and, in consequence of enlargement of the mesenteric glands, noted by Italian observers, called adeno-typhoid. It has also been thought to be an anomalous form of malarial fever, and again has been ascribed to "chronic poisoning with fæcal accumulation." The disease attacks young persons for the most part.

Morbid Anatomy.—Our knowledge as to the morbid anatomy of Malta fever is as unsettled as that of its etiology. Thus Bruce says no characteristic lesion of typhoid fever is found, while Perry says of "rock fever" that in 100 autopsies made during four years' residence in Gibraltar, the typical lesions of typhoid were present without exception.

Symptoms.—There is usually a period of incubation of from six to ten days. The onset is gradual, with *headache*, *sleeplessness*, and *loss of appetite*, without chilliness or high fever at first, but with *thirst.* There is no diarrhœa; no spots are found. These symptoms, more or less pronounced, last three to four weeks, when the first remission sets in, simulating convalescence. It lasts a few days only, when the first relapse appears, this time with *rigors*, high *fever*, and often *diarrhœa*, and the symptoms of the first attack intensified. This relapse lasts for from five to six weeks, to be followed by another remission of from ten days to two weeks. Then follows the second relapse, when recur the symptoms of the first relapse, to which are superadded great *debility*, *night sweats*, *pain* in the larger joints, including hips, knees, and ankles, and in the testicles,

one or both. Then follows a third remission, which may last for a month or six weeks. Then a third relapse, adding to the other symptoms a heavily *coated tongue*, a high temperature, 105° F. (40.5° C.) and above in the evening, but normal in the morning, the night sweats and especially the rheumatic pains being markedly severe. All the joints now seem to be involved and motion is an agony. The fibrous tissues are also often involved in this relapse, especially the tendo-Achillis and fibrous structures about the ankle; also the lumbar aponeuroses and sheaths of the nerves from the sacral plexus.

Treatment.—This is symptomatic, being directed to the relief of the symptoms and the support of the patient against the exhaustive effects of the disease.

MILIARY FEVER.

SYNONYMS.—*Febris miliaris; Sudor anglicus; Sweating Sickness; the Sweating Disease of Picardy; the English Sweat.*

Definition.—An infectious fever of unknown cause, characterized by profuse sweats and an eruption of miliary vesicles.

Historical.—The disease first appeared in London in an epidemic of extreme severity in the summer of 1486, a year characterized by very wet weather. There were other epidemics in 1507, 1518, and 1529. During the latter the disease passed on to the continent of Europe. There was not another epidemic until 1718, when there appeared "the sweating sickness" of Picardy, France, extending thence into Italy, Germany, Austria, and Belgium. Then there followed 194 epidemics up to 1879.

Etiology.—As to the specific cause nothing is known. It is not contagious nor inoculable, and not favored by crowding. Most epidemics occur in summer, fewest in the autumn; second in frequency is the spring and third the winter. Moist, warm, and unchanging weather favors the disease. Contaminations of the soil, such as arise from neglected drains and collections of refuse, also contribute to its causation. More women are affected than men, and the vulnerable age seems to be between twenty and fifty years. The healthy and strong are as likely to be attacked as the weak, and the rich as well as the poor.

Symptoms.—After a *prodrome* of two or three days, the patient goes to bed apparently well, and wakes up in the night dripping with *sweat*. With this is a *sense of oppression*, and even *pain* in the præcordial region, *tenderness* and pain in the epigastrium, *palpitation*, *headache*, *dizziness*, and *muscular cramps*. The *temperature* is abnormally high, the *pulse* and *respirations* frequent; there is even *dyspnœa*, sometimes very violent. The perspiration continues, saturating the bed-clothing and diffusing an unpleasant odor throughout the room.

On the third or fourth day, as the result of the profuse sweating, miliary *vesicles* make their appearance, at first so minute as to be scarcely visible, though they may be felt by passing the hand over the skin. As they become larger they are easily visible by their crystalline contents, which later becomes turbid and even milky. They appear first on the neck and breast, then over the back and extremities, less frequently on

the abdomen and scalp. After two or three days they burst, dry up, and form crusts, which subsequently desquamate. With the appearance of the eruption the other symptoms disappear rather suddenly, but there is often noted a burning and prickling sensation of the skin. There is generally loss of appetite, sometimes nausea, seldom vomiting, scanty urine, and especially constipation.

The *duration* of the disease is usually six to eight days, although it may be prolonged beyond this, the eruption being sometimes delayed to the seventh, tenth, and even fifteenth day. Relapses sometimes occur. Sometimes the disease assumes an intermittent character.

Diagnosis.—This is not difficult. The prevalence of an epidemic, profuse sweating, and rash scarcely permit an error.

Prognosis.—The prognosis has varied greatly in different epidemics, the mortality in some of the earlier reaching as much as 50 per cent., while in others none died. The average may be put down at from eight to nine per cent.

Morbid Anatomy.—No characteristic anatomical changes have been noted in miliary fever. The internal organs are generally hyperæmic. The spleen is often enlarged. The most striking feature is the tendency to rapid decomposition, "beginning almost during life," as has been said. The blood is thin and dark colored.

Treatment.—The treatment is mainly expectant and symptomatic. Simple febrifuges and acid drinks are indicated. Warm baths and sponging of the skin with warm water are soothing and comforting. The precordial distress and apnœa may require anodynes, preferably subcutaneously administered. The sweating itself, if alarming, may be treated by hypodermic injections of atropine, $\frac{1}{100}$ to $\frac{1}{60}$ grain (0.00064 to 0.00108 gm.), p. r. n., or this drug may be given by the mouth in the same dose.

SECTION II.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE MOUTH.

THE COATED TONGUE.

The *natural color* of the tongue at its anterior two-thirds is a pale red, on which the fungiform papillæ stand out as brighter red points. The epithelium covering the filiform papillæ, which are much more numerous and uniformly spread over the dorsal surface of the tongue, is thicker, and they are therefore less distinctly seen. As the base is approached, a greyish color is assumed on account of the greater thickness of the epithelium, on which the walls of the circumvallate papillæ appear in two rows of red circles. In the "furred" tongue the epithelium is abundant, though it is doubtful whether it is produced in increased quantity, or is simply raised by hyperæmic swelling of the papillæ. The "fur" is also contributed to by various forms of fungi. Too much stress should not be laid on the coated tongue. Some persons have a coated tongue and are perfectly healthy, while others have fair looking tongues and are ailing seriously with those derangements which are commonly attended with coated tongue, especially gastro-intestinal disturbances. Food such as milk also contributes to the coating of the tongue.

The *dry*, brown color of the tongue in low fevers is due to a drying of the rapidly exfoliating epithelium, admixed sometimes with mucus or saliva. It may also be found coated with dried food and sometimes with dried blood, due to capillary hemorrhage, which imparts to it a black color—the *black tongue* of certain malignant fevers. The tongue is sometimes *pale* and *anæmic* in persons whose blood is poor and deficient in red blood discs. The tongue in these cases is sometimes enlarged and flabby, while its edges are easily indented and marked by the teeth. A *bright red* or even a raw appearance of the tongue is met with in certain fevers, particularly in the early stages, when it may also be dry and glazed. It may be coated at the beginning, but later the epithelium desquamates freely and the whole surface may be red; or the fungiform papillæ may be hyperæmic, swollen, and unusually distinct, constituting the "strawberry" tongue so characteristic of scarlet fever. The raw-beef appearance of the tongue is often seen toward the close of exhausting diseases, like tubercular consumption.

DERANGEMENT DUE TO DENTITION.

The most serious accident of dentition is what is known as the reflex convulsion, which will be considered among nervous affections. Other derangements of gastro-intestinal nature will be discussed under diarrhœa. These are not always reflex. They may be excited by irritation of the swallowed saliva, which is not only increased, but also altered in quality. Other anomalous conditions are observed in the natural order of eruption and certain markings on the teeth, ascribed to stomatitis.

The order of natural eruption of the milk teeth is well shown in the accompanying diagram. The first to appear are the lower cen-

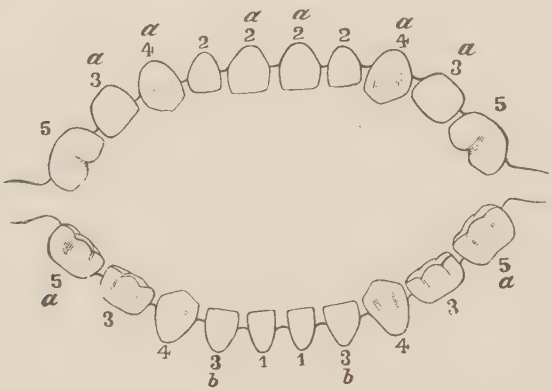


FIG. 15.—DIAGRAM SHOWING ERUPTION OF MILK TEETH.

The numerals 1 to 5 indicate the groups in which the individual teeth are erupted. The letters *a* and *b* indicate the precedence of eruption in the different groups.

1, 1. Between the fourth and seventh months; pause of three to nine weeks. 2, 2, 2, 2. Between the eighth and tenth months; pause of six to twelve weeks. 3, 3, 3, 3, 3, 3. Between the twelfth and fifteenth months; pause until eighteenth month. 4, 4, 4, 4. Between the eighteenth and twenty-fourth months; pause of two to three months. 5, 5, 5, 5. Between the twentieth and thirtieth months.—(*From Starr.*)

tral incisors (1, 1) at the age of from four to nine months, then a few weeks later the upper central incisors (2, 2), and next the upper lateral incisors (3, 3). Not until the beginning of the second year come the lower lateral incisors (3, 3) and almost simultaneously the four anterior molars (3, 3, 3, 3). In the second half of the second year come the four canines (4, 4, 4, 4), two "eye," two "stomach" teeth, and finally the four posterior molars (5, 5, 5, 5). So that by the end of the second or beginning of the third year the first dentition is completed. The milk teeth begin to be replaced by the permanent set in the fifth or sixth year.

In some children (usually the rachitic, the feeble, and badly nourished) the appearance of the milk teeth is greatly delayed,—the lower incisors do not appear until the eleventh or twelfth month,—but the completion of dentition is not much delayed thereby. Under these circum-

stances dentition is sometimes not completed until the end of the third year. In others they appear earlier, in the third or fourth month, and occasionally children are born with them. It has always seemed to me that the first appearance of the teeth is more apt to be delayed in blondes and anticipated in brunettes.

The diet of children during dentition should be very carefully watched, as the whole gastro-intestinal tract is sensitive and irritable and readily thrown into inflammation. The mouth is tender, the saliva flows freely, and the child is disposed to bite on anything. The term "tooth rash" is applied to certain eczematous eruptions which sometimes appear during teething. Their relation to teething is not certainly established.

Very rarely a purulent conjunctivitis makes its appearance during the eruption of the upper canines or "eye teeth," which is ascribed to dentition and explained by contiguous extension of inflammation through the antrum of Highmore and the lacrymo-nasal duct.

Certain markings are often found on the teeth as a consequence of stomatitis. They include pittings and linear depressions, the result of defects in the development of the enamel. Extreme degrees produce a honey-combed appearance. These, as well as the syphilitic teeth of children, have been studied by Jonathan Hutchinson, and are not to be confounded with the latter. (See Fig. 13.) The "honey-comb" changes are most conspicuous in the permanent teeth, of which the first molars, according to Hutchinson, are the test teeth, though he says the incisors are almost as constantly pitted, eroded, and discolored, often showing a transverse line which crosses all the teeth at the same level. These transverse furrows are also ascribed by Magitot to infantile convulsions or other severe illness in early life.

STOMATITIS.

Simple Acute Catarrhal Stomatitis.

Definition and Etiology.—A simple erythematous inflammation of the mouth, commonly caused by diffuse chemical or mechanical irritants, such as over-heated food (very hot drinks), acids, alkalies, stimulating condiments (red pepper, horseradish, and the like), by excessive smoking and use of alcohol. It occurs in adults and children from the action of such causes, independently of the state of health, but is prolonged when its subjects are unhealthy and ill nourished. Dentition is also a cause, while stomatitis may accompany also indigestion and the acute fevers.

Symptoms.—The mucous membrane is reddened wherever the irritation has reached, but the redness may be greater in certain situations, as on the tongue, gums, lips, and cheeks. There may be at the very beginning dryness, but it is soon followed by increased secretion and slight swelling. There is always discomfort, which may amount to pain, which is increased by the introduction of food and its mastication. A corresponding slight febrile movement may be present.

Treatment.—The treatment of simple catarrhal stomatitis will be considered in connection with that of the other forms of stomatitis to be described.

Aphthous Stomatitis.

Synonyms.—*Aphtha; Canker; Follicular Stomatitis; Vesicular or Herpetic Stomatitis.*

Description and Symptoms.—Some confusion attends the use of this term. It is sometimes confounded with thrush, but it is not a parasitic disease, as is thrush; nor is it herpetic or vesicular, as it is sometimes called; nor follicular. The little greyish-white spots which characterize it consist primarily of an exudate of fibrin and wandered-out leucocytes, which pervades the superficial layer of the mucous membrane and is at first covered by epithelium. Hence an attempt to remove the spots by forceps is futile and followed by bleeding. They are small, round, usually not more than a few millimeters in diameter, and surrounded by a red areola of hyperæmia. They are most common on the cheeks and lips, especially in the gingival groove at the base of the latter. They also occur on the tip and edges of the tongue, more rarely on the dorsum. The epithelium dies and desquamates, leaving a superficial ulcer, which under favorable circumstances heals up rapidly. Under more unfavorable conditions the ulcer grows deeper and becomes more painful, constituting one of the forms of ulcerative stomatitis. Young children are especially subject to it, but it is common also in adults, especially at times of temporary physical depression, as in women during menstruation, pregnancy, and lactation.

The aphthæ are commonly associated with a variable amount of simple stomatitis, a slight "heaviness" of the breath without fetor, increased secretion, and a stinging sensation, especially when brought in contact with food and even when the tongue and lips are moved in speaking. There is often a slight degree of fever.

Ulcerative Stomatitis.

Synonyms.—*Stomacace; Fetid Stomatitis; Putrid Sore Mouth.*

Definition.—This is a much more serious disease of the mucous membrane of the mouth, attended with necrosis of the mucous membrane and resulting ulceration.

Etiology.—It is also a disease of children, but is not limited to them and may be epidemic in public institutions. It may begin as an aphthous stomatitis, taking on the more serious form in the illy fed and badly cared for, or in those who are indifferent in the care of their mouths. In these, an abrasion or laceration due to any cause, as the tooth-brush or a sharp carious tooth, may be the initial lesion. It is not unlikely, too, that an ulcer may begin in a herpetic vesicle, which, on rupturing, leaves an ulcer that may remain isolated or unite with others. It is a frequent attendant of mercurialization—*mercurial stomatitis.* The ulcer sometimes starts in the mucous follicles of the mouth. In all these cases the stomatitis is probably the result of infection by some organism as yet not isolated; it may be the omnipresent streptococcus or staphylococcus, to which the sound mucous membrane in health is invulnerable, but which finds a nidus in the abrasions and conditions referred to.

Symptoms.—The ulcers may occur in any of the situations already

named, the lips, cheeks, and, more rarely, the tongue. They vary in size, but are usually of an ashen-grey color, with red areolæ, and often exhibit a tendency to bleed.

A form of ulceration is described by Parrot as occurring in newborn children, consisting of small, symmetrically placed ulcers on both sides of the median line. Bednar's aphthæ are similar, though not regarded as identical. This variety is thought to be traumatic in origin, at least in most cases, either the result of pressure of an artificial nipple against the hard palate, or undue pressure in washing the mouth. Both are described as unusually harmless, but in poorly cared for children may be converted into extensive and deep ulcers. Especially is this the case in the form described by Parrot.

Additional symptoms are profuse *secretion*, exquisite *pain* and *tenderness* in the ulcers and vicinity, a *fetid odor* of the breath, which sometimes pervades the apartment. The *gums* become spongy and, in extreme cases, the *teeth* are loosened. There are proportionate constitutional disturbances, *fever* and often *swelling* of the glands at the angle of the jaw.

With reference to *mercurial stomatitis*, or mercurial ptyalism, mentioned above, this condition is due to mercury administered as a medicine or absorbed in the course of occupations in which mercury is handled. Acquired in the former way, ptyalism, at the present day, is usually accidental rather than designed, in persons exhibiting a peculiar susceptibility. In such persons even fractional doses frequently repeated sometimes produce salivation in a day or two. The symptom first observed is usually *fetor of the breath*, unless the patient be closely watched during the administration of the drug, when a tenderness may be ascertained on closing the jaws with some force. Examination will then discover a swelling of the gums about the teeth. Or a metallic taste may make its appearance as the first symptom. To these symptoms *salivation* is soon added, and becomes more or less profuse according to the severity of the poisoning. In severe cases, the entire mucous membrane of the mouth becomes swollen, as does also the tongue. In such cases, also, ulceration and loosening of the teeth take place. This form of stomatitis was not infrequent in the older treatment of syphilis, which used to fill a hospital ward with a sickening fetor at once recognizable. Actual loss of teeth was, perhaps, less common than is supposed even in those days, yet necrosis of the jaw has, in rare instances, resulted.

Syphilitic stomatitis is also ulcerative, and the ulcers exhibit the same grey color. But the syphilitic ulcers are found in the throat as well as on the gums and cheeks and in the angles of the mouth. They are less disposed to bleed than those of ulcerative stomatitis, and are really less angry looking, but penetrate to greater depth.

Diagnosis.—Scurvy, though a general disease, happily rare of late, is characterized by local symptoms about the mouth, which include ulceration. There are swelling and bleeding of the gums, which rise up around the teeth. The latter become loosened and ulceration may extend even to the lips and cheeks. The tongue and fauces are not invaded by ulcers, but are subject to ecchymoses. Salivation and fetor of the breath are also symptoms, though less decided than in severe ulcerative stomatitis.

On the other hand, in extreme cases deep-seated gangrenous processes are met with. Along with these are, however, the general symptoms of scurvy, by which it is commonly easily recognized.

Mycotic Stomatitis.

Synonyms.—*Parasitic Stomatitis; Thrush; Soor; Miguet.*

Definition.—This is characterized by greyish-white deposits in the buccal and pharyngeal mucous membranes, due to the development and interpenetration of the epithelium by a fungus variously known as *oidium albicans* or *saccharomyces albicans.* It is a variety of yeast fungus made up of branching filaments, at the ends of which oval cells develop. It forms minute white and yellowish spots scattered copiously over the palate, tongue, and cheeks, uniting at times to form larger areas. It may extend into the œsophagus and even larynx. Stenoses of the œsophagus have resulted from its accumulation. The little areas are commonly surrounded by an inflammatory areola and may be scraped off, though with some difficulty, leaving the mucous membrane sometimes intact and sometimes slightly excoriated and bleeding. Thrush is chiefly a disease of nursing children, and is favored by feeble and dyscrasic states, by the want of cleanliness, especially in the care of nursing bottles and nipples where children are brought up on the bottle. Thrush is often unattended by other symptoms, though the mouth may be sensitive and painful. There should be no difficulty in diagnosis, the microscope at once removing all doubt.

Other Varieties of Stomatitis Due to Fungi.—The mouth is a favorite seat for the development of fungi, because of the warmth, moisture, and organic matters constantly present. Though ordinarily harmless, in certain states of the system they may play an important rôle in producing ulcerative stomatitis, as already suggested. Especially worthy of mention are the pneumonia bacteria of Fränkel and the pneumonia-bacillus of Friedlander; also the delicate, thread-like leptothrix buccalis thought to exert a significant part in the production of caries.

Treatment of Different Forms of Stomatitis.

The simplest forms of stomatitis usually get well of themselves without active treatment, while the severe forms and degrees are so similar that they may be considered once for all. In adults as well as in children a proper prophylaxis will greatly diminish them. After every meal of the adult, when possible, and after every feeding of the infant, the mouth should be thoroughly cleansed. For children a saturated solution of boric acid is at once the most convenient and best. Saturated solution of chlorate of potash is suitable for young and old. The mouths of infants should be washed out with a soft sponge wet with either of these solutions. For adults listerin, diluted with twice as much water, is an elegant and efficient wash. Equal parts of phenol-sodique and water are also efficient. Compounds similar to listerin and

much cheaper may be made up. The following is one known as *spiritus thymol comp.* in the Dispensary of the University of Pennsylvania :—

℞. Acid. Benzoic,
Pulv. Sodii Borat., ... āā . . . . . gr. 64
Acid. Boracic, ... gr. 128
Thymol, ... gr. 20
Eucalyptol, ... ♏ 5
Ol. Gaultheriæ, ... ♏ 5
Ol. Mentha. Pip., ... ♏ 3
Ol. Thymii, ... ♏ 1
Spts. Vini Rect., ... ℥ iij
Aquæ Distillat., ... q. s. . . . . ℥ xiiv.
Mix and filter, and color with fluid extract of Hydrastis.

Any of these substances may be used on the tooth-brush as a simple mouth-wash. The tincture of myrrh, a teaspoonful to four ounces of water, should not be forgotten, and though less agreeable, carbolic acid may be used in the same proportion. Permanganate of potassium in the shape of Condy's fluid, a teaspoonful to a tumbler of water, is an excellent wash.

If stomatitis is established, cleanliness is no less important and may be secured by the same antiseptic measures. In addition, the mouths of children may be treated with honey and borax, the mel boracis of the pharmacopœia, to which alum may be added. Alum itself is an admirable astringent, too much overlooked of late. A moderately strong solution may be made, 30 grains (1.95 gm.) to the ounce (31.1 gm.), or the powdered alum itself may be applied to the aphthous (sore) mouth.

For the painful aphthous ulcers of adults there is really nothing so efficient as touching with a pointed piece of nitrate of silver. A single application will often suffice, but where healing does not follow it may be made daily. A very good application also is a solution of equal parts of tincture of the chloride of iron and glycerine, applied to the ulcers with the brush. Chlorate of potassium in saturated solution is also a very good mouth wash, to eight ounces (240 c. c.) of which ½ drachm to a drachm (two gm. to four gm.) of tincture of the chloride of iron may be added.

General treatment should not be overlooked. Many persons the subjects of stomatitis are much run down, and require iron, quinine, and strychnine, with nutritious food, to build them up. Attention should also be paid to the bowels.

The management of mercurial stomatitis is in no way different from that of other forms. Astringents and disinfectants are especially indicated. It goes without saying that the administration of the drug itself must cease.

Cancrum Oris.

Synonyms.—*Gangrenous Stomatitis; Water Cancer; Noma.*

Definition.—A rare disease, characterized by hard infiltration of the cheek near the angle of the mouth, succeeded by rapid gangrene proceeding outward and inward from the central focus until the cheek is perforated, followed by separation of the gangrenous mass. It may start

in the gums and produce necrosis of the jaws. It is confined to one side of the face.

Symptoms.—It occurs in girls and boys from two to five years old, affecting more of the former than of the latter. It is usually confined to the badly fed and those surrounded by unsanitary conditions, especially when convalescent from infectious fevers, one-half of all cases having arisen during convalescence from measles, others after scarlet and typhoid fevers. It may, however, be primary, and rarely affects adults. A parasitic origin seems likely, while damp regions seem to favor it. Its approach is insidious, and it is generally well advanced when discovered. In its extreme severity it may involve the bones of both jaws, the eyelids, and ears; but in its mildest form its result are limited to perforation of the cheek. The dead tissue comes away in dark, offensive shreds.

The constitutional disturbance corresponds to the degree of local involvement, there being high *fever*, reaching often 104° F. (40° C.), with frequent pulse and rapid exhaustion. The adjacent lymphatics are swollen. Inhalation pneumonia of corresponding virulence often succeeds, while intense irritation of the stomach and bowels follows the swallowing of the ichorous discharge.

Diagnosis.—Noma has rarely to be discriminated from anything else. Malignant pustule is less local in its invasion, furnishes the history of contagion, is even more severe in its constitutional effects, and exhibits the appropriate bacillus. Very bad cases of ulcerative stomatitis sometimes suggest cancrum oris, but the devastation is not so rapid nor is there such a tendency to invasion of the external integument.

Prognosis.—This is almost invariably fatal at the end of three or four days, only the promptest and most energetic treatment occasionally saving life.

Treatment.—This consists in the prompt use of the glowing cautery, Paquelin's being sufficient. In its absence cauterization with strong nitric acid may be substituted. Local antiseptic treatment should be carried out in the most thorough manner, syringing with antiseptics being most efficient, while stimulating and nourishing food should be administered.

Glossitis.

Inflammation of the substance of the tongue, or parenchymatous glossitis, is a rare disease, but occurs as the result of violent injury to the organ, as by accidental biting or poisonous stings. Apparently idiopathic inflammations are probably the result of concealed causes of the kind described.

Symptoms.—The tongue is enormously *swollen* and *painful* and sometimes extruded from the mouth. There is great difficulty in speech, mastication, and deglutition, and in extreme degrees these are scarcely possible. The discomfort is almost indescribable, and there may even be *obstruction to breathing*. If exposed, the tongue becomes dry and cracked. There may be suppuration. There is *fever* corresponding to the amount of local disturbance.

Treatment.—This consists in the constant application of ice, of frequent antiseptic cleansing of the mouth, and sometimes of scarification.

Evidence of the presence of pus must be followed by the prompt use of the lancet.

Glossitis Dessicans.

A chronic affection of the tongue, characterized by deep fissures and indentations, giving it an uneven, ragged appearance. Associated therewith are excoriations and occasionally superficial ulcers. Severe pain is caused by contact of acids and even the usual food. Its etiology is not known, but it is sometimes due to gastro-intestinal derangements.

Treatment.—This should be directed to the cause, if it can be discovered. Washes of chlorate of potash should be employed, and if there are ulcers they should be touched with solid silver nitrate.

Lingual Psoriasis.

Synonyms.—*Eczema of the Tongue; Geographical Tongue.*

Definition and Symptoms.—A localized superficial hyperplasia and desquamation of the epithelium of the tongue, sometimes associated with similar spots in the cheeks and lips. The central parts tend to heal, while the periphery spreads. The patches fuse and extensive areas are formed, bounded with sinuous outlines. The appearance has been compared to that of a map—*lingua geographica.* The condition is chronic, sometimes lasts years, but does not usually cause inconvenience, though often a cause of great anxiety. It is occasionally mistaken for syphilitic disease. It requires no treatment.

Leucoplacia.

Synonyms.—*Ichthyosis linguali's; Buccal Psoriasis; Keratosis mucosæ oris; Smoker's Tongue.*

Definition and Symptoms.—A condition in which there are intense white spots on the mucous membranes of the mouth and tongue, consisting in thickened epidermis. They are also sometimes mistaken for syphilitic plaques. The spots on the sides of the tongue are often notched, giving them a scar-like appearance. Those on the inner surface of the cheek are simply flat, tabular swellings. They disappear to be replaced by others; they rarely give rise to inconvenience. Sometimes those on the sides of the tongue become ulcerated, when they are painful if brought into contact with irritants. They have been ascribed to smoking, and, though acknowledged to be of non-syphilitic nature, it is said they occur in those who have had syphilis. They occur in adults of both sexes. They sometimes become papillomatous, and are said to have been the starting-point of true epithelioma.

Treatment.—They are harmless and require no treatment unless ulcerated, when the usual stimulating measures for healing the ulcers may be applied. Should they develop into papillomatous or epitheliomatous structures, they should be operated on by the surgeon.

Mucous Patches.

The true mucous patches or flat condylomata are opaque, white, flat tabular swellings on the lips, tonsils, tongue, and arches of the palate, and especially at the border line between skin and mucous membrane. They consist of an irregular imbricated thickening of the superficial layers of the skin; the cells are swollen and papillæ of the mucous corium hypertrophied.

Treatment.—The treatment of the mucous patches of syphilis is that of syphilis constitutionally, and locally by applications of nitrate of silver.

DISEASES OF THE SALIVARY GLANDS.

FUNCTIONAL DERANGEMENTS.

Ptyalism, or *excessive secretion* of saliva, which is a symptom of mercurial poisoning, also of poisoning by gold, copper, and iodine. Some persons are very susceptible to iodine, so that a few grains of iodide of potassium will cause intense salivation, with pain in the salivary glands. Vegetable substances producing the same effect are jaborandi, muscarin, tobacco. Indeed, almost anything which admits of constant chewing without solution or destruction produces salivation. This is the mechanism of the various agents used in the disgusting practice of chewing gum.

Xerostomia, or *dry mouth*, is the opposite condition of arrest of salivary and buccal secretion, not due to fever,—a rare condition, first described by Jonathan Hutchinson. As a consequence the tongue and mucous membrane are red, dry, and shining. It is more common in women, in whom it follows intense emotion, such as fright, or is associated with hysteria and hypochondriasis. It is probably a neurosis, the result of some cause operating on the centre which controls the secretion of saliva and other buccal glands.

Treatment.—The treatment of ptyalism and xerostomia is that of the conditions producing them.

INFLAMMATION OF THE SALIVARY GLANDS.

Acute Parotitis, or Parotid Bubo.—Apart from mumps, or specific parotitis, considered under infectious diseases, in which any or all of the salivary glands may be involved, the parotid is subject to inflammation from the following causes:—

(1) In the course of infectious diseases, especially typhoid fever, but also scarlet fever, typhus fever, pneumonia, pyæmia, and secondary syphilis.

(2) In connection with diseases or injury of organs in the abdomen or pelvis, including the alimentary canal, urinary tract, abdominal wall, peritoneum, pelvic cellular tissue or genital organs—a very interesting

group of cases which have been especially studied by Stephen Paget. Sometimes simple transient irritation, such as a blow on the testis or the introduction of a pessary, may produce it.

(3) In association with facial neuritis. A fatal case apparently of such origin has been reported by Gowers.

In (1) and (2) septic infection is doubtless the cause of the inflammation, which is often intense, going on to suppuration in more than one-half of the cases. Its possible origin through the duct of Steno was considered in treating typhoid fever. In (3) there is probably some vaso-motor disturbance which is responsible.

Treatment.—This should consist, at first, in attempts to allay the inflammation by leeches and the application of cold, especially ice. Failing in this, fomentations should be applied, while the lancet should be used at the first indication of suppuration.

CHRONIC PAROTITIS sometimes succeeds on acute inflammation, as that of mumps; also on mercurialization or lead poisoning, syphilis, and Bright's disease. I have a case now under observation which has arisen gradually without apparent cause. It may be painful or tender or painless.

ANGINA LUDOVICI.

SYNONYMS.—*Ludwig's Angina; Cellulitis of the Neck; Cynanche gangrænosa.*

Definition and Symptoms.—An inflammation of the floor of the mouth, beginning in the submaxillary gland. It occurs first on one side as a secondary inflammation in the specific fevers, including, especially, typhoid, diphtheria, and scarlet fever, but it may also be primary. It is probably a streptococcus infection. It spreads rapidly over the floor of the mouth and the anterior surface of the throat, sometimes invading the glottis by œdema, and sometimes terminating in sloughing of the soft parts—*cynanche gangrænosa.* Or it may go on to abscess, pointing externally or internally. More rarely, resolution takes place.

Further Symptoms.—The symptoms are *swelling* and extreme *pain*, first in the neighborhood of the submaxillary gland, increased by chewing, swallowing, and talking. The swelling may produce compression of the larynx with resulting dyspnœa, which is suffocative if the glottis becomes involved. Constitutional infection may take place with its grave array of symptoms and fatal termination. There may be remissions and exacerbations.

Treatment.—This should consist in energetic measures calculated to combat the inflammation, such as the use of ice and leeching, but very early surgical interference is likely to be called for.

III. DISEASES OF THE TONSILS AND PHARYNX.

QUINSY.

SYNONYMS.—*Acute Parenchymatous Tonsillitis; Phlegmonous Tonsillitis; Tonsillar Abscess; Cynanche tonsillaris.*

Definition.—An acute inflammation of the substance of the tonsil.

Etiology.—This is a disease of later youth and adults, being rarely met with in children under ten years of age, and not often after forty. Some persons are much disposed to tonsillitis, scarcely a season passing for them without an attack, and sometimes more than one attack. In such, almost every cold terminates in acute tonsillitis. Others, after a single attack, never have another, and others still are entirely exempt. Exposure to wet and cold is by far the most frequent cause. There are those who hold that tonsillitis is always the result of infection. Persons predisposed to tonsillitis are often the subject of chronically enlarged tonsils. Over-distention of the follicles with inspissated secretion may also be a cause of inflammation and suppuration.

Morbid Anatomy.—The tonsil, more frequently on one side, sometimes on both, or on two sides in succession, becomes rapidly enlarged, red, and painful. It is at first hard and resisting and very tender to the touch, but, if suppuration takes place, it gradually softens until rupture happens or the abscess is opened with the knife. The lymphoid parenchyma of the gland becomes more and more distended with leucocytes until the entire gland, or a large part of it, is converted into a *pus* sac. When both tonsils are involved, the throat is often almost closed.

Symptoms.—The superadded symptoms are a *difficulty of deglutition*, attended by pain, which is often agonizing. The jaws are stiff and the mouth cannot be opened above a half of an inch without extreme suffering. The difficulty in opening the mouth is contributed to by the swelling of the external glands of the neck. The pain is not confined to the interior, but extends to the neighborhood of the angle of the jaw, the front of the ear, and the floor of the mouth. The voice is greatly altered, having the characteristic nasal drawl, and the diagnosis can sometimes be made from the altered speech alone. There is increased salivation, and the saliva dribbles from the mouth because of the pain in swallowing it, while it also often becomes fetid. Respiration may be seriously interfered with.

There is high *fever*, the temperature reaching 104° and 105° F. (40° to 40.5° C.), while the *pulse* is full, bounding, and frequent, 110 to 130 in a minute. The *face is anxious* and tells the tale of suffering. From two to six days are occupied in the completion of the process, at the end of which time the gland begins to point, usually toward the interior of the mouth, when relief is obtained by spontaneous rupture. But more fortunate is the patient who is relieved early by the lancet. Sometimes the abscess points toward the pharynx. The importance of relief at the earliest possible date is emphasized by the fact that death by suffocation has resulted from the rupture of a quinsy whose contents have gone into the larynx.

Prognosis.—Apart from the rare accident just referred to, the prognosis is favorable, though it must be mentioned also that death from suffocation has occurred where the obstruction by double-sided quinsy was so great as to prevent respiration.

Treatment.—The physician who suggests a successful plan for "backing" of a quinsy will well deserve the thanks of untold sufferers. As yet such measure remains hidden. Free scarification is sometimes useful in shortening an attack, but it is painful, unreliable, and sometimes difficult to do thoroughly. If deferred to about the third day it will often coöperate with the advancing suppuration and favor an early rupture. Other applications to the tonsils are useless, though some relief from pain may be secured by painting the surface with a five per cent. solution of cocaine. Cold, so soothing in other forms of sore throat, often occasions more discomfort than relief. Then poultices and fomentations to the exterior of the throat are apt to be more soothing. And since nothing can be done to prevent suppuration, these measures are indicated to hasten it. The tonsil should be frequently felt with the finger, and as soon as there is evidence of suppuration the lancet should be used. A curved bistoury, guarded with adhesive plaster almost to the end, is best. The incision should be made from above downward parallel to the anterior half-arch. If danger of suffocation is imminent the tonsil may be shaved off, while extreme cases may even demand tracheotomy.

FOLLICULAR TONSILLITIS.

Synonyms.—*Angina follicularis; Lacunar Tonsillitis.*

Definition and Symptoms.—A form of catarrhal inflammation of one or both tonsils, associated with whitish-yellow spots corresponding in situation with the lacunæ or follicles of the gland. The inflammation may also extend to the soft palate, but the white or yellow spots are the most conspicuous feature. In a day or two they drop out or may be pressed out, when they are found composed of epithelial cells, pus corpuscles, bacteria, and debris, to which are sometimes added cholesterin plates and fat crystals. If left alone, they are apt to disappear rather suddenly, so that if seen one day they may be gone the next, having evidently disintegrated and dropped out spontaneously. More rarely the little follicle is converted into a small abscess. The disease occurs in children and young adults, and is one of the affections which are sometimes mistaken for diphtheria, and is also called diphtheritic sore throat. It is, however, something very different. It is a much less serious disease, of shorter duration, and patients never die of it. It is, however, probably infectious in origin, caused by a germ other than the diphtheritic, perhaps the streptococcus or staphylococcus. There is often very decided fever.

Treatment.—This is that of aphthous tonsillitis, to be next considered.

APHTHOUS TONSILLITIS.

SYNONYMS.—*Necrotic Tonsillitis; Diphtheroid Sore Throat.*

Definition and Symptoms.—I prefer this term for that form of tonsillitis in which, along with a catarrhal inflammation of the tonsils, which may extend also to the half arches and soft palate, one or more greyish-white patches are scattered over the tonsil, well set into the mucous membrane and not detachable, as are the white spots in follicular tonsillitis. It is a condition distinct from follicular tonsillitis, though often confounded with it. The term aphthous is chosen because not only are these patches closely set and undetachable, but they seem to have nearly the same histological composition as the patches in aphthous stomatitis, namely, an infiltration of the superficial layer of the mucous membrane with pus cells and bacteria, forming a layer which becomes necrotic. More rarely they are found on the half arches and the posterior part of the soft palate. Their resemblance to the false membrane of diphtheria is very close, whence this condition, as well as follicular tonsillitis, is also appropriately called *diptheroid sore throat.* But the layer does not strip off as easily even as the false membrane of diphtheria, it does not spread, and, above all, it does not contain the Klebs-Löffler bacillus.

The clinical course differs from that of diphtheria in being very much shorter, though the symptoms are by no means always milder. On the other hand, the *headache* and *backache* and *fever* are often very much more severe than in diphtheria of well-defined type. Hence caution should be observed in diagnoses, and cultures should be immediately made when possible. The patches last longer than the spots in follicular tonsillitis and often when they come off a superficial ulcer remains. Such a patch remains five or six days, but it does not extend throughout the whole time. The constitutional disturbance, though at first considerable, does not increase, and the patient, instead of getting worse, slowly improves. There may even be some enlargement of the lymphatic glands in aphthous and follicular tonsillitis, but it falls far short of what is attained in diphtheria.

Treatment of Follicular and Aphthous Tonsillitis.—The treatment of these forms of tonsillitis is definite and easily carried out. In the first place, cold should be applied to the neck by cloths wrung out in cold water or by ice, which is conveniently applied in little muslin bags made to fit under the angle of the jaw and held in place by a bandage. Then iron and chlorate of potassium are, without doubt, the remedies *par excellence*, and to these may be added the bichloride of mercury, if diphtheria is not certainly eliminated from the diagnosis. The antiseptic measures recommended for the throat in diphtheria are not necessary. The disease is an acute one and subsides rapidly without any of these applications. There is, however, a very decided drain on the strength of the patients thus affected, however short the duration of the illness. Hence quinine and iron should be given and continued during convalescence.

CHRONIC TONSILLITIS AND HYPERTROPHY OF THE ADENOID TISSUE OF THE PHARYNX.

SYNONYMS.—*Chronic Enlargement of the Tonsils; Chronic Naso-pharyngeal Obstruction; Mouth Breathing; Aprosexia.*

Definition.—A chronic inflammatory enlargement of the tonsils or of the adenoid tissue of the pharynx, of the lingual tonsil, or of two or more of these structures.

Etiology.—The most frequent cause is repeated attacks of acute tonsillitis and of inflammatory processes associated with hyperæmia of the tonsils and vicinity, including scarlet fever and diphtheria, while chronic illness, especially skin affections, bad hygienic and insufficient and unsuitable food favor it. It is, therefore, naturally more common in children, in whom it is also sometimes congenital, but it is found usually at the ages of five to fifteen years, and rather more frequently in boys. Adenoid overgrowths of the pharynx and lingual tonsil are due to the same causes.

Morbid Anatomy.—The enlargement of the tonsils is a true lymphoid overgrowth, usually symmetrical. The occasional presence of fibrous stroma produces a harder and smoother tissue. The lumen of the throat is variously encroached upon, sometimes almost closed. The pharyngeal adenoid overgrowths vary in extent from a slight increase in natural unevenness to the formation of actual sessile and pedunculated tumors. The same is true of the tonsillar structures at the base of the tongue, which may encroach upon the glottis.

Symptoms.—Simple chronic enlargement of the tonsils may give rise to no symptoms except when the seat of further enlargement due to acute inflammation. Then *obstructed breathing* is immediate, while it is also a permanent symptom in the more advanced forms. It is proportionally contributed to by overgrowth in any of the situations named. The result is *mouth breathing*, which is, perhaps, earlier necessitated by pharyngeal than tonsillar overgrowth, while it may be due altogether to the former, the latter being entirely absent. Tonsillar obstruction is, however, more frequent. The effects are usually first apparent at night, when the child is found to be breathing, more or less noisily, with its mouth open and head thrown back. *Disturbed rest* is an inevitable consequence, the patient often waking up with a start, after which it may again relapse into sleep or continue permanently aroused because of the dyspnœa, which often only gradually passes away.

As the conditions persist a *changed expression of countenance* is gradually acquired. The face becomes apathetic, staring, and vacant, an appearance chiefly produced by the constantly open mouth. To this succeed actual mental failure and even stupidity, with sullenness and general bad temper. Further changes in expression are occasioned by contraction of the nostrils and projection of the upper jaw and lip. If the condition is still unrelieved, *deformities of the chest* make their appearance, of which the most conspicuous is the well-known *chicken breast.* In it the upper sternum projects, the manubrio-gladiolar articulation being most prominent, while the lower part is depressed, causing a groove at

the gladiolo-xiphoid articulation. There is a cup-like depression of the lower costal cartilages and a horizontal circular depression in the thorax corresponding to the attachment of the diaphragm. The ribs are separated from each other anteriorly and closely approximate posteriorly, especially in the lower thorax. Posteriorly the lower angle of the scapula projects.

Other symptoms are an *altered nasal voice* in which the letters *m* and *n* are especially badly articulated, the senses of smell, taste, and hearing are deranged, the breath is fetid from decaying secretion, the appetite is impaired, and with it the nutrition of the body. A gradual mental as well as physical deterioration takes place.

Among the symptoms ascribed to this condition are *habit chorea* and *stuttering*. The former will be considered in a later section. There is an almost *constant cough*, which is well termed "throat cough," since it is due to irritation of the respiratory passages by the throat outgrowths and the secretion caused by them. This secretion is generally swallowed by children, but is in part expectorated by adults by the aid of troublesome hawking and coughing, which is stimulated by a sensation as of "something in the throat" or larynx which demands clearing. The absence of discharge from the nose in both children and adults is surprisingly frequent, sometimes misleading the physician as to the true cause.

Defective hearing is another symptom due to obstruction of the Eustachian tube by encroachment of the adenoid growths, or by inflammations, or to retraction of the drum. *Impaired taste* and *smell* are due to involvement of the gustatory papillæ and the terminal distribution of the olfactory nerve. *Extreme fetor* of the breath is sometimes present, due to the retention of cheesy masses in the crypts of the tonsils. These are often easily visible, are sometimes expectorated, and can usually be expressed. The odor of these masses when compressed between the fingers is indescribably disagreeable. Sometimes they are found in the tonsils of persons not otherwise affected. The very great susceptibility of the subjects of this disease to "cold" is constantly adding aggravation to the symptoms described.

Diagnosis.—This is not usually delayed at the present day, since the more thorough examination of the throat and nose has become common—thanks to the throat and nose specialists. Most important is to remember that there may be no tonsillar disease, and all the symptoms may be due to advanced adenoid growths of the pharynx. Digital examination by the finger affords the most ready and accurate means of diagnosis. Especially thorough must be the examination behind the pillars of the fauces. In children this can only be done with the finger, but in adults the half arches may be drawn forward, while the laryngeal mirror is availed of.

The "chicken breast" of mouth breathing in childhood is different from the "violin" shaped chest of the rickety child. In the latter there is a *prominence* of the *whole sternum* and a vertical flattening of the sides of the thorax, leaving a large curve behind the costo-chondral articulation and a similar one in front, in addition to the horizontal depression of the lower thorax which is common to both kinds of deformity.

Prognosis.—This depends upon the early discovery of the condition, before the secondary effects have established themselves. If the trouble is purely a tonsillar one it is comparatively easily removed by shaving off the organ. If the overgrowth is pharyngeal little can be done until children are old enough to submit to the proper treatment. Hypertrophied tonsils begin to atrophy of themselves after puberty, and they have generally disappeared by thirty. The face and chest deformity may be outgrown if the cause is removed.

Treatment.—Most important are local measures to reduce the size of the overgrowth or to remove them and to prevent recurrence of acute attacks. The patient should be discouraged from hawking and clearing his throat. If the tonsils manifestly encroach on the faucial lumen they should be shaved off by the guillotine or a bistoury or galvano-cautery loop. The same treatment is demanded by the pharyngeal adenoid growths. They may be curetted and sometimes scraped off by the finger-nail. There is sometimes copious hemorrhage, but it is usually easily controlled. If not requiring this they should receive on alternate days or every third day applications of powdered alum, solution of iodine of the strength of iodine eight grains (0.5 gm.), iodide of potassium 24 grains (1.5 gm.), glycerine half ounce (30 c. c.); of tincture of the chloride of iron and glycerine equal parts, glycerole of tannin, or silver nitrate one to 20. The solid stick of the latter may be used if there is evident lacunar disease, but far better is electrolysis, by which the crypt is obliterated and the gland may be gradually destroyed. Great patience and perseverance are required, for the result is but slowly attained.

The general health of the patient should be carefully looked after. Suitable woolen under-clothing should be worn, and it should be graduated to temperature and exposure. Cod-liver oil, iron, quinine, and strychnine are the best roborants. It is most important that every effort should be made in the direction of so hardening the patient that he may be able to resist the effects of exposure, a task not easy to accomplish. Cold bathing of the neck and throat, indeed, of the whole body, is useful, while nourishing food, physical exercise, and out-door life, with suitable dressing, are means to this end.

SIMPLE CIRCULATORY DERANGEMENTS OF THE PHARYNX.

Hyperæmia of the pharynx is a very common condition in smokers. It is also almost always present when there is chronic nasal catarrh. Under these circumstances the mucous membrane is constantly red, angry looking, often streaked with mucus, and is very easily thrown into a state of active inflammation.

In such obstructions to the circulation as are caused by mitral valvular disease, cirrhosis of the liver, or pressure upon the ascending vena cava by aneurism or tumor, there is venus stasis and the venules may often be seen distended. Occasionally they burst, producing small hemorrhages which stain the mucous secretion. The same causes may produce œdema of the mucous membrane of the pharynx, but especially does this occur in Bright's disease. The œdema may extend thence to

the uvula, which becomes greatly swollen. In aortic regurgitation the capillary pulse may be seen in the pharynx, and the internal carotid also seen to throb strongly.

ACUTE CATARRHAL PHARYNGITIS.

SYNONYMS.—*Sore Throat; Simple Angina.*

Definition.—An acute inflammation of the mucous membrane covering the pharynx and tonsils, sometimes extending upon the palate.

Etiology.—Acute pharyngitis occurs at all ages, but is more frequent in children. Exposure to cold and wet is its most frequent exciting cause. The delicate are more predisposed than the robust, and where there is the hyperæmia above referred to, a trifling cause lights up an inflammation. Rheumatism and gout are also frequent causes.

Symptoms.—The first symptom is usually *pain* on swallowing, which is associated at first with a *dryness* and *soreness*, producing a desire to "clear the throat." To this is soon added a *full feeling*, and then pain independent of swallowing. The inflammation may extend into the Eustachian tube, producing partial *deafness*, or into the larynx, producing *hoarseness*. There is a varying degree of constitutional disturbance, and sometimes the *fever* is quite high.

On examining the throat it will be found red and congested, sometimes plainly swollen, especially over the tonsils. There is often considerable mucous secretion. The various forms of ulcer of the tonsils described under tonsillitis may be associated with the pharyngitis, increasing the constitutional disturbance and local discomfort.

Treatment.—Many simple sore throats pass away without treatment. Astringent washes and gargles are indicated, but the patient should be warmly housed and even in mild cases should go to bed. Twenty-four hours in bed is by far the best medicine for an ordinary cold. A gargle of alum or tincture of the chloride of iron in the proportion of a teaspoonful of either to a full tumbler of water may be used, while applications of a mixture of equal parts of the iron tincture and glycerine may be applied to the throat two or three times a day. Solution of nitrate of silver, 20 grains (1.3 gm.) to the ounce (30 c. c.), may be similarly applied, also the glycerole of tannin.

In severe cases, cold cloths wrung out in ice water and applied to the outside of the throat, the clothing being protected by the interposition of a dry towel, make an excellent measure; or the little ice bags referred to in the treatment of acute tonsillitis may be applied to the throat, with a dry towel outside of this. Occasionally counter-irritation by mustard is more efficient, as every throat does not bear cold equally well.

The fever should be met in the usual way by aconite, sweet spirit of nitre, and citrate of potash, while chlorate of potash and chloride of iron should also be administered internally. The bichloride of mercury may be added under the same circumstances as in diphtheroid tonsillitis. There is no advantage in giving large doses of iron. They are not absorbed and the excess remaining in the alimentary canal locks up the

secretions and irritates. From two to ten minims (0.12 to 0.6 gm.) every two hours are quite sufficient. The bowels should be kept open and the treatment may be advantageously commenced with a saline aperient, such as the solution of the citrate of magnesium, calcined magnesia, or Hunyadi water.

Where the disease is traceable to rheumatism or gout, suitable treatment for these diseases should be instituted. The salicylates are the best remedies for both, but guaiacum has some reputation, the tincture or ammoniated tincture being the best preparation.

CHRONIC CATARRHAL PHARYNGITIS.

SYNONYMS.—*Clergyman's Sore Throat; Granular Pharyngitis; Chronic Angina; Chronic Follicular Pharyngitis.*

Definition.—Chronic pharyngitis, when not associated with ulceration, presents much the same appearance as chronic hyperæmia, plus the addition of a granular appearance due to enlargement of lymphatic glandules, with which the pharynx is studded.

Etiology.—The disease is rather one of adults than children. Its causes are repeated attacks of acute pharyngitis and excessive smoking and alcohol drinking. Chronic nasal catarrh with its irritating discharges trickling down the fauces is a frequent cause, as is also disease of the third, or Luschka's tonsil. It also occurs in those who use their voices largely, as hucksters, public speakers, and singers, while the inhalation of dust and irritating gases is also held responsible.

Prognosis and Treatment.—This is very much more unsatisfactory than in the acute type. It is most important to treat the causes or remove them. Post-nasal catarrh is responsible for so many cases that this region should at once be investigated and its diseases treated. Smoking and the use of alcohol, if responsible, should at once be discontinued. The same local measures useful in the acute disease may be employed in the chronic, but they are less promising as to results. The little granules, which are apparently a source of irritation as well as a result, can only be removed by the galvano-cautery needle. Other measures to this end are unsatisfactory and insufficient. The general health of the patient should be carefully looked after, and occupations tending to keep up the irritation should be discontinued.

ULCERATION OF THE PHARYNX.

The ordinary form of chronic pharyngitis rarely produces ulceration. Syphilis, tuberculosis, diphtheria, inflammation, and lowered nutrition, such as is found after the infectious diseases, like typhoid fever and scarlet fever, are frequent causes of sluggish ulcers indisposed to heal. The chief symptom of these various varieties of ulceration is pain, increased during deglutition, with more or less copious mucous secretion, which often adheres firmly to the pharynx.

Diagnosis.—It is not always easy to distinguish the different forms of ulceration. The syphilitic ulcer is least painful, in fact, often pain-

less, and is commonly situated in the posterior wall of the pharynx. It occurs both as a secondary and tertiary symptom. As a secondary symptom it is superficial and associated with mucous patches, while as a tertiary it forms the cavity left by a softened, gummy tumor and is correspondingly deep. It is associated with the history of syphilis.

The tubercular ulcer is more painful—indeed, the most painful of all. It is irregular, not very deep, has a greyish base, and is also seated in the posterior wall of the pharynx, considerable areas of which may be involved, producing an uneven, worm-eaten appearance. It is associated with the presence of tuberculosis elsewhere. The ulcers of lowered nutrition are also often insidious and occasion few active symptoms. After the separation of the membrane in diphtheritic pharyngitis there are sometimes left ulcers more or less extensive, which are slow to heal.

Treatment.—This consists locally in the application of stimuli and antiseptics, the former represented by nitrate of silver and the latter by thymol and its class, together with general treatment appropriate to the condition, such as tonics of which iron and quinine are the types.

Phlegmonous Pharyngitis.

Definition.—This term is applied to any suppurating inflammation involving the pharynx, however induced, except post-pharyngeal abscess, which is a separate condition. It may be a part of the process which constitutes suppurating tonsillitis or quinsy extending to the adjacent pharyngeal structures. It may include the acute infectious phlegmon of the pharynx described by Senator, in which, along with swelling of the external neck, the pharyngeal mucous membrane is swollen and injected and becomes rapidly the seat of suppuration. It may include similar conditions induced by injury, the inhalation of scalding liquids, or the swallowing of corrosive poisons. Or it may be the result of pharyngeal erysipelas or of the lodgment of foreign bodies.

Symptoms.—These are correspondingly intense. There is *painful swelling*, interfering not only with deglutition but also with respiration. There is high *fever* and rapid *exhaustion*. It may terminate in *gangrene* of the part or gangrenous pharyngitis.

Treatment.—The treatment is locally antiphlogistic, including cold by ice or otherwise, scarification and liberation of pus at the earliest possible moment, together with restorative and stimulating internal measures. If gangrene results, cauterization and antiseptic applications must be added. The aid of the surgeon should be early sought.

Post-Pharyngeal Abscess.

Definition.—A phlegmonous inflammation behind the proper pharyngeal tissues, subperiosteal in some instances, arising in suppurative inflammation of the post-pharyngeal lymphatics or caries of the cervical vertebræ. It is a disease of children and adults, more frequently of the former, often a sequel of one of the pharyngeal conditions already considered, favored by bad hygiene and depraved constitutional states, hereditary or acquired.

Symptoms.—Its symptoms are intense *pain*, *swelling*, and interfer-

ence with deglutition and respiration, with more or less early appearance of a tumor in the posterior wall of the pharynx which can generally be recognized by the finger before it can be seen,—a fact which emphasizes the importance of frequent examination of the throat by the finger in diseases of these parts. There is also *stiffness of the neck*, sometimes *nasal voice* or even hoarseness, suggesting croup and œdema of the glottis, but there is never absolute loss of the voice, as in the latter, while croup and œdema are not associated with painful deglutition.

Treatment.—This consists of incision of the abscess as soon as discovered. It should be made in the median line and the head should be brought forward to avoid the entrance of pus into the larynx. Anodynes are necessary to overcome the intense pain, but it is to be remembered that they may so mask the symptoms as to permit destructive inroads of the disease before it is discovered.

DISEASES OF THE ŒSOPHAGUS.

EXPLORATION OF THE ŒSOPHAGUS WITH THE BOUGIE.

This is a manipulation so frequently necessary that its description is demanded at the outset.

The œsophageal bougie is made of flexible whalebone, on the end of which is firmly fixed an olive-shaped piece of ivory. The ivory ends are made of different sizes. The ordinary stomach tube may also be used for the same purpose, and is the safest instrument to use in earlier exploration.

In introducing the bougie, or tube, the patient should sit on a low chair with his head thrown back. The index finger of the left hand is then introduced well back into the pharynx, along the median line. The bougie, or tube, is then passed along the side of the finger to the posterior wall of the pharynx and then down into the gullet. Usually a slight resistance is encountered at the level of the cricoid cartilage, but it is easily passed, and after this the descent is easy. Caution should, however, always be exercised, as the bougie has a few times been pushed through an ulcer of the œsophagus into the pleural cavity or lung, while I have also known ulceration to be produced by the repeated use of the bougie in simple nervous spasmodic obstruction.

ŒSOPHAGITIS.

Acute Œsophagitis.—An acute inflammation of the œsophagus is practically limited to inflammation induced by the swallowing of very hot or corrosive liquids, such as strong acids and alkalies, or by the lodgment of foreign bodies. It is true, diphtheritic inflammation sometimes extends from the pharynx downward, while the œsophagus has also been invaded by a vesicle of small-pox, but these conditions are not

likely to be differentiated from the primary disease. Mycotic œsophagitis, producing stenosis of the œsophagus in sucklings, has been alluded to as a possibility on p. 210.

Morbid Anatomy.—Appearances vary with the cause. In addition to the usual redness, sloughing and disintegration of tissue may result. Milder degrees of inflammation produce less conspicuous alteration. A granular appearance may succeed desquamation of the epithelium. The diphtheritic false membrane presents the same characters here as elsewhere.

Symptoms.—These are chiefly *pain* beneath the sternum, increased by swallowing, which in extreme degrees of inflammation becomes agonizing and, indeed, renders swallowing impossible. Milder grades of inflammation are without symptoms, intermediate grades present corresponding symptoms. If healing results after destructive inflammation the cicatricial tissue behaves as it does everywhere else, contracting and distorting the parts, oftentimes with resulting stenosis.

Treatment.—Little can be done to aid healing. For the most part, therefore, it must be given over to nature. If deglutition is possible, demulcents may be thus used, while the swallowing of pieces of ice sometimes gives comfort. When deglutition is impossible, the patient must be fed with nutritious enemata.

Chronic Catarrhal Œsophagitis.—This affection is a possibility favored by valvular heart disease, cirrhosis of the liver, or other cause of venous obstruction. A hemorrhoidal state of the veins may be thus caused, of which the treatment is that of the cause.

SPASM OF THE ŒSOPHAGUS.

This is not an unusual affection in hysterical women, and even in male hypochondriacs. These are generally past middle life. It also occurs in hydrophobia, chorea, and epilepsy. The spasm is commonly excited by an effort to swallow solid food, and rarely even liquids act similarly. A possible result of spasm is a dilatation, as shown in a case of my own to be again referred to.

Diagnosis.—The diagnosis is readily made by the bougie, which, though it may be stayed for a minute at the seat of spasm, ultimately passes it without the application of force. It is also associated with other symptoms of hypochondriasis, while extreme pain, the gradual emaciation, weakness, and ultimate cachexia of cancer are wanting. Errors of diagnosis have, however, been made, and death has even occurred when autopsy disclosed no lesion to explain it.

Treatment.—This is that of the hypochondriacal state and the frequent use of the bougie, of which the moral effect is also good. One introduction has sometimes been sufficient. On the other hand, I have known the repeated passage of a bougie to have produced ulceration, whence the caution already enjoined in the use of the instrument.

CANCER OF THE ŒSOPHAGUS.

This is usually a hard epithelial tumor most frequent in the middle third of the œsophagus, though it may involve the cardiac orifice of the stomach, and more rarely other portions. E. Rindfleish, especially, describes a sôfter and more superficial form which invades larger areas in a diffuse way. It is rather more frequent in men, and appears first as zonular infiltration of the mucous membrane, which ulcerates. The resulting ulcer may also extend around the tube, acquiring a width of two to three inches (five to six cm.). The primary and usually permanent result, unless ulceration does away with it, is a stenosis of the œsophagus followed by dilatation of the tube, with hypertrophy of the walls above the stenosis.

Symptoms.—*Difficult* and *painful deglutition* is usually the first symptom of stenosis, though *pain*, independent of deglutition, may precede. *Swallowing* becomes more and more difficult, and ultimately, even liquids may be regurgitated. *Regurgitation* may not be immediate, and the date of its appearance is usually dependent on the seat of the obstruction and extent of dilatation above it. Death commonly takes place from exhaustion or actual starvation. But before this happens there may be a rupture into the larynx or a bronchus, producing death by suffocation, by gangrene, or by an inhalation pneumonia. There may be ulceration into the aorta or one of its large branches, causing fatal hemorrhage; into the pericardium, producing fatal pericarditis. Ulceration into the mediastinum or erosion of the cervical vertebræ sometimes occurs with more delayed fatal ending. Emphysema is a sign of rupture into the lung. The adjacent lymphatic glands of the neck are sometimes invaded. Rarely the disease is latent throughout its entire course.

Diagnosis.—This may have to be delayed a short time, but is soon clear. The continued obstruction, the emaciation, and weakness soon distinguish the case from one of spasmodic stenosis. Compression by adjacent growths should be remembered as a source of obstruction, aneurism being perhaps the most frequent cause of this kind; but aneurism may generally be recognized by its other signs.

Prognosis.—This is always, ultimately, fatal.

Treatment.—Treatment can only be made to prolong life. The bougie should not be used after the diagnosis of cancer is established, because of the danger of causing perforation. So long as liquid food can pass the obstruction it should be used, after this nutritious enemata in the manner recommended under cancer of the stomach. Œsophagotomy or gastrostomy may be presented for the patient's consideration. The former promises nothing, but life may be prolonged by the latter.

STRICTURE OF THE ŒSOPHAGUS.

The most frequent causal agent is contraction of the scar tissue of a healed ulcer, caused, commonly, by some corrosive agent or syphilis. Next in frequency is pressure by external tumors, such as aneurism, enlarged lymphatic glands, or mediastinal tumors. Next is congenital narrowing, and, finally, polypoid tumors projecting from the mucous membrane. If the stenosis be cicatricial the precise cause is to be deter-

mined by the history of the case, and its situation by the œsophageal bougie.

Treatment.—This is altogether by the careful use of the bougie. Dilatation of the cicatricial stenoses is often quite successful. The largest bougie should be first introduced very gently, without force, really as a sound, *as far as the obstruction only.* Then smaller sizes should be tried until one is found which will pass, and from this point, again, larger sizes should be successively employed. At each sitting the original size should be started with and followed more rapidly by the larger sizes as the physician becomes familiar with his patient's case.

In congenital cases less is to be expected, while obstruction by external growths is practically irremediable and grows gradually worse. Even cicatricial stenosis may be such that the smallest bougie cannot pass, in which event nourishment by the rectum alone remains unless gastrostomy be decided on.

DILATATION OF THE ŒSOPHAGUS.

Dilatation of the œsophagus may involve the whole circumference of tube, when it is known as *diffuse* or *total;* or it may affect only one spot, when it is circumscribed, or constitutes a *diverticulum.*

Diffuse Dilatation.—In every case of stenosis of the œsophagus from whatever cause, there is sooner or later dilatation above it, delayed at first by hypertrophy of the muscular coat, which is thus enabled to force the food through the narrowing. Sooner or later this coat becomes paralyzed, the wall yields to the pressure of accumulated food, and dilatation follows. The resulting sac is usually spindle-shaped and is naturally larger the lower the seat of obstruction.

Rarely dilatation occurs without previous organic stenosis. It would appear, however, that it must be preceded either by some traumatic cause which weakens the wall of the tube, or by repeated spasmodic stenoses. The fact remains that such dilatations occur.

Diverticula.—Diverticula or circumscribed pouches in the walls of the œsophagus are of two varieties. They have been especially studied by Zenker, who has divided them into *pressure* diverticula and *traction* diverticula according to their mode of origin.

Traction diverticula are the more frequent, yet clinically are of less interest because often not recognized until their subjects are on the necropsy table. They are small, scarcely ever exceeding a centimeter (0.4 in.) in diameter, and relatively frequent in children. They are ascribed to some traction effect exerted on the wall of the œsophagus. This may be, as Rokitansky and Zenker suggested, due to the contraction of a tissue which has formed adhesions to the œsophagus. Such a tissue is afforded by the bronchial glands, which become inflamed, caseate, and contract, and as they are situated at the bifurcation of the trachea, the more frequent occurrence of traction diverticula at this situation in the anterior wall of the gullet is thus explained. Such diverticula may be multiple.

Pressure diverticula are much rarer. They occur almost always in men, rarely in children. They are found most frequently at the junction of the pharynx and œsophagus, where the muscular wall, formed

chiefly by the inferior constrictor of the pharynx, is weakest, and are caused by pressure from within. This may be exerted by the bolus of food itself, especially if it be habitually large, as in rapid eaters, while its operation may be further facilitated by some traumatic injury to this part of the throat, such as may be caused by the lodgment of a bone.

The sac is found to be bounded by mucous membrane and thickened submucous coat, the muscular coat giving way to let the mucous coat pass through it, as in a hernia. It is found invariably in the posterior wall and hangs in front of the spinal column.

Symptoms.—In cases originating in stenosis, apart from the inference that where there is stenosis there must ultimately be dilatation, the first symptom to attract attention is the *feeling* on the part of the patient that his *food does not enter the stomach* but lodges higher up, though the quantity swallowed is evidently more than would be held by an œsophagus of ordinary caliber; usually, sooner or later, follows the *regurgitation*, or gulping up of this accumulation. The same symptoms are said to attend dilatation without stenosis. The latter event can only be explained on the supposition that in consequence of the paralyzed state of the muscular wall of the œsophagus there is no force to push the food down, while the gradual widening of the tube affords support for its lodgment, which is further favored if the enlargement takes the shape of sacculations or a pocket.

Traction diverticulum rarely causes symptoms. Those arising from pressure diverticulum are first those of *dysphagia*, as the diverticulum grows larger, and food lodges more and more; *regurgitation*, though the sac is rarely thoroughly emptied, and the retained food sometimes undergoes decomposition, giving rise to fetid breath. The difficulties increase until after a while it is almost impossible to get food into the stomach, though extraordinary efforts are made by the patient to do so with greater or less success. Complete closure results when the diverticulum becomes so large as to flex upon the gullet and compress it.

The sound should be used in the study of all forms of the disease. By its means the situation of the stenosis can be ascertained. Should it pass readily into the stomach there is no stenosis, but there may still be a diverticulum, for at one sitting it may pass the opening into the sac, while in another it may enter it and resist further attempts to complete the transit. Zenker and v. Leube have devised a diverticulum-sound bent at an angle, so as to facilitate its entrance into the diverticulum, advantage being taken of the fact that we know about where these diverticula are most frequently found, that is, opposite the cricoid cartilage.

With the prolongation of the condition the proper nourishment of the patient becomes more and more difficult, he emaciates, grows weaker, and ultimately perishes from exhaustion unless carried off by some other disease.

The following case recently under my care illustrates the symptoms of the condition: C. G. is an actor thirty-three years old. When only twelve years of age, while eating his supper, his food suddenly regurgitated and he had to leave the table. Returning, another effort was followed by the same result. The next morning his breakfast came up in the same manner. After a time he discovered that by rapidly

drinking a large amount of liquid after his meal he could, by a great effort, cause most of the food to enter the stomach. This has to be done at every meal by some indescribable effort which is painful and exhausting. Furthermore, it is rarely completely successful, some food being always regurgitated, commonly later in the day. Since twelve years of age this regurgitation has continued, and he loses, on the whole, about one-third of the food ingested, while at times his efforts to get it down are totally unsuccessful, in which event the full amount is regurgitated. Further, the difficulty of successfully getting food into the stomach is gradually increasing.

In this case there would appear to be a certain degree of stenosis, for while a part of the food can be forced to enter the stomach, and small sounds can be passed into that organ, larger ones cannot be made to enter. Yet from the suddenness of its occurrence and the early age in the life of the patient, the stenosis, if one is present, has not arisen from the usual causes. Can there be a diverticulum? If so, it is lower than pressure diverticula generally are, and larger than other traction diverticula.

Treatment.—The treatment of diffuse dilatation and diverticula is essentially the same. It consists, first, in measures to maintain the nutrition of the patient. Generally he is able to pass a certain amount of food by his own efforts, of which those detailed in the case of my own patient are an illustration. After this the stomach tube becomes the most ready way. This, too, he should be taught to use himself. Rectal alimentation may help somewhat, but is alone inadequate for any length of time, while the inconvenience of any and all of these procedures renders the patient anxious for more complete relief.

This may be accomplished by operation, by which diverticula have been successfully removed. The difficulties in the way of operation are, however, great. The operative treatment of dilatations due to stenoses resolves itself into that of the stenoses themselves. In both forms gastrostomy may be the ultimate measure which promises relief for a time.

DISEASES OF THE STOMACH.

DIAGNOSTIC TECHNIQUE.

The very great value that modern medicine has discovered in a proper technique for the diagnosis of diseases of the stomach makes its preliminary consideration indispensable to their sufficient and exact study. It is conveniently divided into the internal and external examinations.

External Examination.

This embraces inspection, palpation, percussion, and succussion or splashing. Because of the difficulty of separating the external examination of the stomach from that of the intestines they are usually considered jointly. The most important point to be remembered in the medical

anatomy of the stomach is that a very small part of it lies to the right of the median line, not more than one-fourth, the remainder occupying the upper left quarter of the abdominal cavity. The *cardiac orifice* is behind the sternal attachment of the 6th or 7th cartilage on the *left* side, while the *pylorus* lies on the *right* side in the line of the tip of the sternum, between it and the junction of the 7th and 8th cartilages, and under the left lobe of the liver. The seats of both orifices—the cardiac and pyloric—and the outline of the stomach vary somewhat with the degree of distention, but when the stomach is moderately distended the highest part of the fundus is in the 5th interspace at the mammillary line, and the lowest part of the organ in the median line three to five cm. (1.2 to two inches) in men and four to seven cm. (1.6 to 2.8 inches) in women above the umbilicus.

The information given by INSPECTION may be of no value whatever, or may possess considerable import. Commonly, however, the stomach and bowels are the seat of such a moderate distention with gas that precise information is rendered unattainable by this method. This is especially the case in little children and men past fifty. In very thin subjects the stomach may be recognized in outline, and exaggerated contractions may even be seen in it and the intestines. Such contraction does not, however, imply of necessity an effort to overcome obstruction at the pylorus or in the bowel below. It may be purely nervous. Morbid growths in the stomach may sometimes be recognized by inspection. So may uneven growths of the liver in thin persons, while the end of a distended gall bladder may, in rare instances, project at the edge of the thorax, near the end of the cartilage of the 10th rib on the right side. Epigastric pulsation is sometimes strikingly conspicuous. Enlargement of the superficial epigastric and abdominal veins is always to be looked for. More frequently there is a circumscribed distention recognized in the region of the stomach, or the entire abdomen may be distended, a symptom which may be due to atony, or, among others, points to some obstruction in the lower part of the bowels. In other instances the opposite state of undue flaccidity is observed—the belly flattening out laterally as the patient lies on his back, or falling forward when he stands up—a condition known as *enteroptosis*. The latter condition occurs especially in persons who have been corpulent and have grown thin, and in women who have borne many children. The patient may also be examined in the knee-elbow position, which will permit movable tumors to fall forward and facilitate their recognition by inspection as well as by palpation.

PALPATION furnishes at times more definite information than inspection. It should be practised by laying the hand flat upon the abdomen and depressing the ends of the fingers as the hand is moved about, rather than by "poking" with the fingers of the straight hand obliquely placed. The abdominal walls, too, should be relaxed by semi-flexing the thighs on the abdomen and the legs upon the thighs. Thus we learn of the consistency and situation of various organs and abnormal growths, whether they are smooth or uneven, whether there is tenderness or tenseness. In so doing, the degree of pressure must vary. Some pains are relieved by pressure, others aggravated. The former are more apt to be due to neuralgia or colic, the latter to be inflammatory. Our knowledge

of the more precise situations of morbid growths is often aided by changing the position of the patient. A tumor of the pyloric orifice of the stomach, which is more apt to be felt toward the median line, above the umbilicus, is also characterized by its greater mobility, as well as change of location with varying degrees of distention of the stomach. Such a tumor may be subject to a peculiar rotary motion.

Percussion of the gastro-intestinal region is practised with the patient on his back in a relaxed position, like that described for palpation. A pleximeter is here conveniently used. The stomach and intestines approach the surface in health in such a way as to make their limitation quite possible by percussion. They require, also, delicacy in discriminating shades of sound, more particularly as to pitch. The quality met with in percussing these organs is, for the most part, tympanitic, and it is chiefly variations in the pitch which are to be discriminated. The same organ may exhibit different degrees of pitch under different conditions. Thus, the stomach when moderately distended with gas gives a low-pitched tympanitic sound when percussed; when more distended it gives a higher pitch; when distended to a maximum it may give a dull sound, because all vibration is stopped. Given the stomach and intestine in an equal degree of tension, the stomach will respond to percussion with a lower-pitched tympany than the intestine because it is a larger cavity. This is sometimes spoken of as less tympanitic. Sometimes the stomach percussion note is ringing, amphoric, echoing. By means of these differences, when present, we may distinguish one hollow organ from another. Again, the presence of liquids or solids in the stomach influences the percussion note.

The hollow viscera *en masse* can be mapped out by determining the boundaries of the solid viscera around them. But we want to do more than this. We want to separate one hollow organ from another—the stomach from the small intestine, the small intestine from the large. For this the patient must be recumbent. As stated, the stomach tympany is ordinarily lower pitched than the bowel tympany. Bearing this is mind, we can generally determine the stomach boundaries when the organ is moderately distended with gas. The upper border of the stomach, as recognizable by percussion, corresponds with the lower edge of the left lobe of the liver. To the left of the apex of the heart, the stomach tympany is mixed with the resonance of the lung. At this point, about the 5th rib, is the cardiac end of the stomach. Percussing downward and a little backward from this point, we are generally able to find a difference of note—a higher pitch, a purer tympany, belonging to the transverse colon. Following this line anteriorly we find it crosses the left edge of the thorax at about the cartilaginous attachment of the 10th rib, the median line just above the umbilicus, and passes thence upward to the junction of the right lobe of the liver with the edge of the thorax. It is the line of the greater curvature of the stomach. Between this and the upper dull area furnished by the liver is a hand-breadth space of tympany called by Traube the *half-moon space*. It is rather *crescentic* in shape. Leichtenstern has applied the name pulmono-hepatic angle to the point of junction between the lower edge of the left lobe of the liver and the lower border of the left lung. The tip of this angle is behind

the 6th rib, just below the apex seat, and is bisected by the pleural angle, which is only filled by the lung during deep inspiration. Into this angle the stomach protrudes, and it is a point pretty constantly maintained. The half-moon tympanitic space may be converted into one of percussion-dulness by filling the stomach with food, or the line of demarcation may be made more distinct by having the patient drink a glass of water just before the examination; or, as originally suggested by Frerichs, by taking in rapid succession the two portions of a Seidlitz powder, tartaric acid and sodium bicarbonate, or a glass of soda-water. A better method is to inflate the stomach with air, as suggested by Runeberg, by means of the double bulb of a spray apparatus. This should be done, if possible, in connection with the use of the tube for some other purpose, as removing the stomach contents after a test meal. The possibility of air passing through the pylorus is to be remembered, but, commonly, if any excess is introduced it passes out alongside of the tube, which is not the case with carbonic acid gas, which excites rather a spasmodic contraction of the cardiac orifice. Both gas and water may distend the stomach beyond the limits described, but the normal limit of the lower curvature may be put above the umbilicus, although it cannot be said to be abnormally low when at the umbilicus, an event not unusual after fifty years of age. As already mentioned, the greater curvature is not quite as low in women as in men, and in working women not so low as in those of leisure. When the lower curvature is below the umbilicus, the stomach may be said to be dilated.

As stated, the percussion note of the large intestine is higher pitched and more purely tympanitic than that usual to the stomach. When containing fæces it is rendered duller, and in consequence of this fact there is often less resonance in the left iliac fossa than in the right, although fæces may also accumulate in the latter, and an impaction in the head of the colon give positive dulness. The colon may also be artificially distended with gas, for examination, if desired.

The percussion note of the small intestine is usually still higher pitched than that of the large, and by this it may be distinguished from that bowel, if not filled with solid matter or liquid. The differences in percussion note alluded to are not always equally marked and it is not always possible, in consequence, to demark the organs. Especially difficult is it at times thus to distinguish the transverse colon when distended with gas from the stomach above it. If the stomach be filled with water a dull note is brought out on percussion which contrasts strongly with the tympanitic note of the gas-distended colon.

Whether determined by inspection, palpation, or percussion, a stomach whose greater curvature reaches the umbilicus or below is abnormally dilated, while certain dilated stomachs go far below the umbilicus. The vertical diameter of the normal stomach from the highest to the lowest points of tympany as determined by Wagner was 11 to 14 cm. (4.4 to 5.6 inches) in men, and about 10 cm. (4 inches) in women. The width of the zone was 21 cm. (8.4 inches) and 18 cm. (7.2 inches). Other measurements are somewhat different, so that some latitude must be allowed.

Auscultation has a less useful application to diagnosis of diseases

of the stomach. It is confined to the so-called *deglutition murmurs*, of which there are three. One is heard at the beginning of swallowing, when the food is transmitted from the pharynx into the œsophagus, which has no significance. In addition, Ewald describes two murmurs, best heard near the cardiac orifice, just below the ziphoid, which he calls the first and second. The first is heard immediately after the beginning of deglutition, and is a hissing sound as if produced by fluid squirted directly into the stethoscope (Spritzgeräusch). Six or seven seconds later is heard the second sound, consisting of a series of tones rapidly following each other, either gurgling, clucking, sprinkling, or splashing (Pressgeräusch). The first or hissing sound is heard but rarely, and is said to denote a relaxation of the cardia and the direct passage of food into the stomach. The second is quite constant and is absent only when the first is heard. Its cause is not so well known, being ascribed by some to audible vibrations of the cardia, caused by the passage of food over it; by others to a pressing through of the air swallowed with the food.

It is the absence of the deglutition murmurs rather than their presence on which diagnostic value depends; that is, they are apt to be wanting in obstructive disease of the cardiac orifice, although too much stress must not be laid upon their absence, since it may occur in health, and repeated observation is required before conclusions dare be drawn.

Gastroscopy and gastrodiaphony have not as yet been sufficiently perfected to be available in diagnosis of stomach affections.

The investigation of the large intestine by percussion is sometimes aided by distending the bowel with gas in one of the various ways suggested for the stomach, the bowel being previously evacuated by an enema. The large bowel may also be explored for a considerable distance from its anal end by specula.

Internal Examination or Chemical Examination of Gastric Contents.

For removing the gastric contents for examination the gastric tube or catheter is used. That usually employed is a thoroughly soft, flexible, red rubber tube, open at the inner end, or if closed at the end provided with lateral openings like a Nelaton's soft catheter. W. v. Leube insists upon the latter, since he himself on one occasion drew in the open end of the sound a portion of the mucous membrane of the stomach which had been aspirated into it. The tube should be about 95 cm. (*circa* three feet) long. From the fundus of the stomach to the incisor teeth is 60 to 65 cm. (23.5 to 25.5 in.), and the tube is usually marked at this point, thus enabling one to judge whether it has entered the fundus. Sufficient lubrication is secured by moistening it with water. It is carried into the back part of the pharynx in the manner directed for the sound (p. 225), when the patient is directed to swallow. At the end of the act of deglutition, the tube is pushed gently downward, and the patient again directed to swallow. This is kept up until the tube enters the stomach. A long tube permits the stomach to be emptied by syphonage after a little pressure on the abdomen has been exerted by the

hand to start the motion of the contents. This is safer than aspiration by a pump, as sometimes practised.

For analysis of the gastric contents the *test meal* heretofore commonly employed is the test-breakfast of Ewald, consisting of an ordinary roll * weighing about 35 gm. (nine drachms) and 300 c. c. (10 oz.) of water or weak tea without milk or sugar. At the end of one hour after the meal is ingested, the stomach is emptied by expression and syphonage as described. There should be 20 to 40 c. c. It is first examined by the microscope for blood or other abnormal morphological constituents and then filtered, being previously well shaken.

Acids of Digestion.—In healthful conditions, in ten or fifteen minutes after food ingestion the gastric contents are acid, the acidity depending on free acids or acid salts. At this stage the free acid recognized is lactic. Up to thirty to forty-five minutes the lactic acid predominates, while the tests for hydrochloric acid are negative. Then comes a stage in which traces of HCl can be demonstrated, co-existing with lactic acid. Finally, the lactic acid disappears altogether, and at the end of an hour HCl only should be present. HCl is present from the beginning, but its recognition is interfered with partly because it is in combination with bases. Free HCl gradually increases in amount until at the acme of digestion it reaches 0.15 to 0.2 per cent. after a light meal, and 0.2 to 0.33 per cent. after an abundant meal.

For simple *qualitative testing of the free acids*, organic and inorganic, the *congo-red* paper or *tropæolin* † paper is most convenient, the former being turned blue and the latter brown, but neither is influenced by these acids when combined with bases.

To test for free *hydrochloric* acid only.—For this Günzburg's phloroglucin vanillin or Boas's resorcin test is used. Günzburg's reagent consists of *phloroglucin* two grams (grains 30), vanillin one gram (15 grains), alcohol 30 c. c. (f℥j). The solution is pale yellow, and has a decided odor of vanilla. On exposure to light it assumes a dark golden-yellow. It must, therefore, be kept in dark-hued bottles or freshly made as required. A drop or two of the reagent is placed on a porcelain plate or capsule with an equal quantity of the gastric filtrate, and a gentle heat applied, not to boil, but simply to evaporate. If free HCl is present, very soon a beautiful rose-red tinge will appear at the edge of the mixture, or red stripes will be observed. Blowing at the edge will favor the appearance of the red stripes. This test is unmistakable, and surpasses all others in delicacy, being available when HCl is present in the proportion of one to 20,000, or 0.05

* Such a roll, containing about seven per cent. of nitrogen, five per cent. fat, and four per cent. sugar, 52.5 per cent. of non-nitrogenous extractive substances, and 1 per cent. of ash, includes, therefore, the usual elements of a mixed diet.

† These papers are made by dipping strips of filtering paper into watery or alcoholic solutions of the anilin dyes, congo-red or tropæolin 00 (l'orange Poirier), allowing to dry, and preserving for use. The paper is, however, less delicate than the solution.

The congo strikes a beautifully *sky-blue* reaction with a solution containing but 0.02 per 1000. Acid salts produce no change. The tropæolin solution is dark yellowish-red, and a solution of free acid, as HCl, 0.025 to 1000, changes it to a deep dark brown. It is slightly less delicate, therefore, than the congo-red. Acid salts, as acid sodium phosphate, make it straw-yellow. In all of these tests it is necessary to use an excess of the fluid to be tested. This is accomplished by placing five or ten *drops* of the reagent in a test glass or porcelain capsule and adding one to two *c. c.* of the filtered contents.

per mille. The reaction is not simulated by albuminates nor interfered with by salts present in the normal proportion, nor by organic acids.

Boas's test is based upon the fact that resorcin strikes a similar reaction with hydrochloric acid. The solution consists of

Resublimed Resorcin,	5 parts (gr. lxxv)
White Sugar,	3 parts (gr. xlv)
Dilute Alcohol,	100 parts (f℥ iiiss).

Three to five drops of the reagent are poured into a porcelain dish and an equal quantity of stomach contents added. Heat is applied as in Günzburg's test, and a purple-red color appears at the edge of the drop. It is said also to detect 0.05 per mille of HCl.

To Estimate the Total Acidity.—The reaction of the filtered fluid being determined by litmus or congo-red paper, the total acidity is then determined by titration. A Mohr's burette is filled with a deci-normal solution of caustic soda. Ten c. c. of the filtered solution are placed in a beaker and one or two drops of an alcoholic solution of phenol-phthallin added as an indicator. The solution is then slowly dropped from the burette until the red color produced in the fluid by the action of the alkali on the phenol-phthallin no longer disappears on shaking. As a rule, the acidity of the gastric contents, an hour after such a meal, requires four to six c. c. of the deci-normal solution to neutralize it in normal digestion. Figures above and below this are therefore abnormal. The acidity may be expressed in percentage; thus, if four c. c. were required to neutralize ten c. c. there would be 40 per cent., or if six c. c., 60 per cent. total acidity.

If the acidity is due to free HCl alone, *i. e.*, if there are no organic acids present to contribute to the total acidity, this titration will represent the quantity of HCl, and its percentage is easily estimated. One c. c. of the deci-normal soda solution is equivalent to 0.00365 HCl.* If, therefore, the number of c. c. used to neutralize ten c. c. of the solution be multiplied by 0.00365 and again by ten the result will be the percentage. Thus, if six c. c. of the deci-normal solution be used the percentage will be $6 \times 0.00365 \times 10 = 0.219$, within the normal range, which is from 0.14 to 0.24 per cent.; if four c. c. be used the HCl percentage will be $3 \times 0.00365 \times 10 = 0.10$, or less than normal.

The *quantitative* estimation for hydrochloric acid by Mintz's method is as follows: To ten c. c. of the filtered gastric contents add the deci-normal soda solution from a burette until Günzburg's reagent no longer gives a reaction with a drop of the gastric fluid. Thus, if 2.8 of the deci-normal solution are so used, the percentage of free hydrochloric acid will be $2.8 \times 0.00365 \times 10 = 0.1$ per cent.

To Determine the Total Physiologically Active HCl.—By this is meant not only the free HCl, but also that united with albumin and which has commenced its physiological office. Braun's method is highly recommended by v. Leube, as follows:—

Ten c. c. of the filtered gastric contents are neutralized with the deci-normal soda solution, using the pheno-phthallin solution as an indicator. A second ten c. c. is then taken, and to this amount a few

* Deci-normal solution of soda $\frac{N}{10}$ NaHO = 4 gm. Na HO dissolved in 1000 c. c. distilled water. Each c. c. of this solution exactly neutralizes 0.00365 gm. HCl.

more c. c. of the deci-normal soda solution are added than are sufficient to neutralize it. This alkaline fluid is now carefully evaporated in a platinum capsule and incinerated in an open crucible, by which the organic matter is destroyed and the organic acids are converted into carbonic acid. The ash is now treated with as many c. c. of a deci-normal acid solution, preferably *sulphuric acid*, as were required of the deci-normal soda solution to alkalize the ten c. c. of gastric contents; the solution is gently warmed to drive off the CO_2, four drops of a phenophthallin solution added, and the whole titrated with the deci-normal soda solution. The number of c. c. of the soda solution required corresponds to the capacity of the gastric fluid in free and organically combined hydrochloric acid.

Example:—If ten c. c. gastric contents require for neutralization six c. c. deci-normal soda solution, this corresponds to a total acidity represented in HCl = 6 × 0.00365 × 10 = 0.22 per cent.

Then to ten c. c. more of the gastric contents are added say 12 c. c. of the deci-normal soda solution incinerated, and the ash dissolved in 12 c. c. of a deci-normal sulphuric acid solution and titrated with the deci-normal soda solution. Now if five c. c. of the last are required, the total HCl combined and uncombined will be 5 × 0.00365 × 10 = 0.182 per cent. This is minus the organic acids.

Determination of Organic Acids.—These include lactic acid, acetic acid, and the true fatty acids, especially butyric. Acetic acid and fatty acids are not formed during normal digestion, and if present, as they sometimes are, they are either introduced with the food or are produced in a fermentation of the carbohydrates set up by bacteria introduced with the saliva.

The physiological presence of *lactic acid* during what may be termed the first stage of digestion, heretofore regarded as physiological, is now called in question, especially by Boas. Boas, because of his recent discovery that all baker's bread contains lactic acid, substitutes for the ordinary test meal a thin gruel made of a tablespoonful of oatmeal flour to a quart of water and flavored with salt. With this meal he maintains that lactic acid is never found in the stomach *unless cancer is present.* The matter, however, is still *sub judice*, though the following careful data gathered from the experiments of Ellenburger * and Ewald on the subject indicate that there is a primary evolution of lactic acid:—

Kind of Food.	Number of Observations.	Number of Times Lactic Acid was Found.	Time after Taking Food at which Lactic Acid was Found (in Minutes).	Observations at which Lactic Acid was Absent after Taking Food	Free HCl. When First Appeared.
Mixed Diet.	31	26	10–100	5	After 120 minutes.
Bread.	31	13	10–30	18	After 30 minutes.
White of egg.	15	1	75	14	Seldom before 60 minutes.
Scraped meat.	23	17	10–100	16	Seldom after 120 minutes.

* Boas, *Deutsche Med. Wochenschrift*, 1893, pp. 913–940; Ellenburger and Hoffmeister, *Du Bois Reymond's Archiv. f. Physiologie*, 1890, p. 280; Ewald and Boas, *Virchow's Archiv*, 101, pp. 325, 375.

Lactic acid is recognized by its effect upon a very dilute, almost colorless, solution of neutral ferric chloride, which is converted into a canary-yellow color by its action. This is Uffelman's test. It is rendered more certain if the solution is made by mixing a few drops of a neutral ferric chloride solution with one or two drops of pure carbolic acid, and adding water until the solution assumes an amethyst-blue color. Instead of the pure carbolic acid about 2½ fluidrachms (ten c. c.) of a two to five per cent. solution of the acid may be used and the dilution completed by adding iron to the proper tint. A few drops of even a 0.05 per mille solution of lactic acid (one to 20,000) will change the blue to the distinctive yellow color.

There are, however, sources of error. The lactates cause the same reaction, but this matters not, because we desire to recognize the lactic acid, whether in combination or not. The reaction, however, takes place with alcohol, sugar, and certain salts, especially phosphates, which are often found in gastric contents. The color produced by phosphates is not identical, but if the filtrate operated with has a yellow tinge the resulting color may approximate it very closely.

Under these circumstances the lactic acid must be extracted with ether. One-half to 1½ fluidrachms (two to five c. c.) of the stomach contents are thoroughly shaken with three or four times the amount of ether. The ether is allowed to rise to the top, which it does rapidly, and is then poured off into a glass beaker. More ether is added and the washing repeated until in all about one fluidounce (30 c. c.) of ether has been used. The ether is then evaporated by placing the beaker, with its contents, in a vessel of hot water. The residue is redissolved in a few drops of water and *one* or *two* drops of Uffelman's reagent allowed to fall from a pipette into the solution. Too much of the solution may mask the reaction. This test is much more delicate than tropæolin, which may fail to show a reaction for free acid because of its concealment by acid salts.

The *fatty* acids, especially *butyric*, strike a tawny yellow color with a reddish tinge with Uffelman's chloride of iron solution, but 0.5 per 1000 or one to 2000 is required before the reaction occurs.

Fatty acids may also be detected by heating to the boiling point a few c. c. of the gastric filtrate in a test-tube, over the mouth of which a strip of moistened neutral or blue litmus paper is placed. On this the vaporized acid will produce the usual change.

The oily particles of pure *fat* may be recognized floating in the gastric contents or in the aqueous solution of the residue after evaporating the ethereal extract. Butyric acid may also be separated in the form of drops by adding small pieces of calcium chloride.

Acetic acid is easily recognized by its odor, but it may also be detected by neutralizing with sodium carbonate the watery residue after the removal of the ethereal extract, and then adding neutral ferric chloride solution. A striking blood-red color is struck, also produced by formic acid, but this is never a constituent of gastric contents.

Alcohol, which is sometimes formed in the stomach in intense yeast fermentation, may be detected by Lieben's iodoform test applied to the distillate of the stomach contents, as follows: To a portion of the distillate add a small quantity of liquor potassæ, then a few drops of a solution

of iodine and iodide of potassium (1, 2, 50). If alcohol is present, a yellowish precipitate of iodoform takes place slowly, which may also be recognized by its odor. The same precipitate occurs with acetone, but rapidly.

Examination of Products of Albumin Digestion. — The term *proteolysis* is applied to albumin digestion, in which, if complete, all proteid food stuffs are converted into peptone. It takes place partly in the stomach through the agency of pepsin-hydrochloric acid, but probably even to a greater degree in the small intestine, by the action of trypsin, the pancreatic digestive ferment. In this process the first step is the production of certain substances, intermediate between albumin and peptone, which are called albumoses. Those which are of chief importance in the study of gastric digestion are syntonin or acid albumin and the various proteoses.* In the ordinary process of digestion with a normal gastric juice, some or all of these substances should be some time present. So far as they are the products of gastric digestion, they may be studied by the aid of a test meal and removal of the gastric contents, as already described.

The products of pepsin proteolysis may, in a general way, be divided into three groups :—

(1) Those precipitated by neutralization and represented mainly by syntonin or acid albumin.

(2) Those precipitated by saturation of the neutralized fluid with ammonium sulphate and represented by the proteoses.

(3) Those non-precipitable by ammonium sulphate and represented by what is commonly known as peptone. The relation of these products to each other is shown by the following diagram, proposed by Neumeister :—

Native Proteid.

Syntonin

Protoproteose	Heteroproteose (dysproteose)
Deuteroproteose	Deuteroproteose
Peptone	Peptone.

Proto and heteroproteoses are primary bodies formed directly from the initial product, syntonin, by the further action of the ferment. Again deuteroproteose is a secondary proteid, being formed by the further hydration of the primary body. Finally, peptones, the ultimate products of pepsin-proteolysis, are the result of the hydration and possible cleavage of deuteroproteoses. The two primary proteoses differ from each other

* The so-called propeptone or hemialbumose is a mixture of proteoses.

more particularly in that protoproteose is readily soluble in water alone, while heteroproteose is soluble only in salt solution, dilute acids, and alkalies.

To Separate Proteoses (Propeptone) and Peptone.—Take two or three c. c. of the stomach filtrate and remove any acid albumin or syntonin by neutralization and filtration. To a portion of the filtrate apply the Biuret test, adding one c. c. of liquor potassæ and a few drops of a one per cent. solution of cupric sulphate. A purple-red color indicates the presence of proteoses and peptone.

The neutral filtrate is then treated with an equal quantity of a saturated solution of sodium chloride and one or two drops of strong acetic acid added. Proteoses if present are precipitated, and may be filtered out. To the filtrate again apply the Biuret test. A purple-red color indicates the presence of peptone and its quantity may be approximately estimated by the intensity of the reaction, provided we always use the same proportion of stomach contents, solution of potash and cupric sulphate. Should it happen that a handsome Biuret reaction is struck before removing the proteose or but a faint one or none at all afterward, the proportion of proteose is large and peptone small. Cahn has shown that in dogs, at least, the quantity of peptone remains at a certain percentage, being probably kept at that figure by its removal as formed. Hence the only index of the rapidity and extent of albumin transformation is the amount of proteose formed or remaining. Finally, Ewald, Gumlich, and most recently R. H. Chittenden, conclude that the formation of true peptone in the human stomach is small. It is true, peptone may be found in relatively large amount, but a quantitative estimation of the proteoses and peptone always shows the former to be in excess. Gastric digestion is rather, therefore, to be considered as a preliminary step in proteolysis, and is preparatory to the more thorough office of pancreatic digestion.

To Estimate the Activity of Proteolysis, or Albumin Digestion.—By Ewald's method, coagulated white of egg is cut into thin slices and out of these small discs are cut by a cork-borer or similar instrument. These may be prepared in quantity and kept for use in glycerine, which should, however, be washed off before using. An equal quantity of the filtered gastric fluid is placed in four small test-tubes and one or two discs of albumin put into each. To the first nothing else is added, to the second enough hydrochloric acid to make a solution of about* 0.3 to 0.5 per cent. This is accomplished by adding two drops of hydrochloric acid to 90 minims (five c. c.) of stomach contents. To the third is added a definite quantity of pepsin, about three to 7½ grains (0.2 to 0.5 gm.), to the fourth both hydrochloric acid and pepsin.

The test-tubes are placed in an incubator at about 100° F., and from time to time examined with a view to learning how far the liquefaction of the discs of albumin has proceeded. The rate of this will inform us whether digestion would have occurred without the addi-

* The difference between the strength of the acetic acid of the German pharmacopœia (25 per cent. of the anhydrous acid), intended by Ewald, and that of the U. S. P. (32 per cent.) is not sufficient to necessitate a change of proportion.

tion of anything, or whether acid or pepsin or both were necessary. We will learn, also, whether by adding more hydrochloric acid we have made the acidity excessive.

It must be remembered, however, that after the peptone has reached a certain percentage its further production is retarded, or even suspended, so that there may be an apparently slow reaction with even a very active gastric juice. Ewald happily reminds us that all laboratory attempts to imitate digestion are defective in the important respect that with our test-tubes and flasks we can neither imitate absorption on the one hand, nor, on the other, allow for the onward movement to the intestines of the gastric contents, two important functions by which the stomach strives to maintain a fairly uniform degree of concentration of its contents.

The Action of Rennet, the Milk-coagulating Element of the Natural Gastric Juice.—The simplest method of estimating the action of rennet is that of Leo. To 2⅔ fluidrachms (ten c. c.) of *raw* milk are added two to five drops of stomach contents. Raw milk is used because it coagulates ten times more rapidly than boiled milk, while neutralization is unnecessary because of the relatively small quantity of gastric juice used. The mixture is placed in the warm chamber at 100° F. (37.8° C.), and coagulation should take place in from one minute to several hours. The characteristic coagulating of rennet is a cake of casein floating in clear serum, while acids produce lumpy and flaky masses.

The rennet-ferment, or enzyme, does not exist primarily as such, but as a rennet-zymogen or proenzyme, which itself has no action on milk, but is converted into rennet by the action of any acid, as hydrochloric, or of warm chloride of calcium. This may be shown as follows: If the spontaneous coagulating action of gastric juice or milk be destroyed by neutralization by an alkaline carbonate, this property may be restored by digesting with dilute hydrochloric acid, or by the addition of a five per cent. solution of calcium chloride. While fasting, and at the beginning of digestion, zymogen only is present in the stomach, but, later, both it and the ferment are found. An acid reaction for the curdling action of rennet is not absolutely necessary.

Digestion of Starch and Sugar.—It is well known that during digestion starch is converted into grape sugar, and cane sugar is converted into *invert* sugar—a mixture of cane and grape sugar. This action, commenced in the mouth by the ptyalin of saliva, is continued to a less degree so long as the acidity is slight (0.01 per cent. for HCl, 0.1 or 0.2 per cent. for lactic, 0.4 per cent. for butyric) in the stomach, and is finished in the small intestine by the trypsin of the pancreatic juice. As in albumin digestion, there are intermediate substances between albumin and peptone, so between starch and grape sugar there are similar intermediate products. The order is as follows:—

(1) Starch.
(2) Dextrins (Erythrodextrin, Achroödextrin).
(3) Maltose.
(4) Dextrose = grape sugar.

Starch is recognized by the deep blue color struck with iodine or Lugol's solution (iodine 1, iodide of potassium 2, distilled water 200), and

the reaction grows less vivid as the starch is converted. Of the dextrins, erythrodextrin strikes not a blue, but a purple color, while solutions of achroödextrin, maltose, and grape sugar take on only the yellow color of the iodine solution. Where a mixture of these substances occurs, the first few drops of the iodine solution produce no color at all, or only a transitory one, being taken up by the dextrose and maltose, while the addition of more iodine strikes the purple of erythrodextrin or blue of starch.

If, therefore, amylaceous transformation has progressed normally in the mouth and stomach, so much starch should be changed into achroödextrin, maltose, or dextrose that the addition of small quantities of Lugol's solution does not strike the characteristic color. If, however, the blue or purple reactions appear, conversion has not been sufficiently rapid into maltose, the principal product of gastric conversion, the change into dextrose being completed in the small intestine. This may be due either to a deficiency of ptyalin or a too rapid production of acid in the stomach. From such event we might also infer a hyperacidity of the gastric juice.

To Determine the Rate of Absorption from the Stomach.—Penzoldt's method is that generally followed. A capsule containing iodide of potassium, 1½ grain (0.1 gm.) is swallowed, being first carefully wiped to remove any adherent particles. The appearance of the iodide in the saliva indicates that absorption has taken place from the stomach. To determine this, starch paper is first prepared by moistening with starch paste and drying. Then, after the salt is swallowed, a piece of the paper is moistened every five minutes with the saliva, and the moistened spot touched with fuming nitric acid. As soon as the iodine appears in the saliva the characteristic blue reaction is struck.

When absorption is normal this reaction usually takes place in ten or fifteen minutes, but where absorption is abnormally delayed, the reaction is also delayed half an hour or more, or it may not occur at all.

To Test the Motor Function of the Stomach.—Three methods are practised. In one the gastric contents are withdrawn six to seven hours after the ingestion of a large meal, or two and a half hours after an Ewald's breakfast. There should be no solid residue. The more suitable meal for this purpose is the larger one composed of 13 ounces (400 c. c.) of beef soup, 6½ ounces (200 gm.) beefsteak, 1½ ounce (50 gm.) bread, and 6½ ounces (200 c. c.) water. In a second, salol is administered, and the products of its lysis sought for in the urine. In the third, a definite quantity of olive oil is introduced into the empty stomach, and the remnant unabsorbed is withdrawn at the end of two hours. The second, though not without drawbacks, is preferred. Salol is composed of phenol and salicylic acid, into which it is broken up by the action of the pancreatic juice, but not by the acid gastric contents. Salicyluric acid, a product of decomposition of salicylic acid, appears in the urine forty to sixty, or at most seventy-five, minutes after taking 15 grains (one gm.) of salol when gastric peristalsis is normal. Salicyluric acid is readily detected in the urine by the violet color produced on the addition of neutral ferric chloride solution. The method employed is to place a drop of urine on a piece of filter-paper and bring in contact with this a drop of a ten per cent. ferric chloride solution. The edge of the

drop will strike a violet color in the presence of a mere trace of salicyluric acid.

The salol test is objected to on the ground that its decomposition may be delayed by undue acidity of the gastric contents discharged into the duodenum. Practically this is not a serious drawback, tolerably constant results being obtained. To meet this objection, however, Huber suggested that the outside limit of excretion of salicyluric acid be determined; that is, the point noted when salicyluric acid fails to appear in the urine after the ingestion of 15 grains (one gm.) of salol. This should occur at the end of twenty-four to thirty hours. If, therefore, it continues after this, peristalsis must be slow.

In Klemperer's oil test 3½ ounces (100 c. c.) of olive oil are introduced into the stomach by the tube after the organ is thoroughly washed out. Two hours later the stomach is aspirated, and if there is motor sufficiency there should be a very small residue. If any decided quantity remain, it is fair to conclude peristalsis is slow.

ACUTE CATARRHAL GASTRITIS.

SYNONYMS.—*Acute Gastric Catarrh; Acute Dyspepsia; Gastric Fever.*

Definition.—Acute inflammation of the stomach, of moderate intensity, due to simple non-specific irritation.

Etiology.—This form of inflammation occurs at all ages and is commonly due to the irritant effect of indigestible food or food in a state of incipient decay and fermentation. Simply overloading the stomach, even though the food be wholesome, may be a sufficient cause. The introduction of large quantities of strong alcoholic drinks, such as is practised in a debauch, is one of the most common causes of acute gastritis of the simple variety. The susceptibility of different individuals and of different families to the above-named causes of irritation varies greatly.

Morbid Anatomy.—A more or less uniform coating of the stomach with mucus is the most constant feature of simple acute gastritis, and justifies for it the name, gastric catarrh. The removal of this mucous coating reveals a hyperæmic redness, which in the highest degrees may be associated with punctiform hemorrhages and hemorrhagic erosions. The mucous membrane is swollen, and œdematous and minute examination recognizes numerous mucus-laden cylinder cells, which have been extruded from the mucous glands everywhere present, while even the peptic gland cells are cloudy and granular.

Symptoms.—These are a natural sequence of the morbid state. A *want of appetite* and *loathing of food*, *nausea*, rarely *pain*—these are the more constant subjective symptoms. To them may be added an *unpleasant taste* in the mouth, sometimes bitter, sometimes metallic, a *pasty* sensation or dryness, and even *thirst*, a sense of *fulness in the head* rather than headache, and *dizziness*, and often extreme *mental depression*.

Objective symptoms are epigastric *distention*, more rarely *tenderness*, a *coated tongue*, *dryness* of the *lips*, rarely *herpes*, a *heavy* breath, acid or bitter *eructations*, sometimes a scanty secretion, at others an excess

of saliva, finally *retching* and *vomiting* with greater or less relief. The *bowels* are constipated, though sometimes there is diarrhœa. *Jaundice* is occasionally present and indicates that the inflammation extends into the duodenum and produces obstruction of the common bile duct. There is slight *fever*, sometimes decided, with a temperature of 101° to 103° F. (38.3° to 39.4° C.), and a corresponding pulse. On the other hand, the pulse is not infrequently slowed below the normal, being inhibited by the gastric irritation. The *urine* is "feverish," scanty, and high colored, with a corresponding specific gravity and a tendency to deposit urates. There are, however, cases without febrile symptoms. Indeed, v. Leube says that in a few instances only is fever the result of acute gastric catarrh, but the latter condition is rather the result of some acute febrile process, as, for example, one of the infectious fevers. It has occasionally happened that gastritis has been ushered in with a chill.

The vomited matter and gastric contents removed after a *test meal* show a deficiency in hydrochloric acid, an excess of mucus, lactic and fatty acids, and more than the normal residue of undigested food. Digestion is prolonged, the stomach-washing exhibiting a considerable amount of undigested food seven hours after the ingestion of a test meal. Indeed, it often happens that from twelve to twenty-four hours after the beginning of such an attack, large quantities of undigested food are vomited in much the same condition in which they were swallowed.

Diagnosis.—This is not usually difficult, except in the case of the febrile form. In this form, especially where the disease has been ushered in with chill, it is sometimes difficult to decide between it and some one of the infectious fevers, but a few days' expectation will soon remove the doubt by the appearance in the latter of eruptions or other distinctive symptoms. The presence of a cause sufficient to excite gastric inflammation will add to the probability of the presence of acute catarrhal gastritis.

Prognosis.—This is invariably favorable in cases of true simple gastritis.

Treatment.—Many mild cases recover spontaneously if left alone and all food withdrawn for twenty-four hours. The symptoms gradually subside and the patient recovers. In a few cases where there is evidently retained food, an emetic will give relief; in all a brisk saline purge is helpful. A bottle of cold solution of citrate of magnesium in divided doses, say a fourth every half hour, is one of the most agreeable and efficient aperients to relieve the congestion and the symptoms. Or some one of the natural aperient waters, such as Hunyadi Janos or Friedrichshalle or Carlsbad, may be substituted. If there is great sensitiveness of the stomach, small doses of calomel frequently repeated, say $\frac{1}{6}$ to $\frac{1}{4}$ grain (0.011 to 0.016 gm.) every hour, may be substituted, or 10 grains (0.666 gm.) of calomel may be given in one dose. In either event a saline should be given sooner or later, as in this way is secured copious depletion of the upper alimentary canal. The alkaline mineral waters, represented by the Vichy, Vals, and Contrexville waters in France, but which have unfortunately no equivalent in any of the natural mineral waters in this country, are admirable adjuvants, since they aid in clearing the stomach of mucous secretion and in producing osmosis. The saline

mineral waters represented by the well-known Saratoga waters of this country are also efficient, more especially by their aperient qualities.

CHRONIC CATARRHAL GASTRITIS.

SYNONYMS.—*Chronic Gastric Catarrh; Chronic Catarrhal Dyspepsia.*

Definition.—A condition of chronic hyperæmia, associated with excessive mucus secretion and deranged gastric juice formation, with ultimate structural changes in the mucosa.

Etiology.—Any cause which will produce continuous moderate irritation of the mucous membrane of the stomach is capable of producing chronic catarrhal gastritis. The immoderate use of alcohol is probably the most frequent of these, but constant over-eating is also a common cause, especially rapid eating.

Very frequently, too, chronic gastritis is secondary to primary disease elsewhere, and especially mitral disease of the heart and interstitial hepatitis. Both of these affections cause a passive congestion of the stomach, which ultimately produces the lesions characteristic of chronic gastritis. Thrombosis of the portal vein acts similarly. Chronic pulmonary disease, and even diseases of the pleura impeding the circulation in the lungs, produce similar effects through stasis. A predisposition exists in certain families to chronic gastric catarrh.

Morbid Anatomy.—The fundamental condition is a hyperæmic swelling of the gastric mucosa. This is favored by the superficial situation of the venous plexuses about the mouths of the gastric glands as contrasted with the deep-seated position of the arterial network around their bases, by the thinness and compressibility of the venous walls, and sluggishness of circulation necessitated by the peculiar secretory function of the stomach. The hyperæmic surface is, however, more or less obscured by a tough yellowish-white covering, made up of mucus and emigrant pus cells. The changes are more marked at the pyloric end.

These may constitute the sum of changes, but in more chronic cases minute examination reveals a varying degree of hyperplasia of the connective tissue, and even of the mucous glands, which exhibit in places an atypical branching, like the fingers of a glove. The tubules are distended by secretion in some places, and in others stenosed by the contraction of the overgrown connective tissue surrounding them. The hyperplastic process may result in plication of the mucous membrane such as is natural at the pyloric end, and lead finally to the *mammillated stomach* by atrophy and contraction of certain portions, and to more pronounced swelling of the remaining parts. An ultimate result is sometimes the rare condition known as *polyposis ventriculi.* Atrophy of the mucous membrane may be extensive, and even almost total.

Symptoms.—These naturally result from the morbid state. The mucous membrane is bathed with *mucus.* The *gastric juice* is *imperfect* in quality and quantity. Especially is the hydrochloric acid deficient. Digestion is therefore imperfect, the residue of ingested food undergoes fermentation and decomposition, generating lactic, acetic, and butyric acids and alcohol. Peristalsis is delayed because of the absence of its

natural stimulus, and thence follows a further retention of food in the stomach. The natural consequence of such morbid changes is *loss of appetite* and even *disgust for food*, an unpleasant *taste*, a *pasty sensation in the mouth*, a *coated tongue*, *heartburn*, *acid eructations*, *nausea*, often vomiting, sometimes immediately, sometimes an hour or two after taking food. The vomitus consists of undigested food, usually mixed with a large amount of mucus. Its reaction may be neutral or acid, sometimes even acridly so, but the acidity is not due to hydrochloric acid, which is diminished, but to the organic acids generated in fermentation. Analysis of the gastric contents, withdrawn after a test meal, shows a similar deficiency of pepsin as well as hydrochloric acid, while the other tests described discover retarded peristalsis and delayed absorption. Occasionally there is a little blood present, and frequently fungi, especially yeast-spores and sarcinæ ventriculi.

To these symptoms may be added *headache*, or a dull, unpleasant feeling in the head, *vertigo*, *disturbed sleep*, *depression of spirits*, a sense of weariness and disgust of life, often *distention* and a sense of fulness in the stomach, causing even *pain*, which adds not a little to existing discomforts. There may be *tenderness*, but it is diffuse, and not circumscribed. There is usually *constipation*, while the urine is apt to be scanty. Reflected symptoms are *palpitation*, *frequent*, *slow*, or *irrregular pulse*, shortness of breath. There is no fever. *Cough*—the so-called "stomach cough"—is sometimes present, but more frequently what is called by the patient stomach cough is the cough of tubercular phthisis, which the sanguine patient easily convinces himself is due to stomach derangement. Should the disease progress to total atrophy, the gastric contents, after a test meal, may even be devoid of mucus as well as free and combined hydrochloric acid, and pepsin, blood, and epithelium, and be made up mainly of undigested food, with bacteria and a few round cells. Repeated examinations of stomach contents, after a test meal, may be necessary before a confident knowledge can be arrived at of its features.

Diagnosis.—With the symptoms detailed, and the altered state of the secretory, absorptive, and motor functions of the stomach, ascertained as directed, there is usually no difficulty in diagnosing a condition of chronic gastric catarrh. It is to be remembered, however, that chronic gastric catarrh may accompany ulcer and carcinoma of the stomach, in which the otherwise distinctive symptoms of the former are obscured, while with the exception of tumor and occasional coffee-grounds vomit the symptoms of carcinoma may not differ from those of chronic gastric catarrh, hydrochloric acid and pepsin being deficient in both. Dilatation of the stomach is also accompanied with symptoms of gastric catarrh, including even the clinical characters of the gastric juice, and careful examination must always be made for the physical signs of dilatation.

Prognosis.—The prognosis and treatment will depend upon the etiology. If the chronic gastric catarrh is a result of chronic cardiac or hepatic disease, it is curable only so far as these affections are curable, and is relieved as these are relieved. Careful physical examination is always necessary in each case, that obscure cases may be recognized.

Chronic gastric catarrh, not the result of organic heart or pulmonary or liver disease, and which has not already resulted in atrophy of the

mucous membrane, may be cured by careful and persevering treatment. If there is extensive atrophy of the gastric mucous membrane, a proper assimilation of food becomes impossible, and the symptoms of anæmia are ultimately added. Their close resemblance to those of pernicious anæmia has been pointed out, while an essential cause of pernicious anæmia has been held to be gastric atrophy, in evidence of which a case of William Osler and Frederick P. Henry is often quoted.

Treatment.—The treatment of chronic gastric catarrh caused by chronic liver or heart disease is largely that of these affections, but the treatment useful in the ordinary primary forms of the disease may be with advantage associated with that of the more chronic affection.

A successful treatment of catarrhal dyspepsia requires considerable patience, but if the diagnosis is correctly made and the cause is removed, the patient may be promised a cure in time. Of primary importance is the elimination of the cause, whether it be alcohol or bad habit in eating. Simple, wholesome, and properly cooked food, thoroughly masticated and slowly taken, should be the rule of every life, and the simple forms of the disease may sometimes be cured by a return to such a habit, especially if a proper action of the bowels is also habitually secured.

It is not easy to select a diet which will suit every case, and after the injunction that articles evidently difficult of digestion, such as pastry, oils, and fats, are to be excluded, it is often sufficient, and even necessary, to leave the choice of special articles to the patient, with the direction to discard what his experience teaches is harmful. Often, however, the patient cannot be trusted to do this, and often the moral effect of specific directions is good, but even then our bill of fare must often be tentative. The measures by which the regular habit of bowel movement is brought about must vary with circumstances, but when it is remembered that we have to deal with a congested mucous membrane, it is plain why the salines which deplete the upper alimentary canal are so efficient, especially when associated with mercurials. Among these are the numerous natural aperient waters, such as Friedrichshalle, Hunyadi Janos, Carlsbad waters, and our own Saratoga and Bedford waters, all of which are said, in common parlance, to act upon the liver, though, in fact, they simply deplete the alimentary canal. In fact, the useful effect of these waters is so often availed of to remove the uncomfortable effect of a debauch in eating, that their use is abused. No remedies are, however, so useful when needed, and the fact that almost any of them can be taken before breakfast, securing an effect after that meal, makes them doubly convenient. A fit substitute for the water, especially when traveling, is the Carlsbad Sprudel Salt, obtained by evaporating the Carlsbad water. Carlsbad salt, of which the dose is usually a teaspoonful, is best taken in a glass of hot water. The natural waters are, however, to be preferred if they can be obtained. The occasional associated treatment by mercurials, especially blue mass, in doses of three to ten grains (0.2 to 0.66 gm.) the evening previous sometimes adds to the efficiency of the salines. Calomel may be substituted in doses of five to ten grains (0.33 to 0.66 gm.), with as much sodium carbonate. If there is nausea, calomel may be given in smaller doses, say $\frac{1}{10}$ to $\frac{1}{5}$ grain (0.0011 to 0.0132 gm.) hourly. Podophyllin may be substituted for the mercurials or added to them in doses

of $\frac{1}{10}$ to $\frac{1}{4}$ grain (0.006 to 0.06 gm.). Cascara sagrada is one of the most valuable of aperients. The best preparations are the solid and fluid extracts. The former may be given in two-grain doses (0.132 gm.) in a pill after dinner and after supper. The fluid extract, in 15- or 20-minim (one to 1.3 gm.) doses, can be given in the same manner, but the dose of each must be modified to suit the requirements of individual cases. In lieu of the saline aperients before breakfast, a glass of hot water alone, slowly sipped while dressing, is often useful and tends to relieve the morning sickness which sometimes attends chronic gastric catarrh. It probably liquefies the mucus and washes it away into the duodenum.

As to medicines intended to aid digestion, the most efficient is hydrochloric acid, which may sometimes be substituted by nitro-muriatic acid. It seems now definitely settled that hydrochloric is the acid to which the gastric juice owes its efficiency, and as well settled that it is diminished in chronic gastric catarrh. Another very important rôle is, however, assigned to hydrochloric acid, viz., an antiseptic effect, in checking the multiplication of pathogenic bacteria—bacteria of fermentation and decomposition—which are continuously introduced with the food into the stomach. A third rôle of hydrochloric acid is the conversion of the granular pepsinogen in the protoplasm of the peptic cells into the enzyme, pepsin. Its scantiness, therefore, not only impairs the activity of the gastric juice, but also favors the acetic and lactic acid fermentations, whose products keep up irritation. On the other hand, pepsin is seldom abnormally scanty, because so little is required for its purpose. As it does no harm, however, it may with propriety be administered with hydrochloric acid. The latter has, heretofore, been administered in too small doses. Not less than 15 minims (one gm.) of the dilute acid should be given, and from 30 to 60 minims (two to four gms.) are sometimes required. It should be given, further diluted, fifteen minutes after a meal, through a glass tube carried back into the fauces, not merely to save the teeth, but also to avoid the unpleasant taste. The pepsin should be given in solution with the hydrochloric acid in doses of five to ten grains (0.33 to 0.66 gm.). The wine of pepsin has always been a favorite preparation with me, notwithstanding the small proportion of pepsin contained in it. I have been in the habit of combining it with the nitro-muriatic acid rather than hydrochloric, and not infrequently adding $\frac{1}{30}$ grain (0.002 gm.) of strychnine to each dose of half an ounce (120 c. c.) or two fluid drachms (60 c. c.) of the wine.

Trypsin or pancreatin is also much used. It is commonly used in the tablet form, five grains (0.33 gm.) at a dose, sometimes keratin coated, that it may not be dissolved until it passes into the small intestine, where alone in the presence of an alkali it is capable of acting.

It is usual also to employ the bitter tonics in the treatment of this form of dyspepsia, including gentian, quassia, columbo, angustura, cardamon, and nux vomica. They are supposed to stimulate the secretion of gastric juice, and should be taken immediately before meals or with food. A moderate amount of alcohol in the shape of a little whiskey with water during meals or a glass of dry sherry is often serviceable, but care should be taken in the use of alcohol, lest a habit be contracted. The persons to whom it is advised should be well selected, and it should not

be recommended to the young. Stimulating condiments, such as red pepper and mustard, often give temporary relief, but they ultimately aggravate the local congestion and should be forbidden. Common salt, on the other hand, is a rational adjuvant, furnishing chlorine for the formation of hydrochloric acid.

Nitrate of silver is also a useful drug in cases of chronic gastric catarrh in doses of ¼ grain (0.0165 gm.), fifteen minutes to half an hour before meals, dissolved in a quarter of a glass of water. I have never found bismuth of much use in this form of dyspepsia. In fact, its tendency to produce constipation is a contraindication to its use. Where there is acidity it may be useful, as may also be sodium bicarbonate and mint, but it is better, if possible, to strike at the root of the evil by preventing the fermentations which produce the flatulence and acid.

In obstinate cases the milk treatment may be resorted to with advantage, and should be carried out with skimmed milk or whole milk diluted with water or Vichy. The efficiency of the milk treatment is largely due to the fact that the quantity of food is greatly reduced. Not more than two ounces should be given at first, every two hours, the quantity increased only as the hunger of the patient demands more. There will be at first a loss of weight, but weight is again recovered with the increase in quantity. Having secured a tolerance for milk, of which from three to five pints (1½ to 2½ liters) are required in twenty-four hours, the interval may be prolonged and other articles of food cautiously added, a little bread and butter, an egg, a chop, or a small piece of steak, broiled. Gradually the simpler vegetables, such as rice and potatoes, may be added, then weak tea and coffee cautiously, the effect of each article being carefully watched. If flatulence is caused by the farinacea and sugars, they should be withdrawn. Hot bread and fats will rarely ever be permissible to such patients. The same may be said of ice cream and iced water with meals, though a moderate amount may be permitted between meals, especially of ice water. Ripe fruits, on the other hand, are very desirable foods and should be allowed tentatively.

In bad cases of chronic gastric catarrh *lavage* is one of the most useful measures. Not only does it wash away the coating of mucus which is at once a hindrance to the secretion of the gastric juice and a cause of nauseous discomfort to the patient, but it also stimulates glandular activity. It should be done in the morning before breakfast, with the stomach tube already described, with funnel attachment. Simple tepid water may suffice, or if there is much mucus a two per cent. solution of sodium bicarbonate or Carlsbad salt, or a one per cent. solution of sodium chloride. If antiseptic fluids are indicated, a two per cent. solution of resorcin may be used, or one per cent. solution of salicylic acid.

The stomach tube having been introduced, as directed on p. 225, the tepid water or solution employed is run in slowly and removed by syphonage, the outer end of the tube being lowered for the latter purpose. This process is repeated until the stomach is thoroughly washed out. Autolavage is easily practised by the patient himself by means of the apparatus illustrated in the text.

It is in these cases, too, that a course at Carlsbad is very efficient, and remarkable cures are reported. Here, too, the restricted dietary and depletion of the upper alimentary canal by the natural mineral waters are the benefiting agents. Similar courses are carried out at Kissengen, Wiesbaden, and Ems, but, unfortunately, we have no such places in America. Saratoga fulfils the conditions so far as an aperient water is concerned, but the majority of persons who go to Saratoga continue eating and drinking as at home. Finally, the habitual use between meals of the alkaline mineral waters alluded to, viz.: Vichy, Vals, and Contrexville, is undoubtedly useful, relieving and averting gastric catarrh.

FIG. 16.—ARRANGEMENT FOR AUTO-LAVAGE.—(*After v. Leube.*)

PHLEGMONOUS OR SUPPURATIVE GASTRITIS.

A form of gastritis in which there is diffuse purulent infiltration of the submucosa, but sometimes also circumscribed abscess, causing a possibly detectable tumor in the gastric region, a tumor which disappears if the abscess ruptures.

Etiology.—Phlegmonous gastritis is a result of infectious processes, among which have been puerperal fever and other forms of pyæmia. It has been found associated with peritonitis and trauma. In more cases a cause is not discoverable. It has been met more frequently in men than in women.

Symptoms and Diagnosis.—Epigastric pain and tenderness, general abdominal pain and tympany, vomiting, diarrhœa, fever, delirium, dry tongue, small, frequent pulse, coma, collapse, and death—symptoms which closely resemble those of peritonitis, with which, as has

been said, it is sometimes associated—are those met in phlegmonous gastritis. The vomited matter very rarely contains pus. It is plain, therefore, that these symptoms, associated with an infectious process, can only give rise to suspicion that the disease is present, since the same symptoms may be caused by peritonitis. Even the vomiting of pus is not diagnostic, because pus may arise from other sources between the mouth and stomach. The presence of a tumor which subsides after vomiting of pus furnishes better ground for suspicion, though vomited pus may also come from an abscess in the vicinity of the stomach which has ruptured into that organ.

Treatment.—This can only be symptomatic, as nothing can be done to avert a termination which is invariably fatal.

Traumatic or Toxic Gastritis.

An inflammation of the stomach caused by the ingestion of corrosive poisons, such as the strong mineral or organic acids, caustic alkalies, phosphorus, arsenic, corrosive sublimate, and the like.

Morbid Anatomy.—The appearance differs a good deal, according to the degree of irritation. In extreme degrees, such as are produced by the strongest acids and alkalies, the mucous membrane is disintegrated, shreddy, and may be converted into a black eschar, the borders of which are lightened up with intense inflammation. In milder forms, such as are produced by phosphorus, arsenic, and strong alcohol, there is cloudy swelling and fatty degeneration of the gastric gland cells and vessel walls, producing ulceration and hemorrhagic extravasation. The fury of the irritation is expended on the fundus, as the part first reached, and its ravages become less extensive as the pylorus is approached.

Symptoms.—These also vary with the degree of irritation, but there is always intense burning pain, tenderness on pressure, thirst, vomiting of blood and even fragments of mucous membrane. To these are added, in severe cases, small, frequent pulse, cold sweat, and collapse. These latter symptoms point to peritonitis, a very frequent complication, the direct result of the deep-seated action of the irritant. If the patient does not perish promptly, symptoms indicating blood dyscrasia supervene, including albuminuria, hæmaturia, jaundice, subcutaneous blood extravasations, and the like. Where recovery takes place, or death is long delayed, varying areas of mucous membrane may be replaced by cicatricial tissue, and there may be subsequent contraction and distortion.

Diagnosis.—This is based on a knowledge that the patient has swallowed a corrosive poison. In the absence of this knowledge, the odor of the breath may suggest the cause, and evidences of corrosive action in the mouth and pharynx often disclose unfailing signs.

Prognosis.—This varies with the degree of lesion. The gastritis caused by the powerful corrosive poison is always fatal. The lesser degrees may be followed by recovery.

Treatment.—This consists, first, in the use of chemical opposites, as vinegar and other weak acids for alkalies and alkalies for acids. The antidotes called for by special substances are freshly prepared ferric hydrate for arsenic, lime water for oxalic acid, cold water and ice after

the specific action of the poison has been counteracted, and ice externally to the abdominal walls. These should be followed by the free use of diluents and demulcents, of which the various mucilages and milk are examples. (See concluding section of book on the treatment of poisons.)

Diphtheritic Gastritis.—This occurs sometimes as an extension from faucial or laryngeal diphtheria, but more frequently it is secondary to typhus or typhoid fever, small-pox, scarlet fever, pneumonia, and sometimes primarily in weak children. There is no way to recognize such condition during life.

Mycotic Gastritis.—It is very doubtful how far fungi can cause inflammation of the stomach. The bacteria which flourish in the mouth are destroyed by the acid gastric juice, while the fungi which thrive in acid fluids, such as the yeast fungus, the penicilium, and the sarcina, are probably accidental results of the retention of the gastric contents beyond the natural time and are not harmful. The possibility of their producing noxious results cannot, however, be denied. Ulceration has even been ascribed to them. On the other hand, the larvæ of certain insects must also be acknowledged as possible causes of inflammation.

NERVOUS DYSPEPSIA.

Synonym.—*Gastric Neurasthenia.*

Definition.—A form of dyspepsia due to nervous influence, in which, notwithstanding the presence of a train of annoying symptoms, the act of digestion is accomplished within the normal time limit of seven hours.

Etiology.—The nervous temperament predisposes to nervous dyspepsia. Any cause which develops an over-excitability of the nervous system may become a factor of nervous dyspepsia. It is this form to which the neurasthenic and over-worked are especially prone.

Symptoms.—It must be admitted that the only constant feature of nervous dyspepsia is the etiological one, yet we may find symptoms to aid a diagnosis apart from the cause. *Pyrosis*, accompanied by loud, noisy eructations, is quite characteristic, while noisy rumbling of the bowels is often heard, caused by *hyperperistalsis*. On the other hand, *vomiting* is *rare*. There is also often *palpitation* of the heart, with other nervous symptoms. *Constipation* is sometimes present. The nervous dyspeptic is also *very anxious about himself*, dwells on his ills, is restless and sleepless, depressed in spirits, though in this latter respect he resembles also the catarrhal dyspeptic. Like him, too, he may have *loss of appetite*, an *unpleasant taste* in his mouth, *nausea*, *dizziness*, *headache*, pressure on the head. As in gastric catarrh, there is a *sense of discomfort* and *fulness* during digestion, but in nervous dyspepsia, if the interest of the patient is strongly excited by external matters, as, for example, pleasant society or even business interest, he may for the time forget it.

Wilhelm v. Leube, who has given the subject of nervous dyspepsia much attention, makes three clinical varieties:—

(1) That of normal secretion, which he says may be regarded as a fundamental type.

(2) That of diminished acidity, and
(3) That of hyperacidity.

In each of these cases the digestion is complete at the end of the normal time, except that sometimes it may be delayed for starches in the third type of hyperacidity. Thus, while nervous dyspepsia is chiefly a sensory neurosis, it is to a less degree secretory and also, to a degree, motor, as evidenced by the associated hyperperistalsis.

An annoying symptom which is sometimes associated with nervous dyspepsia and is at times its chief manifestation is *peristaltic unrest.* Borborygmi and gurgling set up very soon after eating, so loud as to be heard at a distance and thus to become often a mortification to the patient, while this very emotion reacts to increase it. The movement extends to the bowels. The associated discomfort varies greatly and is sometimes extreme. Peristalsis may be reversed, and in extreme cases it is said that enemata and even fecal matter have been discharged per orem.

Diagnosis.—The frequent dependence of nervous dyspepsia on other conditions requires a broad etiological study. Thus, as v. Leube suggests, we have first to settle the question as to whether it is an independent affection or a part of a neurasthenia. The urine should be studied because the phenomena of nervous dyspepsia are sometimes a manifestation of uræmic intoxication of a mild degree in contracted kidney. The spleen should also be explored, because it is sometimes a result of malaria. In other cases still it is a symptom of chlorosis or hysteria. In all these cases the nervous dyspepsia is the effect of the operation of the disease on the nervous system, and again in other cases it is the result of sympathy with sexual diseases, especially in women with disease of the uterus and ovaries.

Again we have to distinguish it from ulcer of the stomach, which the hyper-acid form resembles, but from which it differs in that the pain is relieved by pressure and sometimes by taking food, both of which acts increase the pain in ulcer. The occurrence of hemorrhage from the stomach, of course, under these circumstances points definitely to ulcer. Nervous dyspepsia may, however, continue as a sequel of both healed ulcer and gastric catarrh, since their effect is a neurasthenic one. Finally it is to be distinguished from the form of catarrhal dyspepsia with similar symptoms by the delayed completion of the digestive act characteristic of the latter, as well as the etiological factor, which is the most important criterion.

Treatment.—The treatment of nervous dyspepsia varies with the cause, but it is desirable also to determine by chemical examination the state of secretion, whether normal, hyper-acid, or of diminished acidity. The treatment of all three forms is, however, largely a moral one, since nervous influence may be at once a cause of increased or diminished HCl secretion, but this is especially true of the type attended with normal secretion. The patient must be assured that there is no organic disease and compelled to desist from self-study. Along with this his general muscular and nervous tone must be improved. He must be encouraged to take food and not to avoid it, and the moral effect of a systematic arrangement of diet is good. The neuro-tonics, strychnine, gentian, nux vomica, taken with meals are helpful, but too much medi-

cine is harmful. Occasionally the nervous sedatives, including the bromides and valerian, are of service.

Where there is scanty secretion of HCl this acid must be given according to the rules already laid down, 15 to 30 minims (one to two gm.) of the dilute hydrochloric acid fifteen minutes to half an hour after a meal. On the other hand, if there is excessive HCl, alkalies must be prescribed as directed in the section on hyperchlorhydria.

Atonic Dyspepsia.—Atonic dyspepsia is a variety of nervous dyspepsia, especially common in neurasthenics, which scarcely deserves separate description; but as the term is frequently employed some attempt should be made to direct its correct application. If used, it should be applied to cases in which delayed gastro-intestinal activity or muscular atony, with *stasis of the gastric contents*, is the characteristic feature. In such cases a considerable portion of a test meal may be withdrawn at the end of seven hours. It is probably associated with more or less deficient secretory activity, though not always. Under these circumstances, too, there is apt to be flatulent distention of the abdomen, whence the term flatulent dyspepsia and intestinal dyspepsia. A further natural consequence of such delayed mobility is *constipation*.

True gastric atony is also characterized by other symptoms which are not commonly included under those of atonic dyspepsia. Such condition undoubtedly plays a part in dilatation of the stomach, an important morbid state to be separately considered. Such atony, also, involving the cardiac orifice, favors eructation and regurgitation from the stomach, an extreme degree of which is the rare condition of rumination or *merycismus*, in which the patient regurgitates the swallowed food, oftimes voluntarily, and chews it again like ruminants. Such a power of regurgitation had the late Brown-Sequard. It is sometimes hereditary, and may be taught to others.

GASTRALGIA.

Definition.—A term applied to recurring attacks of gastric pain of great severity without discoverable organic lesion or deranged function.

Etiology.—The disease is confined almost exclusively to women, but does occur occasionally in stalwart men. It is more frequent in weak, anæmic women, and those subject to menstrual derangement, in brunettes rather than in blondes. It is especially frequent and severe about the menopause, but does not cease with it. Excessive secretion of gastric juice or hyperchlorhydria is a cause of gastralgia, but the condition is one for separate consideration.

Symptoms.—The attack may come on suddenly or with gradually increasing severity, first in the neighborhood of the ensiform cartilage, whence it radiates into the back and around the lower ribs. It is a boring, burning *pain* of extreme severity, sometimes causing fainting and collapse, relieved by pressure, such as is produced by boring the fist into the epigastrium or pressing it against some hard substance. On the other hand, it is sometimes excited by pressure. The pain is usually the sole symptom, but it may be associated with *nausea* and *vomiting*

or with nervous symptoms, such as *globus hystericus* and *unnatural hunger*. It is usually independent of exciting cause, such as the taking of food, but may be thus induced. The attack, after a variable duration of from a few minutes to an hour or more, may subside gradually or suddenly without other symptoms, though sometimes with vomiting and eructations, at others with the discharge of a large quantity of pale urine. Its most striking feature after its agonizing character is its *intermittent paroxysmal character*. Whence it has been held to be malarial in origin. One case under my care almost always began with a chill, more or less typical, and it is certain there was no malaria. The interval between the attacks varies greatly. It may be a week or it may be months.

Diagnosis.—Gastralgia is to be differentiated from intercostal neuralgia and the attacks of pain due to ulcer and sometimes even to cancer, from the gastric crises of tabes, and from biliary and intestinal colic.

In intercostal neuralgia the pain is not so severe and the paroxysms are of longer duration, while careful examination will discover its focus in an intercostal situation as compared with an epigastric. In ulcer of the stomach there is not that total intermission or longer interval of total intermission characteristic of gastralgia, while the general health of the patient with ulcer is commonly more seriously affected. This is, however, not always so, as gastric ulcer may be associated with robustness of appearance. Not so with carcinoma, which always visibly affects the general health. Careful examination will generally discover a different seat of the pain in biliary colic, while the almost invariable presence of jaundice settles the question. In a well-established case of tabes there need be no difficulty in diagnosis, but in cases where the diagnosis is not well established there may be much doubt. Here the history of attacks in comparatively early life and thence throughout life point to gastralgia. Abdominal colic has a different focus and is more apt to be associated with windy distention.

Prognosis.—True gastralgia never destroys life, but the attacks may continue to recur at intervals throughout it.

Treatment.—The severest attacks of gastralgia can only be relieved by the use of morphine, which is best given hypodermically in the smallest doses which will suffice. Exceeding care must, however, be exercised to avoid a morphine habit. In milder cases chloroform may answer the purpose, or a combination long prescribed in the clinics of the University of Pennsylvania and deservedly popular is, equal parts of chloroformi, compound tincture of cardamom, aromatic spirit of ammonia, and brandy, of which a teaspoonful may be given every half hour or fifteen minutes until relief occurs. If needed, a few drops of deodorized tincture of opium may be added to each dose to increase the anodyne effect.

Anæmia should be treated with iron and arsenic, and a change of scene is often beneficial, while sea bathing is a form of hygiene which is sometimes especially useful. The bowels should receive careful attention. If neurasthenia or hysteria be present, the rest cure, associated with massage, as described under the appropriate section, is often an efficient cure.

HYPERCHLORHYDRIA.

SYNONYMS.—*Nervous Hypersecretion of Hydrochloric Acid; Hyperpepsia.*

Definition.—Hyperchlorhydria, or an excess of hydrochloric acid in the gastric juice, is a symptom of different morbid conditions of the stomach, notably ulcer and nervous dyspepsia. In a certain number of cases, however, being the chief symptom and apparently independent of any stimulus like the presence of food, it may be studied as an independent neurosis. The term hyperpepsia suggested by Hayem is not correct, since this state is not characterized by excess of the digestive ferment, but of the chlorine element, especially hydrochloric acid. In normal digestion the total acidity as represented by free and combined HCl may be put down at 1.80 to two parts per 1000, while in hyperchlorhydria it may reach three and four parts in 1000.

Eliminating the hyperchlorhydria included under nervous dyspepsia and ulcer of the stomach, there remain two varieties of hyperchlorhydria:—

(1) Simple paroxysmal hyperchlorhydria.

(2) Continuous chronic hypersecretion, which takes place spontaneously during fasting, or, even though excited by a food stimulus, continues after the latter has ceased to act. The latter variety is also called Reichmann's disease, after him who first described it.

Etiology.—Both forms of hyperchlorhydria are more frequent in neurasthenics and emotional persons, but occur also in connection with other neuropathies, such as migraine, chlorosis, and tabes. The simple form occurs also where there is ulcer of the stomach and more rarely with cancer and gastritis, and where there is continuous operation of the same causes.

Symptoms.—In paroxysmal hyperchlorhydria there are *pain* and *epigastric discomfort*, *eructations*, *heartburn*, *thirst*, *nausea*, and even *vomiting*, *headache*, and *constipation*. The attacks may last for an hour, or may extend over several days, terminating in vomiting; or by remedial measures, such as drinking large quantities of water, which dilute the acids, or by saturation with albuminous food, with which it enters into combination. The *urine*, because of much ingestion of albuminous food, is apt to be highly charged with urea.

In the continuous form the same symptoms are present, but without intermission. The pain is even more severe, and is especially apt to come on at night; there is a *capricious appetite*, which is often large. Where the appetite remains, pain is apt to occur several hours after taking food. The *vomiting* is often copious, gaseous, and containing remnants of undigested starchy food, and is of *intensely* acid reaction. It is likely to take place several hours after a meal, also at night. The *urine is scanty* and there is *constipation*. The patients gradually emaciate and become anæmic, although they may take a good deal of food.

A very frequent consequence of continuous hyperchlorhydria is *dilatation of the stomach*, as originally pointed out by Riegel, the distinctive symptoms of which may ultimately be added. The dilatation may be caused by spasmodic contraction of the pylorus, due to the irri-

tation of the hyperacid gastric juice, or to the accumulation of fluid and undigested food in connection with a nervo-motor atony of the muscular coat of the stomach. As the dilatation increases there may ensue atrophy of the glandular structure of the stomach, and while the hypersecretion persists the hyperchlorhydria gradually diminishes and may disappear. In such event there may be an excess of fixed chlorides in the gastric juice secreted by the mucous membrane, which is, however, incapable of elaborating hydrochloric acid. Gastritis is also a result of hyperchlorhydria and contributes further to the symptoms, especially pain.

Diagnosis.—A positive diagnosis of hyperchlorhydria can only be made through analysis of the gastric contents. This is done in the sixth hour after a test dinner with a view to discovering the presence of an excess of hydrochloric acid. The same symptoms may, indeed, be caused by organic acids, while the hydrochloric acid is in normal amount. If the stomach is washed out in the evening and the next morning, no food being ingested in the meantime, the contents are expressed and found to be hyperchlorhydric, the condition is one of continuous hyperchlorhydria.

Prognosis.—The prognosis of simple hyperchlorhydria is favorable, that of the continuous form is grave, the disease being incurable after a certain stage has been reached. It becomes, therefore, important to treat the simple form promptly and intelligently before it passes over into the continuous form.

Treatment.—The indications for treatment in hyperchlorhydria are evident. Their measure should, however, be based upon the estimation of the acidity of the gastric contents. They are, first, to neutralize the excessive acid secretion and, second, to restrain its continuance.

The first indication is met in two ways :—

(1) By saturating the acid by nitrogenous food, and

(2) By the administration of alkalies.

The former is fulfilled by the use of meat and milk diet. It has, however, its limits, because when the tendency to acid secretion exists, it is often maintained even after that present is combined with any albuminous food that may be in the stomach. Hence it is that the pain is felt some hours after a meal when the albumin is digested. Since there is a limitation to the ingestion of meat its use must be supplanted by antacids, which farther neutralize the effect of the acid. The alkali most frequently employed for this purpose is sodium bicarbonate, though calcined magnesia is in some respects better because of its greater saturating power. An idea of the amount of hydrochloric acid secreted may be obtained from the fact that probably 500 gm. (*circa* pint) of gastric juice are secreted in an hour, four or five liters (8.4 to 10.5 pints) in three hours, and should such gastric juice contain three parts of HCl in 1000, a proportion often exceeded in hyperchlorhydria, there would be some 12 to 15 gm. (180 to 225 grains) of the HCl to neutralize. Since one gram of hydrochloric acid requires 1.48 gm. sodium carbonate, 20 to 25 gm. (300 to 375 grains) would be required to neutralize the whole amount of acid, a large quantity. The sodium carbonate should be administered some time after meals, just before the time the pains are

expected, dissolved in water or milk, or in capsules or cachets. The doses should be sufficient to counteract the acidity, 10 to 20 grains (0.66 to 1.3 gm.) or more. The quantity of carbonic acid evolved in the stomach is sometimes inconvenient. Smaller doses of calcined magnesia suffice, and it is surprising that its use is not more general. It has the disadvantage of being insoluble in water, but not only are smaller doses sufficient, but there is also absence of carbonic acid evolution. Especially where there is constipation it is indicated.

Other alkalies may be used, such as the potassium salts, and the officinal liquor potassæ in 15 to 30 drops (0.8 to 1.7 c. c.) in milk may be used with advantage. The benzoate of sodium may be prescribed where antisepsis is required or fermentation is present in ten-grain (0.66 gm.) doses. Lime water is also useful, but large doses are required, as its neutralizing power is small. One-half ounce to an ounce (15 to 30 c. c.) or more should be given. Lime dissolves more largely in saccharine solution than in pure water, and larger doses may thus be given in smaller bulk. Dilute alkaline mineral waters, such as Vichy or Vals or Contrexville, may be used during a meal, but it is to be remembered that an alkali given fasting promotes the secretion of gastric juice.

Of course, it is better, if possible, to prevent the excessive secretion of the juice. For this purpose sodium sulphate has been recommended, more particularly in the shape of Carlsbad water. Or the sodium sulphate may be dissolved in Vichy, say 45 to 90 grains (three to six gm.) in a glass. It is given in the morning before breakfast, or if necessary may be given before the other meals.

Diet.—While the medicinal treatment of hyperchlorhydria is in most cases indispensable, the diet is equally important. It has already been said that theoretically a meat and milk diet are indicated, because they consume in their digestion the excess of HCl. On the other hand, the starchy foods are but imperfectly digested. Imbibing the acid secretion, they swell up but do not dissolve, while they favor on the other hand irritating acid fermentation. Others object to meat diet because of its overstimulating effect on the acid secretion and recommend vegetables instead. This is, however, fallacious, and experience sustains the verdict in favor of meat and a minimum of starchy foods. It should be finely cut and well masticated, while meat powder may be substituted. Milk should be the drink, though the alkaline mineral waters may be taken at meals. In extreme cases, a pure meat diet, the meat raw or nearly so, finely minced and spread on bread, may be necessary. A meal may consist of about 3½ ounces (100 gm.) of raw meat, a couple of thin slices of stale bread, a little butter, and a glass of plain water or weak alkaline water, such as Vals or Vichy. Or an exclusive milk diet may be tried, in which event the milk should be well alkalized or peptonized. To these are added, as the case improves, raw or powdered meat or meat juice and eggs and later still starchy foods may be tentatively given, associated with diastasic malt. Where acid secretions and undigested residue of food remain in the stomach long after the ingestion of food the organ should be washed out. This may be done two or three times a week or even daily.

In these cases over-stimulation of the stomach, induced especially

by alcohol, pepper, mustard, and other condiments, should be avoided. In like manner coarse food of any kind is contra-indicated in these cases. On this account constipation is sometimes best treated by enemata in order to avoid the ingestion of irritating food into the stomach.

Of medicines other than those intended to meet the symptoms, arsenic in the shape of Fowler's solution is sometimes efficient. Long courses of it should be practised, but large doses are not often allowable because of the irritation excited by them. Silver nitrate may also be employed in doses of ¼ grain (0.0165 gm.), in which dose it is sometimes sedative when given on an empty stomach.

Anorexia Nervosa.—This term is applied to a condition in which absolute loss of appetite is the chief and characteristic symptom. Associated with this are, naturally, great debility, shortness of breath, dizziness, constipation, and sometimes headache; rarely also vomiting; sooner or later emaciation. In women, in whom the symptoms usually occur, there is cessation of the catamenia. The name was suggested by Sir William Gull.

Prognosis.—This is favorable, cases being rarely, if ever, fatal.

Treatment.—The usual tonic measures are apt to fail to excite appetite in these cases, and nourishment must often be given either by the rectum or by forced feeding. This is done as follows: A short rubber tube, long enough to reach just below the cricoid cartilage, is introduced as directed on p. 225. A bottle or funnel should be attached, and from this liquid nourishment is slowly introduced. This may be milk, plain or peptonized, broths or eggs, Murdoch's or Mellin's food. Estimating that 3½ ounces (100 gm.) of albumin, five ounces (150 gm.) of fat, and ten ounces (300 gm.) of carbohydrates are a sufficient amount per diem, Wiessner recommends one quart (one liter) of milk, two ounces (60 gm.) of butter, six eggs, and 3½ ounces (100 gm.) of sugar to be mixed and warmed while stirring. One third of this amount is introduced three times daily. The food is usually easily digested, for it is not the digestion which is at fault, but the appetite, and the patient, encouraged by the result, is apt to eat for herself.

Nervous Vomiting.—A form of vomiting resulting from direct or reflex irritation of the centres presiding over vomiting, and independent of anatomical lesion in the stomach. It, like nervous dyspepsia, is probably an expression of a general irritable condition of the gastric nerves,*—a manifestation of a general neurasthenia. It has been suggested that the exciting cause is some irritating leucomain of as yet unknown nature.

Etiology.—Its subjects for the most part are hysterical and neurasthenic women, more often of dark complexion; but it is also the result of disease of the brain and its membranes and of the medulla and spinal cord, such as tabes dorsalis, when it may take the place of other symptoms of gastric crisis. It is apt to be associated with headache and

* Garland, G. M., Transactions of the Association of American Physicians, Vol. IV., p. 220, XX.

gnawing sensations in the stomach, with diseases of the kidneys, liver, uterus, and other distant organs. While more usual in adults, it may also occur in children. Pure nervous vomiting is especially seen in neurotic families in which there is a tendency to nervous disease, including insanity and epilepsy. On the other hand, the absence of the hysterical temperament is often conspicuous. It affects rather the upper classes.

Diagnosis.—This is based, in the first place, on the exclusion of those organic diseases of the stomach which cause vomiting, and, in the second place, on the presence of any one of the affections named as possible causes.

Symptoms.—Especially characteristic of nervous vomiting are the *absence of nausea*, the *suddenness* of the act of *vomiting*, and the absence of the straining. More rarely there is nausea. The *appetite* is good and the vomiting generally follows a meal, but it may also occur at irregular intervals. In the absence of organic nervous disease the patient may be well-nourished. There may also be *constipation*, *headache*, *dizziness*, and *epigastric pulsation*. Intense *acidity of the vomited matter* may be present. To this condition Rosenbach has applied the term *nervous gastroxynsis*. In one of his cases the HCl reached four per cent. In the typical form, however, the vomitus is not abnormally acid, and in this respect it differs from acid dyspepsia and Reichmann's disease. The duration of the vomiting varies. It may be a single act or it may last for twenty-four hours.

Prognosis.—Except, too, when associated with organic nervous disease this is ultimately favorable. George M. Garland* reported a fatal case of apparently pure nervous vomiting. At the autopsy the mucous membrane of the stomach was found thin and reddened on its inner surface with minute hemorrhagic points and slight interstitial nephritis, but the latter was too insignificant to have had any effect, and the gastric changes were probably secondary, so that the case may be regarded as purely neurotic.

Treatment.—Where vomiting is the result of organic nervous disease the fundamental treatment must be that of the disease itself. Temporary relief may be extended in such cases from the same measures which are indicated in pure nervous vomiting without organic lesion. The most efficient of these are those which make a profound nervous impression. Such, preëminently, is the blister to the epigastrium. The suddenness and irregularity of the vomiting make it almost impossible to provide against a given event. So that ice, internal or external, sinapisms, dry cupping, and similar measures available in continuous vomiting or vomiting preceded by nausea, are scarcely available. Where, however, circumstances permit their employment they should be used.

Nervous sedatives, including the bromides and valerian, may be used, but hypodermic injections of morphine are often necessary and are usually very efficient. When practised by the physician only, they become a safe measure. Rectal alimentation should be employed where the vomiting is obstinate, and has apparently saved life in many instances. Where there is nervous gastroxynsis, lavage with warm water may be

*Garland, G. M., Transactions of the Association of American Physicians, Vol. IV, p. 205.

used with advantage as recommended by Rosenbach. The headache, etc., apt to be associated with this form is at once relieved.

GASTRIC AND DUODENAL ULCERS.

SYNONYMS.—*Ulcus ventriculi pepticum; Peptic Ulcer; Simple or Round Ulcer.*

Etiology.—There is probably more than one mode of origin of gastric ulcer. It may have its origin in mechanical injury associated with feeble nutrition, which permits the gastric juice to digest out the mucous membrane to various depths, resulting in the formation of an ulcer. Such mechanical injury may be due to pressure exerted in the course of one's occupation, such as shoemaking, washing, tailoring, and the like, in which pursuits the costal cartilages are pressed against the stomach. The second of these conditions, for it is likely that neither would be alone sufficient to produce the lesion, is produced by such states as anæmia, chlorosis, heart disease, Bright's disease, and the like. Over-distention of the stomach, it is claimed, may be a predisposing cause by interfering with its proper nutrition and thus favoring the action of the gastric juice.

Thrombosis and embolism have been held responsible for a certain number of cases of ulcer since Virchow called attention to such causes. Embolism of the gastric blood-vessels is extremely rare, but thrombosis is a not infrequent result of obstinate vomiting, as is also punctiform hemorrhage. The stasis of circulation thus resulting affords favorable foci for the solvent action of the gastric juice, and certainly no theory explains so satisfactorily the crater shape of many gastric ulcers. Böttcher ascribes ulcer of the stomach to micrococci, numbers of which have been found by him in the margins of gastric ulcers. The well-known clinical fact that the gastric juice in ulcer of the stomach exhibits intense acidity, while traumatic ulcers of the stomach produced under ordinary circumstances tend to heal promptly, has led to the suggestion that excessive acidity plays an important rôle in the causation of ulcer. The same causes operate to produce the duodenal ulcer.

Women are much more frequent victims than men. While both the very young and the very old are commonly exempt, the period being between seventeen and twenty-five, gastric ulcer has been found in infants, and adults as old as sixty. Duodenal ulcer, on the other hand, is more common in males, in the proportion of 178 to 41, in the combined statistics of Kraus, Chvostek, Lebert, Trier, and Osler. The last observer found it once in a boy of twelve. Its association with extensive superficial burns and tuberculosis should be mentioned.

Morbid Anatomy.—Gastric ulcer must be distinguished from post-mortem softening or digestion which is found after death in stomachs in which gastric juice happens to be present at the moment of death. In this there may be erosion of the superficial mucosa, but nothing comparable to ulcer. The seat of post-mortem softening is more commonly the fundus and posterior surface, where the gastric juice naturally collects.

The typical gastric ulcer is circular in outline, often with sloping, clean-cut sides, furnishing a crater or truncated cone-shape, with the

broad end superficially placed, corresponding to that of an infarcted area due to embolism or thrombosis. The term "punched out" has long been applied to characterize the appearance of a gastric ulcer. The sides are not always, however, smooth, being sometimes uneven or "terraced." Very rarely ulcer may be multiple. It is far more frequent on the posterior wall of the stomach near the lesser curvature. W. H. Welch's extensive studies of hospital records furnish the total of 783 cases, of which 288 were in the lesser curvature, 225 on the posterior wall, 95 at the pylorus, 69 on the anterior wall, 50 at the cardia, 29 at the fundus, and 27 on the greater curvature. The duodenal ulcer is found just outside the pylorus, but may occur as low down as the biliary papule. It presents the same appearance as the characteristic gastric ulcer.

The floor of the ulcer is usually the muscular coat, but it may be the serous coat, which is sometimes perforated so that the floor may be formed by an adjacent organ to which the stomach has been glued by adhesive inflammation. The ulcer is usually small, not larger than a pea, but it may be ten or even 15 cm. (four to six inches) in diameter, covering the whole lesser curvature and part of the anterior and posterior walls. Ulcers may heal, leaving a cicatrix, which if large causes contraction and deformity, distorting the organ even to an hour-glass shape and producing stenosis of the pylorus. It is not unusual to find healed ulcers at autopsies. Or the ulcer may perforate, causing fatal peritonitis when in the anterior wall, or if apposed to neighboring organs these may be burrowed into. Thus the pericardium and left ventricle, the spleen, the head of the pancreas, the left lobe of the liver, the gall bladder, the omental tissues, the pleura, and even the lungs have been invaded; while fistulous communications have been formed with the duodenum, the colon, and even the external area in the neighborhood of the umbilicus. Perforation of the posterior wall opens the lesser peritoneal cavity and may perforate the pleura, producing subphrenic pyopneumo-thorax.

It is not unusual to see at the bottom of an ulcer an eroded blood-vessel from which there has been a fatal hemorrhage. The vessels invaded may be the gastric artery of the lesser curvature or the splenic artery from the posterior wall, or in the case of a duodenal ulcer the pancreatico-duodenal artery, or it may be the hepatic artery and even the portal vein. Small aneurisms have been found in the floor of ulcer.

Symptoms.—The most prominent symptoms of ulcer of the stomach are *pain*, *tenderness*, *vomiting*, *hemorrhage*, and sometimes a *tumor*, but none of these is invariably present. They require to be separately considered.

Pain with tenderness is the most constant symptom. It is characteristic of the pain of ulcer of the stomach that it occurs almost immediately after taking food, especially after cold or hot and indigestible food; but it may also occur on an empty stomach, that is, several hours after a meal, when all food has disappeared. The latter pain is, however, of a different kind, being of a gnawing character, and is even temporarily relieved by taking food. The pain typical of ulcer, a gastralgia, coming on from ten minutes to half an hour after eating, is perhaps due not so much to the presence of the food as to the irritant effect of the acid gastric juice called out to digest it. It varies greatly in severity, and is further char-

acterized by having a definite centre of greatest intensity, commonly in the epigastrium near the xiphoid, less often at a point behind the shoulders, from which it radiates in all directions. A change of position also sometimes increases it, especially changing to the right side, probably due to the irritation of the ulcer by the moving gastric contents. The paroxysms are sometimes of indescribable severity, requiring the hypodermic use of morphine to relieve them, though they may also be relieved at times by a full dose of sodium bicarbonate, the effect of which also explains their immediate causation. Commonly increased by pressure, it is sometimes relieved by it, and the patient will bend over, pressing his fist into the epigastrium or leaning over the back of a chair to secure relief.

Tenderness on pressure is a characteristic symptom, apart from the paroxysms of pain, and in order to guard against it the patient may wear the waistband low. Boas has devised an instrument by which circumscribed pressure may be conveniently induced and diagnosis facilitated. It is, however, necessary to exercise care in such pressure, as perforation may be produced. The tender point is more frequently an inch or two above the umbilicus. In cases of ulcer of long duration, palpation may recognize a *tumor*, the result of inflammatory thickening in the vicinity, and I well remember a case where in consequence of the distinctness of the tumor I diagnosticated with some confidence a cancer of the pylorus, and a few days later the patient died of a hemorrhage from the stomach. An autopsy revealed extraordinary thickening of the pylorus, penetrated to a great depth by an ulcer, at the bottom of which lay a little perforated artery whence came the fatal hemorrhage.

And this brings us to *hemorrhage*, a most valuable sign of gastric ulcer. Given a copious hemorrhage of pure red blood from the stomach, with the symptoms described or even no other symptoms, it can scarcely be due to any other cause ; since, although cancer gives rise to hemorrhage, it is made up of blood and mucus ; the blood is usually less copious, while a cancer with hemorrhage rarely fails to furnish also the other symptoms of cancer. In a few instances in ulcer the hemorrhage is small, when, of course, the diagnosis becomes more difficult. Where the hemorrhage is large, blood quite black is found also in the stools. Indeed, sometimes the presence of blood in the stools is the first intimation of gastric hemorrhage. Especially is this the case when the ulcer is duodenal. Hemorrhages from ulcer are also often recurrent, and result at times in intense anæmia of the subject. They are not rarely fatal, more frequently syncopal, bringing their subjects to the verge of the grave, from which there are often also surprising recoveries. Vicarious hemorrhage in menstruating women is to be remembered as a possible event, but the hemorrhage is not usually copious and its association with amenorrhœa aids in clearing up doubt. Hemorrhage occurs in more than half the cases, at least in hospital practice, since the severest cases come to hospital. In private practice the proportion is smaller.

The milder *symptoms of dyspepsia* may also be present in various grades, including a sense of *fulness in the epigastrium*, *acid eructations*, *heartburn*, *loss of appetite*, and even *vomiting* of acrid acid matters.

When vomiting is present it occurs usually soon after the ingestion of food, about the same time as the pain.

Patients with gastric ulcer lose in weight and become gradually *anæmic* quite independently of hemorrhage, as evidenced by a blood count which sometimes finds the red corpuscles less than a million to the cubic millimeter. Chemical examination of the stomach contents after a test meal almost invariably shows an increase of HCl. Exception sometimes occurs when chronic catarrhal gastritis is associated.

Finally, it is to be remembered of gastric ulcer that it is often entirely *latent*, quite without symptoms during life, and recognized for the first time at necropsy, when also, as already stated, healed ulcers are sometimes found.

Course and Termination.—The course of ulcer is usually slow, sometimes very protracted. One case confirmed by autopsy came under my treatment which had lasted twenty years. A few cases are acute and rapidly fatal. The symptoms of gastric ulcer quite frequently disappear and after a time, even considerable time, recur, giving rise to the so-called recurrent forms.

Diagnosis.—This in some cases is easy, in others difficult or impossible. If hemorrhage of the kind described is present in connection with the other symptoms named, it affords conclusive evidence of ulcer, but in its absence there must often remain doubt. Outside of hemorrhage the most characteristic symptom is pain, and only in gastralgia and tabes dorsalis do such pains occur.

In gastralgia, as in ulcer, hydrochloric acid is increased and the question often becomes a most difficult one to settle. In gastralgia, however, the general health of the patient is less severely affected, there is less chlorosis or menstrual derangement, and the pain has a less definite relation to taking food, indeed, is often relieved by food, while in ulcer the symptoms of dyspepsia are more constant. There are longer intervals between the attacks in gastralgia. Above all, in ulcer there is tenderness on pressure between the attacks of pain, a symptom absent in gastralgia, while pressure always relieves the pain of the latter. Indeed, in gastralgia there are generally no dyspeptic symptoms between the attacks. If palpation recognizes a hardening there is further reason to believe the case is one of ulcer. We may look for assistance from the standpoint of etiology. Given the causes of ulcer, especially valvular heart disease with possible embolism, the vomiting which produces thrombosis, or the occupations which favor gastric ulcer, their import should be recognized. Gastralgia occurs in neurotic individuals, those subject to hysteria, neuralgia, and uterine disease. V. Leube has called attention to an electrical test between gastralgia and ulcer—viz., if during digestion an electrical current, especially with the anode as a testing-pole, be applied and the pain disappears completely, it is indicative of gastralgia; if, however, it does not cease it may be either gastralgia or ulcer. Only the positive effect, the sudden cessation of pain on the application of the current, is of diagnostic value.

In tabes the gastric crises are almost identical with the severe gastralgic attacks of ulcer. But in tabes the appearance of good health is preserved, while it is not long before the distinctive symptoms of the

disease show themselves if they are not already present. In tabes the extreme acidity of the gastric contents characteristic of ulcer in most instances is wanting.

In rare cases intercostal neuralgia may be mistaken for ulcer, if there be pain in the epigastrium associated with accidental dyspeptic symptoms. But in this affection painful points will also be found in the course of the intercostal nerves, while if a large fold of the abdominal wall be raised, tender points will be found in it.

From cancer of the stomach, ulcer sometimes is distinguished with difficulty in the absence of the more distinctive symptoms of the former disease. Heretofore, much reliance has been placed on the absence or extreme diminution of free hydrochloric acid in cancer as contrasted with its excess in ulcer. It has, however, been realized also that it occasionally happens that the association of chronic catarrhal gastritis with ulcer may work reduction of HCl. The recent researches of Boas have, if confirmed, added a very much more reliable diagnostic sign in the invariable presence of lactic acid in cancer and its constant absence in ulcer, and, indeed, under all circumstances in which it has not been introduced from without. Other facts to be weighed in the balance as to the existence of cancer are a palpable tumor, the greater age of the patient, always over thirty, the extreme emaciation and cachectic appearance, the intermittent vomiting of large quantities of accumulated ingesta, sometimes of blood mixed with mucus, or blood presenting the "coffee-grounds" character as contrasted with the bright, clear blood of ulcer.

Rarely is duodenal ulcer distinguished before death from gastric ulcer. The former may be suspected when pain is toward the right parasternal line rather than in the epigastrium or to the left of it; also, if the blood be discharged by the bowel rather than vomited; or when there is jaundice. The latter symptom is, however, more constantly associated with biliary colic, which has also been mistaken for ulcer. In biliary colic the liver may be enlarged and tender and the gall bladder distended, while the vomiting which attends it as well as ulcer is much less acid in reaction.

Attempts to locate the ulcer still more precisely have generally proved fruitless. Even when a single painful, unchanging, circumscribed spot has been noted, the apparent seat so rarely coincides with the actual seat that little encouragement is afforded further attempt. When pain immediately succeeds deglutition, especially of solids and hot and cold liquids, there is some reason to believe the ulcer is in the neighborhood of the cardia, but it is by no means conclusive.

Prognosis.—Not only the disappearance of symptoms, but also the discovery of numerous healed ulcers at autopsies of patients dying from other causes attest the fact that recoveries are not infrequent. Death is caused, as a rule, by hemorrhage or perforation, the latter followed by fatal peritonitis. It is said that six per cent. of all cases terminate in perforation, which in the vast majority of cases is followed by death in a few hours.

Treatment.—The indications for treatment are evident and in the main easily fulfilled. It is plain, in the first place, that food which taxes the secretory or motor function of the stomach is harmful, and that

recovery will be still more likely to occur if the stomach can be placed at total rest, a condition easily met by rectal alimentation. It is clear, too, that absolute rest of body further fulfils such conditions. The greater the stringency with which these measures can be carried out, the greater the chances of a cure, which is always possible. It goes without saying that all solid food should be disallowed. The typical nourishment in my experience is peptonized milk, which should be given at stated intervals, the quantity adapted to the urgency of the symptoms, say two oz. (60 c.c.) every two hours, though even this amount may have to be reduced in serious cases, to be increased as danger subsides and the appetite of the patient demands it. Beef peptonoids and egg albumin may be substituted for milk, or they may be conjoined with it. So, too, when extreme danger of repeated hemorrhages is present, it is a warning that rectal alimentation alone should be relied upon; for which purpose, too, peptonized milk is also the best nutrient. Great care must be exercised in the use of enemata not to overdo the thing and exhaust the toleration of the bowel. To this end they should not be given oftener than once in four or six hours, and should not at first, at least, exceed four ounces. This quantity, if borne, may be increased to six ounces. The various meat peptones, bouillon, or beef juice may be substituted or alternated with the peptonized milk, or an egg may be beaten up with the milk, though such addition is not often necessary. In this way patients may be nourished for weeks with peptonized food, but it is rarely necessary to continue the rectal alimentation for more than a week or ten days. As the hemorrhage and vomiting cease the stomach may be tested, first with small amounts of peptonoids, gradually increased, and for a time the two methods may be pursued jointly, feeding by the mouth being increased, while that by the rectum is gradually withdrawn. Plain milk and beef juice may be substituted for peptonized milk, and various thin gruels made with flour may be used as a change is demanded.

Lavage, which is of such signal service in chronic gastric catarrh, is hardly safe in ulcer on account of the danger of producing perforation, but in some cases where vomiting has been obstinate lavage has been found beneficial. To arrest the vomiting, it is safer to rely on rectal alimentation, though the usual remedies may be tried.

Medicines are not to be decried in ulcer of the stomach. Silver nitrate maintains the reputation it has so long enjoyed in the treatment of gastric ulcer. One quarter of a grain (0.0165 gm.) three times a day or $\frac{1}{6}$ grain (0.011 gm.) four times are the usual doses, given on an empty stomach. Of late I have been giving it by preference in solution in about two ounces (60 c. c.) of water. If there is pain, the extract of opium should be combined in pill in the same or larger doses, but should be dispensed with as soon as not needed. The extract of belladonna in small doses may be substituted as the opium is withdrawn. Its anodyne effect is perhaps slight, but it has a good effect upon the bowels. Local measures may be employed to relieve the pain, such as warm poultices and other hot fomentations to the epigastrium, and where more potent measures are needed the hypodermic use of morphine can be employed.

The hemorrhage requires also to be met by remedies. At the moment of its occurrence tannic acid in 15-grain (one gm.) doses,

administered every fifteen minutes, should be given until the bleeding ceases. In the absence of this drug alum may be given, dissolving a teaspoonful in a glass of water, of which one fourth should be given at short intervals. Pieces of ice may also be swallowed. After the attack is controlled the persulphate of iron in doses of ¼ to ½ grain (0.0165 to 0.033 gm.) in a pill three or four times a day may be used to prevent recurrence.

The acidity which is characteristic of the secretion in gastric ulcer has sometimes to be met, and for this purpose full doses of sodium carbonate or bismuth subnitrate, 15 to 30 grains (one to two gm.), may be given. When aperients are needed, as is sometimes the case, the Carlsbad salt becomes suitable because of its alkalinity and adaptation to the catarrhal state often associated with ulcer. A teaspoonful may be given in the morning dissolved in a glass of warm water, or a tablespoonful may be added to a pint of warm water and taken in divided portions during the day. Small doses of magnesium sulphate may be substituted, though as a rule the bowels should be regulated by enemata rather than purgatives.

The resulting chlorosis or anæmia may be treated with iron and arsenic. Of the former the neutral preparations are to be preferred, of the latter Fowler's solution, because of the easy regulation of the dose. Large doses of iron should not be given, since the excess of such doses remains unabsorbed, astringing and irritating the alimentary canal. The tincture of the chloride, so valuable usually, is especially contra-indicated, because it increases the acidity of the gastric juice and thus favors the solution of the gastric wall.

CANCER OF THE STOMACH.

Synonyms.—*Carcinoma ventriculi; Gastric Cancer.*

Etiology.—Little definite is known of the etiology of cancer. Heredity is an acknowledged factor, though less potent than is commonly supposed. W. H. Welsh * was able to trace cancer, or at least a family history of it, in 242 out of 1744 cases. There is some evidence to show that abuse of the stomach by eating and drinking may be influential in causing the disease, though it is not conclusive. The same is true of the depressing emotions. There is better reason to believe that ulcer is a predisposing cause, since autopsies have disclosed cancer developing in the floor of ulcers and in cicatrices.

Gastric cancer is a disease of mature life, three-fourths of all cases occurring between the fortieth and seventieth year. One of my patients was thirty-two when he first consulted me and died just one year later. Struempell has seen cases between twenty-two and twenty-five. The disease is slightly more frequent in men than in women.

Pathology and Morbid Anatomy.—After the uterus, the stomach is the organ most frequently attacked by cancer, a little more than one-fifth of all cases of primary cancer being found in this organ,—according

* "System of Medicine by American Authors," Vol. II, Philadelphia, 1886.

to Welsh, 21.4 per cent., from an analysis of the very large number of 30,000 cases. It is far more common in the pyloric end and on the lesser curvature, 1300 cases collected by Welsh being distributed as follows: pyloric region, 791; lesser curvature, 148; cardia, 104; posterior wall, 68; whole or greater part of the stomach, 61; multiple, 45; greater curvature, 34; anterior wall, 30; fundus, 19. Every variety of cancer is found in the stomach in the following order of frequency:—

(1) Cylinder-celled epithelioma, most frequent at the pylorus.

(2) Medullary or soft cancer, most frequent at the smaller curvature.

(3) Schirrus, at the pylorus and in the smaller curvature, causing especially stenosis of the pyloric orifice.

(4) Colloid, diffuse infiltration with a tendency to spread to the peritoneum and adjacent organs.

(5) Melanotic.

(6) Squamous epithelioma, near the cardia. All the forms start from the gland cells of the mucous membrane.

The medullary variety is prone to ulcerate and to form extensive fungoid ulcerated surfaces, from which there may or may not be hemorrhage. It may be associated with scirrhus. While nodular outgrowths are usual, the cancerous tissue may infiltrate the walls, producing diffuse thickening.

Secondary cancer of the stomach is an occasional event; in 17 out of 37 cases, according to Welsh, secondary to primary cancer of the breast. I have met one case succeeding epithelioma of the lip. Much more frequently, primary cancer of the stomach is a cause of secondary cancer elsewhere, most often in the adjacent lymphatic glands, which were the secondary foci in 551 out of 1574 cases collected by Welsh; the liver was involved secondarily 475 times; the peritoneum, omentum, and intestine, 357; pancreas, 122; pleura and lung, 98; spleen, 26; brain and meninges, 9; other localities, 92; among the latter is to be included adjacent integument, especially about the navel.

Marked changes in the size, shape, and position of the organ occur as the result. Most common is dilatation, sometimes due to pyloric obstruction. Medullary cancer, on the other hand, is apt to produce a reduction in the size of the stomach and its cavity. A reduction in size may attend obstruction at the cardiac orifice because of disuse of the organ while the œsophagus itself may be dilated. Further allusion will be made to extraordinary dislocation of parts of the organ in treating of symptoms. Adhesions may also form between the stomach and adjacent organs and between it and the anterior abdominal wall. Peritonitis may occur, also perforation into an adjacent organ, as the transverse colon and even small intestine.

Symptoms.—The initial symptoms in almost every form of cancer of the stomach are those of indigestion, including *anorexia*, *eructations*, *vomiting*, *constipation*, *discomfort*, and *pain*, more rarely *acidity*. These are present for a variable time before a more serious condition is suspected. Increase in the severity of symptoms despite the use of remedies, progressive *debility*, *emaciation*, and *cachexia* invite closer examination, which may or may not result in the discovery of a *tumor*. Before a tumor

is recognized there is often tenderness, which follows sooner or later if it does not precede tumor. Cachexia and wasting may also be present a long time before the tumor is discovered.

A *chemical examination* of the *gastric contents* after a test meal may disclose the absence of free and combined hydrochloric acid or a minimum of it, and Boas' recent results are confirmatory of the persistent presence of lactic acid in decided quantity, to which, as well as the absence of hydrochloric acid, attention was originally called by von der Velden. As to hydrochloric acid, it must be remembered that it is also diminished in gastric catarrh, in atrophy of the mucous membrane, amyloid degeneration, and even nervous dyspepsia at times, while in rare instances it happens that hydrochloric acid is increased in cancer. The motor as well as the secretory and absorbing function will be found impaired, undigested food being found long after the seven hours' limit. Such motor delay characterizes more particularly the pyloric situation of cancer with its resulting obstruction.

At a later stage periodic *vomiting* of large quantities of fluid containing the ingesta of hours and even days previous is a characteristic symptom, and a dilated stomach may now be easily demonstrated. The vomitus may also contain blood, and that peculiar mixture of blood and gastric juice which is called "coffee grounds" vomit. If owing to their disintegration the microscope does not recognize blood discs, Teichmann's hæmin crystals may be easily prepared as directed on p. 71, foot-note. The vomited matter is sometimes very foul-smelling, as are also at times the eructations. Vomiting is by no means an invariable symptom, though even when there is no vomiting nausea is commonly present. The absence of vomiting generally means that the cancer is not at the pylorus. It may be at the middle belt, the fundus, or at the cardiac end. When at the latter point there is almost always difficult and painful deglutition.

By this time the patient is emaciated, anæmic, *cachectic*, with a peculiar yellowish, sallow, swollen appearance, and now a *tumor* is commonly easily recognized by palpation. Very interesting is the varying situation of the tumor as well as at times its great mobility. Almost never, in my experience, is the tumor found in the normal situation of the pylorus, even when the patient is lying on his back, but rather in the neighborhood of the umbilicus, a little to the right or left. It is the weight of the tumor which drags it out of the normal position of the pylorus, and it may be found even lower down toward the symphysis pubis, as in a case of Struempell's. The tumor itself may be fixed in the position it assumes, or it may be freely movable. Its location is usually uninfluenced by breathing, and in this respect contrasts with tumors of the liver and spleen. It rarely gives a positive dull note on percussion, rather a muffled note. In a certain number of cases no tumor can be detected throughout the whole course of the disease. Especially is this the case when the disease is toward the cardiac end. A rotary motion is sometimes characteristic of the tumor.

Toward the end of life *œdema of the legs* and *ankles* often appears and an intensity of cachexia, which simulates pernicious anæmia—in fact, even furnishes the blood changes characteristic of this affection—with

extreme weakness and death. The *urine* is often scanty and may give a decided reaction for indican. In a few cases a *febrile movement* makes its appearance with *chills* and *sweating* at intervals, probably due to intercurrent inflammation. To the symptoms are often added those of *secondary cancer*, especially of the liver, including enlargement of this organ and jaundice. The signs of secondary cancer elsewhere than the liver should be sought. The duration of cases of gastric cancer is from one to two years, it may be less, especially if the cancer is engrafted on a pre-existing ulcer.

Diagnosis.—This is generally easy if time be allowed for the study of a given case. Ulcer is perhaps the disease which furnishes most difficulty, especially as cancer may succeed upon it. On the other hand, the earliest symptoms of gastric cancer are also those of gastric catarrh, which in many cases is mistaken for cancer. The pain and the peculiar intermittent vomiting are the first distinctive signs, and while coffee-grounds vomit may occur whenever moderate quantities of blood are poured into the stomach and mixed with gastric juice, the causes other than cancer are rare. The copious hemorrhage of ulcer gives bright red blood. Bloody vomiting is by no means always present in cancer. To the symptoms described are soon added the emaciation and cachexia and the palpable tumor, more evident after the stomach has been emptied out by vomiting or washing. In the meantime, however, the gastric contents will have been examined, and furnish their quota of information, not pathognomonic, but contributory. Very rarely does it happen that in the vomitus or washings of the stomach we obtain particles of morbid growth whose examination will disclose the structure of cancer.

There is not usually much difficulty in fixing the location of the tumor supposed to be in the stomach. If there is doubt, it may be eliminated in part or altogether by filling the stomach with liquid and noting the effect upon the tumor.

In one instance I mistook cancer of the gall bladder for cancer of the stomach, though I scarcely think it would happen again. The chief reason was because the tumor due to cancer of the gall bladder was in the situation where the tumor of the pylorus ought to have been. There was jaundice and tenderness in the hepatic region, there were no signs of dilatation of the stomach, and the mistake was scarcely excusable. There is usually less interference with digestion in cancer of the gall bladder, no mobility of the tumor, and often suppuration with incident fever.

The distinction of gastric from pancreatic cancer demands some consideration. The tumor may be in the same position, but in a large proportion of cases of cancer of the pancreas there is jaundice. The tumor of a pancreatic cancer is often inaccessible. In the latter there are also symptoms of indigestion, like those of gastric cancer, but there is often also diarrhœa, and frequently the liquid stools contain oil. Such diarrhœa may be checked for a time by ordinary remedies, but in a few days the liquid discharges seem to burst through a barrier which held them temporarily in check. The pancreatic tumor if felt is also more immovable.

Tumors of the liver and spleen are continuous with these organs,

while the gastric tumor is generally easily separated from them by palpation or an intervening tympanitic area. A tumor of the transverse colon may occupy much the same position in the abdomen as one of the stomach and be also quite movable. The filling of the colon and stomach with water or air may also be availed of in diagnosis. As the growth in the intestine increases, obstruction may result and the tumor increase by the accumulation of fecal matter behind the stenosed portion. A rare complication increasing the difficulty in diagnosis is adhesion between the bowel and stomach, restricting motion and possibly causing perforation, through which fecal matter may enter the stomach. Still more difficult, nay, even impossible, in most instances, is the distinction between duodenal and gastric cancer. The absence of hydrochloric acid would point to gastric cancer, though such absence, being due to atrophy of the gastric tubules caused by dilatation, may also occur in obstructive duodenal cancer. The acid might also be neutralized by regurgitated bile, regurgitation being favored by the stenosis of the gut. The presence of jaundice would point to duodenal cancer.

Gastric tumors may be confused with omental tumors, which may also cause dyspeptic symptoms. But the omental tumor is usually a more nodular, uneven tumor, and is sooner or later associated with peritoneal effusion. Finally, every tumor of the stomach is not a cancerous tumor, although most of them are. I have already mentioned my experience with a thickened pylorus associated with gastric ulcer. Such a circumscribed thickening and induration are always possible. We may have the same pyloric stenosis and secondary dilatation. Such non-cancerous thickening may even occur without ulcer. Other forms of morbid growths, such as fibroma sarcoma and the like, are too rare to demand notice from the clinical standpoint. Finally, the gastric tumor is not always demonstrable and may not be throughout the whole course of its existence. It is said to be absent in about 20 per cent. of cases. Then the diagnosis must be made from the symptoms, especially the rapid wasting and cachexia, which are rarely simulated even in ulcer. The age of the patient, generally past forty, the deficiency in HCl, and the presence of lactic acid must be allowed due weight.

Prognosis.—This is inevitably fatal, but something may be done toward prolonging life by the proper cleansing out of the stomach, the selection and regulation of food, and measures to aid its digestion. The operation of gastrostomy should be considered, as it sometimes prolongs life.

Treatment.—Since the cure of cancer of the stomach is impossible, treatment must be directed toward prolonging the patient's life. I am quite sure that a great deal more can be done than is commonly thought possible.

The stomach has no use outside of the preparation of the food for absorption. It is not a vital organ in the sense that the heart and the lungs are vital organs. It is important so far as it prepares the food, but if the food can be prepared for absorption outside of the body, its importance is diminished. So it is if we introduce artificially digested food by the rectum. Or we may use both of these methods. We can, by the use of prepared food, diminish the labor of the stomach, and by

using the rectum, we can, while doing so, relieve the stomach of all labor. This is rendered easier at the present day by the use of peptonized foods of various kinds. The food may be peptonized at home or the peptonized products of the manufactures may be substituted. Three to five grains (0.2 to 0.3 gm.) of the extract of pancreas with about ½ ounce (15 gm.) of sodium carbonate are added to a pint of milk and the mixture placed at a temperature of 100° F. (37.8° C.). In one hour all the casein will be peptonized. A curd is first produced, which subsequently undergoes solution. If peptonizing is complete, the addition of rennet will not produce coagulation. Milk thus prepared makes little demand upon the stomach for digestion, and it can be introduced advantageously by the rectum. Peptonized milk has a slightly bitter taste, and unless this bitterness is present its digestion is unaccomplished. The digestion will take place at a lower temperature than 100° F. (37.8° C.), but it takes longer.*

Beef may be peptonized for rectal alimentation as follows: Take half a pound of beef with the fat removed and a quarter pound of fresh pancreas. The pancreas is finely chopped and afterward bruised in a mortar with tepid water at a temperature of 100° F. (37.8° C.). It is then placed in a saucepan, in which a raw egg is beaten up, and is intimately mixed with the meat, previously chopped into small pieces. The product is next allowed to stand at a temperature of 100° F. (37.8° C.) for two hours. It is then strained, after which it is ready for use. This amount suffices for two daily injections. The preparation decomposes very quickly, so that it has to be made fresh every day. I have been surprised at what I have accomplished by this method, which is essentially one recommended by Mayer, of Lyons. In a case where nothing could pass the pylorus, under the use of daily nutritious enemata there occurred each morning an evacuation from the bowel as natural as when the patient was living on a mixed diet and digesting it properly.

The enterprise of the manufacturing chemists and pharmacists has resulted in the preparation of a number of beef peptonoids and extracts which may be substituted, but I never feel quite as sure of them as of the product made at home, troublesome as it is, because I do not know the nourishment equivalent of the manufacturers' product.

However careful the preparation of food, when taken into the stomach only a part is used up, and there accumulates gradually a quantity of unabsorbed material which does not pass the pylorus, and to this a copious mucous secretion is added. Hence, occasionally, once a day or every other day, it is desirable to wash the stomach out with tepid water or alkaline waters, as described in the treatment of gastric

* The following method, slightly modified from that usually recommended, after numerous trials by patients, has been found most satisfactory. Take one pint of skimmed milk, to which add one gill of water. Heat to 140° F. (60° C.)—a temperature at which the finger can be immersed for half a minute. After taking from the fire, stir in three grains (0.2 gm.) of powdered pancreatine and 15 grains (one gm.) carbonate of sodium. Place in a covered kettle or jug and roll up in a cosey (an ordinary gossamer water-proof coat answers admirably well), near a stove or register, to keep warm. Let it remain thus for an hour and a half. It then resembles slightly thickened milk, but there is no curd. Pour it into a covered pitcher and set aside to cool in the open air. Thus prepared, it has the slightest perceptible tinge of bitterness, and is very palatable.

catarrh. The free use of hydrochloric acid as a medicine also aids not only in the solution of the food ingested, but prevents the fermentations, which contribute irritating acids to the gastric contents and cause further mischief and discomfort.

DILATATION OF THE STOMACH.

SYNONYM.—*Gastrectasia.*

Definition.—A permanent increase in the volume and capacity of the stomach, the result of nervo-muscular atony or pyloric obstruction. It is to be distinguished from temporary distention and simple large stomach.

Etiology.—The nervo-muscular atony causing dilatation may be the result of habitual over-eating, especially food of defective quality, resulting in stasis and fermentation; of excessive drinking, as in beer-drinking employees of breweries; of chronic gastric catarrh; of diseases producing general nervo-muscular atony, such as disease of the spinal cord, pulmonary consumption, anæmia, chlorosis, acute fevers, affections of the heart, liver, and kidneys, and other diseases of like import. Mechanical or obstructive dilatation is most frequently due to obstruction from cancer at the pylorus or in the duodenum, or to cicatricial contraction, or to hypertrophic thickening. It is most frequent in middle-aged persons, but may occur even in children.

Morbid Anatomy.—In addition to the increase of volume the coats of the stomach are thinned and the glandular structure more or less atrophied. The average normal stomach of an adult holds about 1½ liters (three pints), while the abnormally dilated organ may attain a capacity of three or four liters (six or eight pints) and even more. Where the dilatation is mechanical there is added the lesion which is responsible for the obstruction.

Symptoms.—The symptoms arising from dilatation are a *sense of fulness in the epigastrium, eructations, flatulence,* and *vomiting,* often of enormous quantities. The *appetite* is sometimes poor, at others quite good, and the patient is hungry and thirsty. The *vomited matters* are largely water, but include also remnants of food and every variety of fungus, viz., bacteria, sarcina, yeast fungus, etc. Their reaction usually exhibits *lessened acidity* because of diminished hydrochloric acid secretion, but it may be normal or even abnormally acid. Such abnormal acidity is the result of fermentations producing lactic, butyric, and acetic acids. Various gases are also thus produced, including carbonic acid and hydrogen. The latter may also arise from decomposition of albuminoid substances, whence also arises sulphuretted hydrogen. These fermentations are favored by the absence of HCl, whose importance in preventing fermentation has been alluded to, and by a stasis of the contents in the stomach. For not only is absorption delayed, but the transit of gastric contents into the intestine is also hindered. Indeed, in some cases, the stomach is never emptied unless by the tube. Nay, more. It would seem that at times it contains more liquid than was ingested, a possible condition, since the endosmosis of crystalloids, viz., sugar, dextrin, alcohol, and

peptones, is attended with the exosmosis of water. From such causes, too, we have *torpor of the bowel*, *scantiness of urine*, and *dryness of the skin*.

Anæmia, *emaciation*, and *debility* sooner or later succeed, and in fatal cases death is commonly preceded by a *drowsiness* which may be due to the absorption of toxic substances arising in the decompositions going on in the stomach. Dilatation of the stomach is also one of the acknowledged causes of *tetany*, as first pointed out by Kussmaul. The cramps, though often quite severe, are of short duration. They occur chiefly in the muscles of the hands, arms, and legs. V. Leube suggests that this tetany may be due to a "drying out" of the nerves and muscles, but it may also be the result of auto-intoxication. *Unconsciousness* may precede death.

Physical Signs.—These may be elicited by inspection, palpation, and percussion. *Inspection* does not always afford information, but in emaciated cases the greater curvature of the distended organ may be recognized as low as the navel and below, instead of from 1.2 to 2.8 inches above it (three to seven centimeters). When the stomach is very low, even the smaller curvature may be recognized a couple of inches below the ensiform cartilage uncovering the pancreas. In obstruction of the pylorus the peristalsis from left to right may even be recognized, stopping short at the pylorus, where the tumor-like thickening may sometimes be seen. In rare instances a reverse peristalsis from right to left takes place.

Palpation may confirm inspection, recognizing the contour of the stomach by its peculiar consistence, which has been compared to that of an air-cushion, but affords little additional information unless there be a tumor at the pylorus which may be felt. Peristalsis if present may also be felt, and may be stimulated by filliping the abdominal walls with the fingers, by which also a splashing sound may be produced in the water-laden dilated stomach down as low as the greater curvature. This is to be distinguished from a similar splashing which may be obtained in the normal stomach and adjacent colon, though the latter is less constant and less intense.

If a stiff *sound* is used its end may be felt through the abdominal walls, while the unusual extent to which it may be carried before meeting resistance will attract attention.

Percussion affords the most valuable evidence as to the presence of a dilated stomach, and in the majority of instances such evidence is conclusive. Percussion should be made in the standing position, if possible, from above downward, beginning at the edge of the ribs in the neighborhood of the parasternal line. The note is tympanitic until the lower curvature is reached, when it is substituted by dulness due to the liquid contents, to be succeeded again by tympany of the bowel when the lower border of the stomach is passed. If the patient lies on his back the dulness disappears and is replaced by tympany. If there is no liquid in the stomach, a change in the pitch of the tympanitic note will indicate the transition from the stomach to intestine. Further information can be gained by means of the sound, by which the stomach can be emptied and refilled with water and its borders determined by percussion. This is more satisfactory than filling the stomach with carbonic acid or air, and even such

procedure is not always necessary. If the larger curvature be found by percussion at the navel or below, the stomach is certainly dilated.

No reliable evidence of dilatation is furnished by auscultation.

The question whether the dilatation is dynamical or mechanical, that is, whether it is the result of nervo-atony or obstruction by a tumor at the pylorus, can generally be decided by recognition of a tumor at this orifice. Vomiting is also more severe and frequent and the peristaltic unrest is more active.

Diagnosis.—This is usually easy by attention to the symptoms and physical signs described. Dilated stomach has, however, been mistaken for an ovarian cyst, and abdominal section has been made for its relief.

Prognosis.—When associated with malignant disease at the pylorus recovery is, of course, impossible, as, indeed, it is in dynamic dilatation, but in the latter case much relief may be afforded to the symptoms.

Treatment.—The most important part of the treatment is washing out the stomach after the method detailed on p. 249. This may be done daily, but sometimes it is sufficient to do it on alternate days, occasionally even twice daily. When practised once a day it is usually best done on retiring at night, as the stomach is thus freed for the night of irritating material which if retained disturbs rest and aggravates the local condition. The patient soon learns the most suitable time for lavage, and when its frequent necessity is determined he should be taught to do it.

Of drugs, hydrochloric acid is the most likely to be useful, not only because of its importance as a digestive agent, but also as a preventer of fermentation. To this pepsin becomes a useful adjuvant, because it is scantily formed in the dilated stomach. Nitro-muriatic acid may sometimes be substituted with advantage, especially where a stimulating effect is desired on the liver. It should be freshly prepared and three drops of a pure acid given to an adult at a dose. Strychnine is a drug which has much to recommend it from the theoretical standpoint as a muscular tonic and has the further advantage of easy absorption. It should be administered in full doses from a small beginning, $\frac{1}{30}$ grain (0.002 gm.) three times a day, increased to $\frac{1}{20}$ grain (0.003 gm.), and even more. Extract of nux vomica may be substituted, but is less easily absorbed.

In addition to the hydrochloric acid as an antifermentative, other remedies for this purpose are charcoal and creasote. The power possessed by charcoal of absorbing gases cannot be availed of, because it possesses this property only in the dry state. Yet it does relieve flatulence and is antiseptic. Such antisepsis may be extended to the intestine. Doses of five to ten grains (0.33 to 0.66 gm.) and even more may be given of charcoal.

Creasote is a useful antiseptic, and may be given alone, in capsule or in pill, in doses of ½ grain to a grain (0.03 to 0.065 gm.), or it may be given in sherry wine, whiskey or brandy, or tincture of gentian. The following one per cent. solution of creasote is a modification, by George Herschell, of Bouchard's well-known formula :—

℞. Creasoti, ... 10
Tr. Gentianæ, ... 20
Vin. Xerici, ... 800
Sp. Vini Gall., ... 170
M. et S.—One hundred minims contain one minim or one grain of creasote.

When the condition is part of the morbid anatomy of cancer of the stomach, only palliation may be expected.

Dietetic Treatment.—Most important is the selection of food in these cases. Solids should be almost totally prohibited, while the typical nourishment are the various kinds of artificially-digested food, such as peptonized milk and beef peptonoids. Of the latter, the dry form of beef powder is suitable, because it absorbs some of the excessive liquid sometimes present in the stomach. Beef juice and rare beef scraped are also easily assimilated, while fatty, and especially starchy, foods are to be used sparingly, if at all.

DISEASES OF THE INTESTINES.

SIMPLE ACUTE CATARRHAL ENTERITIS.

SYNONYMS.—*Acute Intestinal Catarrh; Acute Diarrhœa; Acute Ileo-colitis.*

Definition.—The term employed is applied to a diffuse inflammation which generally pervades more or less of the small intestine and the upper part of the large bowel. More circumscribed inflammations are described and doubtless sometimes occur, but it is not easy to localize them.

Etiology.—The usual cause of simple intestinal catarrh is over-eating and drinking, or the swallowing of mineral substances of an irritating character. Impurities in drinking water, and unripe fruit in the summer and autumn are a frequent cause. The toxic products of fermented and decomposed food are also causes. These sometimes arise inexplicably from substances commonly harmless, such as milk or preparations thereof. Cream-puffs, and even ice-creams, are among these. Irritating minerals are corrosive sublimate and arsenic. Although hot weather favors intestinal catarrhs, especially in infants and older children, they are not so much the direct result of the heat as of its effect in weakening the resisting power of the child and favoring the decompositions and fermentations referred to. The effect of heat on the nervous system of the very young may reasonably be regarded as a factor in increasing the irritability of the gastro-intestinal tract or so diminishing its functional power as to render the ingesta irritating. Cold, or rather a chilling of the body by a fall in temperature, is often followed by enteritis.

Secretion altered in quantity or quality has already been mentioned as a cause of simple non-infectious intestinal inflammation. Much spoken of, but of inferred, rather than demonstrated, import is excessive biliary secretion, producing what is known as bilious diarrhœa. When such diarrhœa is associated with a burning sensation at the anus and the recognized presence of bile in the stools, the term may be justified, but it is to be remembered that an acid reaction of the alvine dejecta produces a similar sensation. A scanty supply of bile to the intestine, by depriving the gut of the important antiseptic property of this secretion, may also favor the fermentations and decompositions mentioned.

Hyperæmia, however induced, favors catarrhal enteritis. Such is

the hyperæmia secondary to hepatic and cardiac disease, to inflammation, whether traumatic or infectious, in adjacent tissues, whence it extends by contiguity. Such is the inflammation occasioned by peritonitis, by intestinal obstruction, and the like. Cachectic and anæmic states, such as are secondary to cancer, to Addison's disease and the last stages of Bright's disease and tuberculosis, are also favoring causes. Enteritis is also a symptom of certain infectious diseases through their specific poisons, which act directly on the mucous membrane, as in the case of cholera, dysentery, and typhoid fever.

Apart from the effect of nervous influence already mentioned, this cannot be said to cause simple enteritis. It is not unusual for fright and other causes of nervous excitement to produce diarrhœa, but this is not the result of an enteritis but of an increased peristalsis and disturbed vasomotor regurgitation, and is properly called nervous diarrhœa.

Morbid Anatomy.—The morbid changes of simple intestinal catarrh are variously distinct. A hyperæmia is naturally to be expected, and in the more decided cases may be manifested by a diffuse redness and injection. It is not often, however, that these are demonstrable. A layer of mucus covering the mucous membrane of the bowel more or less interruptedly is more frequently present. Nor is swelling often evident. At times the solitary follicles are unnaturally distinct, surrounded by a hyperæmic circlet. Such enlargements, commonly as distinct as a pin's head, may be as large as a pea, and becoming filled with pus, form little abscesses which may rupture, leaving an ulcer. Such enlargements may extend to Peyer's patches. More rarely chronic ulceration results.

Symptoms.—*Diarrhœa* is the most constant symptom of enteritis, involving the part of the intestinal tract named in the definition. The resulting stools consist of, first, ordinary fecal contents of the small and large intestine, including bile; but as they continue they become more and more watery, almost colorless. There may be but two or three, or they may equal 20 or more. They contain more or less mucus, and are often frothy and associated with flatus. With diminished consistence the odor may grow less, until totally absent. At other times it is persistently offensive. Minute examination recognizes in these discharges columnar epithelium variously altered, enlarged, granular, and fragmentary, with nuclei obscured or absent; also various non-pathogenic bacilli and cocci, including the bacterium coli commune, yeast fungus, crystals of triple phosphate, oxalate of lime, cholesterin, and undissolved food matters. The reaction of the discharge may be neutral or acid.

Next to diarrhœa is *pain*, usually colicky, varying greatly in degree, often, indeed, in the milder forms, absent. There is rarely *tenderness*, but palpation may elicit gurgling and the signs of gaseous distention. *Thirst* and *oliguria* are natural consequences of the free discharge of water. There is usually little *fever*, the rise of temperature rarely exceeding one or two degrees, and the higher grades suggest specific inflammation of the bowel. The *appetite*, at first little altered, ultimately fails. Very rarely do the ordinary diarrhœas in children and adults terminate in collapse.

It is reasonable to expect modifications of the above symptoms as the result of localized inflammation, as contrasted with those of the more

diffuse form just described. Thus the presence of jaundice suggests the probability that the *duodenum* is especially involved in the inflammation. In such cases the urine may also be jaundiced, and there may be added other symptoms commonly associated with jaundice. In the absence of jaundice there is no sign which points to the duodenum as the special seat of the inflammation. On the other hand, jaundice is by no means always present, even if the duodenum is involved. Duodenitis is often associated with acute gastritis, spreading from the stomach—*gastro-duodenitis*.

An acute catarrhal inflammation of the *jejunum* and *ileum*, unassociated with inflammation of the large bowel, would be unattended with diarrhœa, the slight acceleration of peristalsis incident to such an event being unlikely to produce this symptom. In this respect, therefore, it will differ. On the other hand, distention of the abdomen, colicky pain, borborygmi, discharge of flatus, and fever continue. Nothnagel has called attention to the presence of little lumps of mucus from the inflamed small intestine in intimate admixture with the contents of the large bowel, often, however, requiring the microscope for its recognition. Even if this is true, however, as v. Leube says, it is scarcely available in practice. Whence it is plain that a diagnosis of an inflammation of this part of the intestinal tract is by no means an easy matter. It is probably also a rare condition by itself. Nothing distinctive is added if only the upper part of the large bowel is involved.

Quite different is it where there is also involvement of the whole of the large intestine—*ileo-colitis*. When this is the case, while the lower down the inflammation the purer the mucus and the more there is of tenesmus, the mucus remains separate and unmixed with the fecal matter, which may contain undigested articles of food, such as muscular fibres, starch, and fat corpuscles. A diarrhœa in which these undigested portions of food are visible to the naked eye is known as *lienteric*. Gmelin's nitric acid test for the biliary coloring-matters ceases in health at the sigmoid flexure, so that if this reaction is obtainable in the liquid discharges, it implies that the excessive peristalsis has affected also the large bowel by which the bile is carried through with abnormal rapidity. The green stools of children, and more rarely of adults, also indicate a large quantity of bile. Simple feverish states, however, may have the effect also of interfering with the proper digestion of food matters, which may appear in the discharges in consequence. Some information, not, however, too much to be relied upon, may be derived from the seat of tenderness and colicky pains. When these are in the middle or inferior part of the abdomen they point to the small intestine, when to the upper and lateral parts, the large. (See also Dysentery and Proctitis.)

Diagnosis.—The diagnosis of acute intestinal catarrh is ordinarily easy, by attention to the symptoms above detailed, including those more or less peculiar to the more circumscribed localities referred to. From typhoid fever acute enteritis is usually easily distinguished by its short duration, minor fever, and the absence of the characteristic course the fever takes in the infectious disease and of the spots which so invariably make their appearance on the eighth day.

During cholera epidemics mild cases of this disease are not recog-

nizable symptomatically from the severer colliquative forms of diarrhœa. Under these circumstances, bacteriological examination should be made. The importance of a correct diagnosis will be appreciated when it is remembered that indifference in the treatment of simple diarrhœa may not seriously affect the result, while such treatment of a case of cholera, however mild, may result disastrously.

Prognosis.—This is always favorable with prompt and judicious treatment, recovery taking place in from one to three days, as a rule, rarely longer.

Treatment.—Many cases of acute catarrhal enteritis recover under rest and restricted diet, the degree of which necessarily depends on the severity of the case. The simple withdrawal of all food, the substitution of plain milk, or in severe cases boiled milk, for the usual food generally suffices. A few grains of bismuth subnitrate every two or three hours, fortified with ⅛, ¼, ½ grain (0.0082, 0.015, 0.033 gm.) of opium, or ½ ounce (15.5 gm.) of chalk mixture with a fluidrachm (4 c. c.) more or less of paregoric may be added. No attempt should, however, be made to lock up the bowel until all irritating matters are removed, and it is often desirable to give an aperient, of which castor oil is the best, though the unpleasantness of the dose often precludes this otherwise valuable remedy. In such event, the solution of the citrate of magnesium, Rochelle salts, or Hunyadi water may be substituted. Where there is much pain larger doses of opium may be necessary, especially if hot fomentations fail to produce the desired effect. Astringents are rarely necessary, but in the absence of other measures may be used. Tannic or gallic acid in five-grain doses (0.33 gm.) may be given separately or combined with opium.

The various chlorodynes form convenient remedies when there is pain. The dose varies from 20 to 30 minims (one to 1.9 gm.). In severe cases, especially where there is nausea, a hypodermic injection of morphine, ⅛ to ¼ grain (0.0082 to 0.015 gm.), may be given. For the nausea counter-irritation by mustard-plasters should be used, pieces of ice swallowed entire, while copious draughts of water should be disallowed. Champagne and cold carbonated waters may be used for this purpose. The latter may be combined with milk, while the old reliable remedy of equal parts of milk and lime-water should not be forgotten.

CHRONIC CATARRHAL ENTERITIS.

SYNONYMS.—*Chronic Entero-colitis; Ulcerative Colitis; Mucous Colitis; Chronic Diarrhœa.*

Definition.—A chronic inflammation of more or less of the large and small intestine, with or without ulceration.

Etiology.—Chronic enteritis may remain after repeated attacks of the acute form, or it may arise *de novo*, however induced, favored by whatever occasions passive congestion. Such favoring causes are diseases of the liver or heart, feeble and anæmic states, and the defective nutrition consequent thereon. Chronic exhausting diseases like tuberculosis and Bright's disease may act in this way also. Dysentery is a frequent cause of chronic intestinal catarrh, a remnant of the acute process.

Morbid Anatomy.—The primary condition is that of acute catarrh, and in many cases the morbid changes do not exceed those of acute catarrh, being simply permanent or later more pronounced. In others, still more pronounced changes are found, chiefly in the lower part of the ileum and colon. These are mainly ulcerative, but include also discolorations due to hyperæmia, blood extravasation and pigmentation succeeding it, thickening of the coats of the bowel, and contraction of partly-healed ulcers. There may be stenosis or the opposite condition of dilatation. Such ulceration is distinct from that of tuberculosis, typhoid fever, and syphilis. It may be follicular, as often seen in the diarrhœal affections of children, more rarely in adults, or there may be large ulcers or large areas of ulceration. The remnant of mucous membrane is often pigmented and slate-colored, and a pseudo-polyposis sometimes results from contraction. In the small intestine the pigment is apt to be deposited on the ends of the villi and in rings around the solitary follicles, or in their centres, producing the "shaven-beard appearance." The surface of the bowel is more or less covered with mucous and purulent secretion incident to the inflammation. Still another sort of ulceration, from the etiological standpoint, is found at the bottom of saccules of the large intestine in which have lain a long time scybala or hard fecal masses. Ulceration, too, may result, though rarely from encroachment from without, by various kinds of disease of the peritoneum, including cancer, tuberculosis, and the like. Atrophy of the mucous membrane of the bowel is also one of the results of chronic enteritis, not usually recognizable before death.

There may even be atrophy not only of the mucous membrane, with destruction of the glands, but also of all the coats of the small and large intestines.

Symptoms.—These are not uniform. While there is often more or less *diarrhœa*, this is as often absent, or substituted by *constipation*, while constipation and diarrhœa frequently alternate. More characteristic of the stools is the large amount of *mucous matter* contained in them. This may be present in the shape of "sago"-like masses or "mucous" granules, yellow or brownish-yellow, bile-stained also from the small intestine. Bile-stained mucus is only present when there is abnormally

rapid peristalsis of the large bowel, which causes the mucus to be passed out before the bile is decomposed. Ulceration may be associated with the presence of blood in the stools.

A variety of chronic colitis known as *mucous colitis* or membranous enteritis is characterized by the discharge of large masses of mucus, forming at times complete casts of the bowel. It is more frequent in women, this sex including 80 per cent. of recorded cases, according to W. A. Edwards. It may occur also in children. Its subjects are usually women of the nervous type. It is commonly associated with constipation. At intervals, however, occur attacks of abdominal pain and tenderness, sometimes accompanied by tenesmus and followed by discharges of the mucoid matter referred to. Such attacks may be excited by mental emotion of various kinds. The mucoid material itself seems to be the direct result of an increased activity of the mucous glands, which, with the mucous membrane, are, however, commonly intact after the separation of the large mucous casts. Minute examination recognizes various numbers of cells, round and columnar, entangled in the mucus, sometimes also cholesterin plates and triple phosphate crystals.

Throughout the numerous attacks, nutrition is commonly well maintained, and the woman-subject appears plump and well-nourished. At other times there is gradual emaciation and ultimate death.

Diagnosis.—This is always easy, except as to the determination of the portion of the bowel involved or the presence of ulceration. Differences in the character of the mucus, as above noted, will aid in the diagnosis, while the constant or intermittent presence of blood and pus or fragments of tissue in the stools point to the ulcerative condition. Ulceration is sometimes found post mortem where no symptoms were present before death. In the rectum and, indeed, as high as the sigmoid flexure ulcer may be recognized by specular examination. Deep-seated ulceration may cause circumscribed peritonitis or produce abscess. The presence of scybala surrounded with mucus points to inflammation of the rectum or colon as far up as its transverse portion. It is not possible to diagnose the presence of atrophy of either bowel.

Prognosis.—The prognosis in all forms of chronic intestinal catarrh is grave so far as recovery is concerned, and treatment avails little in many cases. The disease, however, extends over months, and even years, before the patient succumbs, and recovery is sometimes complete.

Treatment.—When it is remembered that chronic intestinal catarrh is seated mainly in the large intestine, it is manifest that to reach it with remedies administered the ordinary way is difficult, and that it is more than likely that such remedies are absorbed or decomposed before they arrive at the seat of the disease. It is barely possible that by the long administration of certain drugs, as nitrate of silver, a certain proportion will, after a while, reach the seat of ulceration and stimulate it to heal. Such a course must therefore be pursued with any remedies thus administered. Nitrate of silver and the sulphate of copper are the two which possess most reputation. The doses are ¼ grain (0.0155 gm.) of each three times a day, or a smaller quantity more frequently. The acetate of lead may be substituted in doses of two grains (0.132 gm.). The latter is more astringent but less likely to excite healing. All of these

remedies are commonly combined with opium in suitable doses. Subnitrate of bismuth in large doses, ½ drachm to one drachm (one to two gm.) is strongly recommended by some. It undoubtedly diminishes the discharges, but how far it is curative is uncertain.

Much more rational is it to select a diet with a minimum of waste, so that there may be as little irritating residue as possible. Milk and the albuminous foods are the types of these. Still less irritating must they be if partially digested before taken into the stomach. Thus milk may be peptonized, and meat also, and the beef peptonoids of the manufacturers may be employed. It is difficult to ascertain the ratio of nourishing power of these peptonoids to that of solid meat. This, then, should be a fundamental principle of treatment—to furnish a diet with a minimum of waste.

The natural astringent waters, such as the Rockbridge and other alum waters in this country, have earned some reputation in the treatment of chronic intestinal catarrh, but improvement under their use is always more marked at the springs themselves, showing that some effect must be ascribed to the change of scene and air and the salubrious climate of the locality.

Should these measures fail, irrigation of the bowel may be practised. This is done by means of a fountain-syringe, or funnel in connection with a tube, which is carried high up into the bowel, the patient being placed on his back with a pillow under his hips. The fluids used are solutions of nitrate of silver, sulphate of zinc, and boric acid. At first tepid water, say at 85° F. (30° C.), should be run in very slowly to the amount of two to three pints (one to 1½ liters). Then solutions of any of the above substances of the strength of three to 4½ parts to 1000, or 20 to 30 grains to the pint (1.3 gm. to two gm. to the liter) of the more active substances, beginning with the weaker solutions. Salicylic acid may be used in two per cent. solution, boric acid in one per cent. solution, or a one per cent. solution of salicylic and boracic acids combined. A one per cent. solution of tannic acid is also recommended, as well as corrosive sublimate, but this is exceedingly irritating and the strength of the solutions should not exceed, at first, one to 15,000, which may be increased if well borne. The nitrate of silver has, on the whole, the best reputation. A preliminary anodyne enema of 30 minims (one gm.) of laudanum may be given if needed, or a suppository of extract of opium, say one grain (0.066 gm.). To be effectual, the treatment must be patiently prolonged, especially the dietetic part, and not weeks, but months of patient perseverance insisted upon. I have already said in treating of dysentery that a careful trial of this form of treatment in my hands has been disappointing in its results.

DIARRHŒAS OF CHILDREN.

The importance of these, and some specialization in their symptomatology, demand a separate consideration. Three forms, more or less distinct, are recognizable, viz., acute dyspeptic diarrhœa, cholera infantum, and acute entero-colitis.

Acute Dyspeptic Diarrhœa.

Etiology.—This disease is mainly due to errors in diet, which do not necessarily consist in the substitution of unnatural foods for the mother's milk. The latter itself may be altered in quality by emotional causes, by improper food, and improper hygiene. Or it may be too liberally supplied by over-frequent nursing. More often, however, it is the ingestion of unnatural food, either of substances palpably unsuitable, carelessly allowed, or surreptitiously taken, or substitutes necessarily employed for mother's milk, when she is unable to nurse her infant.

"Bottle food," the most carefully selected, is unnatural, and is probably the most frequent cause of dyspeptic diarrhœa in children otherwise well-cared for. Two factors in this are active, first, the relatively greater indigestibility of the foods thus supplied and, second, the bacteria and their toxic products which develop in it before or after ingestion. Normally, the feces of infants contain but few species of bacteria, of which the most important are the *bacterium aërogenes* and the *bacterium coli commune*. The former seems to be an exclusive product of a milk diet, depending upon the milk-sugar for its nourishment, and is found in the upper bowel, where it excites fermentation in milk. The habitat of the bacterium coli commune is the lower part of the small intestine and the colon, where it is probably also an agent of fermentation. In infantile diarrhœa the number of species of bacteria is greatly increased, but no one or more species has as yet been shown to possess a specific causal effect.

There are also predisposing influences which facilitate the action of the exciting causes. These are, especially, dentition and the extreme heat of summer. The effect of the former is learned in the experience of every mother, while the extraordinary frequency of infantile diarrhœa in summer attests the latter. It is evident, too, that constitutional weakness and bad hygiene must also co-operate to diminish the resisting power of infants to other causes. Hence it is that the children of the delicate, the poor, and filthy suffer most.

Morbid Anatomy.—This seldom succeeds the stage of catarrhal swelling, already described when treating of the enteritis of adults.

Symptoms.—No symptoms may precede the diarrhœa, but usually there is in the beginning *restlessness* with slight *fever*, which seldom becomes high. Such restlessness may be due to *nausea* or colicky *pain*. The nausea may go on to *vomiting* or not, but *purging* soon occurs. The stools are at first copious and offensive, often yeasty and sour, and generally contain particles of coagulated milk or other undigested food, such as unripe fruit, if the child is old enough to get it. At first infrequent, they become more numerous, more scanty, acquire sometimes a green color

and sometimes contain mucus, rarely blood. There may be but three or four stools or there may be 20 or more in the twenty-four hours.

In other cases, fever is more decided, and the temperature may rise rapidly to 104° F. (40° C.), there is great *thirst*, and *scanty urine*. In either event, there is rapid *emaciation*, and the child falls away amazingly in a few days.

Diagnosis.—The sudden onset and character of the stools are distinctive and scarcely mistakable. The small amount of mucus distinguishes them from those of ileo-colitis, and the absence of serous discharge from those of cholera infantum.

Prognosis.—This, among the better classes, is commonly favorable, but among the weak, puny, and half-starved children of the poor, large numbers perish, especially in hot weather. The disease may pass over into the much more serious affection of entero-colitis.

Treatment.—The principles of treatment are similar to those of enteritis in adults. A primary purge is commonly indicated. Calcined magnesia is very suitable, though castor-oil is here also useful. After the purge, bismuth subnitrate or prepared chalk in doses of 2½ grains (0.165 gm.) for a child a year old, with ½ grain (0.033 gm.) of salol as an intestinal antiseptic, may be given every two or three hours. If there is pain, $\frac{1}{24}$ to $\frac{1}{12}$ grain (0.0027 to .0054 gm.) opium may be added each time or every other dose, as may be demanded by circumstances. Attempt should first be made to relieve pain by gentle counter-irritation, as by weak mustard-plasters or a plaster of mixed spices wet in whiskey or alcohol and known as a "spice plaster" and worn continuously. Instead of the above powder, a mixture may be made up with the same remedies, substituting deodorized tincture of opium or paregoric for the opium. Astringents are seldom necessary in children's diarrhœa, but the compound tincture of kino, which contains a little opium, is an efficient remedy, which probably owes much of its efficiency to the opium. The antiseptic treatment has never commended itself to me, and I am inclined to think that more harm than good has been done by such remedies as resorcin, naphthalin, and the like, which are often irritating. The regulation of diet is of the utmost importance. It is better to give the child nothing except a little cold water or barley-water than unsuitable food, while any food that is given should be very much diluted, and should be scanty rather than overabundant. Too much food is often given. Nothing is better than peptonized milk, if the mother's milk or that of a wet-nurse be unobtainable. It may be diluted with Vichy water, lime-water, or plain water, to which a little brandy may be added. As long as casein appears in the stools, the milk requires further dilution, or the casein may be removed altogether and the whey only allowed. Animal broths, however dilute, do not find much favor with me, though occasionally beef-juice is well borne when milk has not been, especially in children a couple of years old.

The hygienic surroundings of the child are important. Frequent bathing, light, cool dressing in warm weather, and fresh air at all times are indispensable. Removal from city air to the country or seaside, when possible, should be practised, and when this is not possible, frequent excursions should be made to the country or on an adjacent river. It is not desirable to keep the child on the lap any more than is necessary.

Cholera Infantum.

Definition.—A variety of acute catarrhal enteritis of intense severity, corresponding in symptoms and course to cholera morbus in the adult, but much more serious in termination.

Etiology.—The same reasons which lead us to expect a specific cause of cholera morbus would suggest also one for cholera infantum. None has, however, been found. It may reasonably be ascribed to toxines generated in the decomposition and fermentation of foods, since some error of diet is almost always the apparent exciting cause. There are also predisposing causes, of which hot weather, dentition, or both, bad hygiene, the previous presence of dyspeptic diarrhœa or entero-colitis are instances. It is less frequent than either of the last-named affections, including only a small proportion of the summer complaints of children, according to Holt not more than two to three per cent.

Morbid Anatomy.—There is little, if any, deviation from the normal appearance in the affected bowel.

Symptoms.—These consist in copious *serous stools*, at first containing some offensive fecal matter, later a few particles of greenish matter, but ultimately they are almost aqueous, being ejected also with great force. They contain numerous bacteria, but no constant organism has been found. There is also crampy *pain*, and the *limbs are drawn up* or *rigidly extended*. There is decided *fever*, more than in either of the two other forms, the temperature reaching 105° F. (40.5° C.), the *pulse* is frequent and feeble, while *restlessness* is a characteristic symptom. The temperature should be taken in the rectum, as that of the axilla may be misleading. Indeed, the skin sometimes feels cool when the internal temperature is high. There is intense *thirst*, and the child eagerly drinks water. The *purging* may come on suddenly or succeed dyspeptic diarrhœa or ileo-colitis. Simultaneously there is severe and obstinate *vomiting*, including bile at first, but later the vomited matter is also serous. The *tongue is coated* in the beginning, but later becomes dry and red. The child rapidly loses strength and as rapidly emaciates. The restlessness is succeeded by *apathy* and *indifference*, and the condition passes over into collapse. The eyes become sunken, the fontanelles depressed, the skin grey or ashen and closely applied to the frame—producing an appearance which, once seen, is rarely forgotten. Or the more severe symptoms may subside and a condition of *torpor* or *semi-consciousness* supervene. The head is retracted, and there may be *convulsions;* the breathing is interrupted and of the Cheyne-Stokes type; the pupils are irregular; there is clutching of the fingers,—in a word, the "hydrencephaloid" state, so called by Marshall Hall, is present. These "brain symptoms" have often misled the inexperienced, but they are not associated with changes in the brain or its meninges. They may be due to the toxines developed in the intestine by bacteria.

Diagnosis.—This is not difficult. The serous vomiting and purging, the rapid emaciation and prostration, and the hyperpyrexia are significant, while the nervous symptoms described succeeding upon them confirm the nature of the disease.

Prognosis.—Unless the last-described symptoms supervene the

course is rapid through collapse to a fatal termination in from a few hours to twenty-four or forty-eight hours. If the hydrencephaloid state is added, the disease may be prolonged for a few days more. Recovery is not impossible, and begins with abatement of the more serious symptoms within the first twenty-four hours, followed by tedious convalescence. Or there may be a delusive improvement followed by a return of the choleraic symptoms, or the disease may pass over into the entero-colitis.

Treatment.—All that has been said about food in dyspeptic diarrhœa and entero-colitis applies here, but the opportunity for its application cannot, indeed, be availed of unless convalescence sets in. The symptoms must be met with the greatest promptness by the same measures described in the treatment of adults, but adapted to the age of the child. Here, too, opiates are indispensable. Even morphine may be used hypodermically with great caution. One hundredth of a grain (0.00066 gm.) is about the proper dose for a child a year old, and it may be associated with $\frac{1}{500}$ grain (0.0001032 gm.) of atropine. This may be repeated in an hour if the symptoms do not subside, at a longer interval if they do. Laudanum or deodorized tincture of opium, in doses of two to four drops (0.133 to 0.264 gm.) in a couple of drachms of starch-water, may be substituted by the rectum. Minute doses of Dover's powder, say $\frac{1}{10}$ grain (0.006 gm.), may be given in combination with bismuth in doses of a couple of grains (0.12 gm.). Preparations of silver, preferably the oxide, are sometimes of value. They may be combined with opium, the dose of the former being $\frac{1}{12}$ grain (0.0056 gm.), of the latter $\frac{1}{24}$ to $\frac{1}{12}$ grain (0.00275 gm. to 0.0056 gm.).

The hyperpyrexia must be combated by hydrotherapy, the bath at 80° F. rapidly reduced to 70° F. (26.6° to 21.1° C.), or if this cannot be done the child should be wrapped in sheets wrung out in cold water. Sponging is a feeble substitute. Hyperpyrexia is one of the dangers.

Stimulants are indicated, but the difficulty is to secure their retention. Brandy is the best form of stimulant, though iced champagne may be given in small doses often repeated, while the prompt rejection of liquids should not discourage their re-administration. Irrigation of the large bowel may be added, using a flexible catheter, which is introduced six or eight inches (2.3 to 2.7 cm.). A pint (0.5 liter) will suffice for a child six months old, and a quart (one liter) for one of two years. The water may be tepid, or cold if the temperature is high. The one per cent. salt solution may be administered by entero-clysis and even by hypodermo-clysis in extreme cases of collapse. The hot bath should be substituted for the cold in collapse, and strychnine may be administered hypodermically in doses of $\frac{1}{100}$ grain (0.00066 gm.) to a child one year old.

Should convalescence set in, or entero-colitis be substituted, great caution in the giving of food should be observed. Only peptonized milk should be used, substituted occasionally by raw beef juice, which may be increased, if well borne, a teaspoonful at a time. Or dilute egg albumin may be tried if these are not retained.

Acute Entero-colitis.

Synonyms.—*Acute Ileo-colitis; Follicular Enteritis; Follicular Dysentery.*

Definition.—An inflammation more severe than dyspeptic enteritis, chiefly of the ileum and colon, affecting especially the lymph follicles.

Etiology.—It is also a disease of the hot months and of teething, though not confined to the former. It is produced by the same causes as dyspeptic diarrhœa. It is more frequent between the ages of six to eighteen months—second summer—and is not infrequent in the third and fourth years. It may be a termination of dyspeptic diarrhœa or of cholera infantum.

Morbid Anatomy.—The morbid changes are more positive than in acute dyspeptic diarrhœa, and are found chiefly in the ileum and colon. In the first stage the mucous membrane is congested and swollen, while the solitary follicles and Peyer's patches are more distinct. The epithelium appears exfoliated in places. As the disease continues, say after the first week, the enlarged follicles and Peyer's patches become ulcerated. The changes may end here or become more extensive, the ulcers enlarging and deepening to the muscular coat with the separation of a slough. Or there may be a diffuse infiltration of the bowel with small cells, producing a decided thickening of the same with more or less obliteration of its distinctive structure. The process may be so intense as to cause coagulation necrosis—false membrane.

Symptoms.—The disease may begin as a *dyspeptic diarrhœa*, also as a *cholera infantum*. It is much more serious than dyspeptic diarrhœa, as evidenced by the higher *fever*, which rises rapidly to 104°F. (40° C.), but still remains lower than in cholera infantum. *Vomiting* is less common than in dyspeptic diarrhœa or cholera infantum. There is decided abdominal *pain* and a *tense, swollen belly*. The fecal discharges, which are at first painless, are small in quantity, contain much mucus, and even a little blood. They vary in frequency from 15 to 30 in the twenty-four hours, and more frequently during the day. The disease may abate here and convalescence be established, though recovery remains slow. Or the symptoms may increase in severity, the fever persist, the *stools be painful* and *small*, consisting mainly of mucus and blood. Commonly odorless, they may also be extremely fetid. The *urine* is scanty, of high specific gravity, and deposits mixed urates. The child wastes away to a skeleton, the skin becomes loose and flabby, and the "old man" appearance is assumed. Such a case may last five or six weeks, terminating fatally, yet may also get well. A few fatal cases are much more rapid in their course, being ushered in with convulsions and ending in forty-eight hours to five or six days. Relapses after convalescence are not uncommon, and should be guarded against.

Diagnosis.—Acute entero-colitis is characterized by a greater severity than dyspeptic diarrhœa, the high fever, the large amount of mucus in the stools, the greater pain, suffering, and more rapid decline. From cholera infantum it differs in its lower hyperpyrexia, the absence of vomiting, of colliquative diarrhœa, and of collapse.

Prognosis.—This is more unfavorable than in acute dyspeptic diarrhœa, more favorable than in cholera infantum. Recovery is not in-

frequent after a lengthy illness of four to six weeks, while the severe dysenteric form is apt to be early fatal. Much depends upon the promptness with which treatment is instituted and the ability of the parents to carry it out, the previous vigor of the child, its hygiene, its food.

Treatment.—The general hygienic and dietetic treatment of acute entero-colitis is similar to that of acute dyspeptic diarrhœa; the medical treatment is somewhat different. Anodynes are more imperatively demanded because there is greater suffering, and depletion may be needed in the beginning by salines, though good judgment is required, because the child's strength must be husbanded. The colon may be flushed with a one per cent. cold salt solution, or cold water or pieces of ice may be introduced into the rectum, which may also be used for medication, more particularly for opium. I do not think the large rectal enemata recommended in the chronic colitis of adults are to be advised for children. If used, they should be very weak. Solutions of nitrate of silver, one grain to the ounce (0.066 gm. to 30 c. c.), and tannic acid, five grains to the ounce (0.33 gm. to 30 c. c.), are suitable. The mouth should be often examined and, when necessary, the coming teeth scarified, not once, but as often as necessary.

The Cœliac Affection in Children.

Synonyms.—*Diarrhœa alba; Diarrhœa chylosa.*

Definition.—A form of intestinal catarrh of children one to five years old, of insidious onset, and characterized by copious, offensive, loose, frothy stools, resembling oatmeal gruel in color and consistence. It was first described by Gee.

Etiology.—This is unknown. Ulceration of the intestine has been found, but there is no definite morbid anatomy. Occasionally ulceration of the intestine has been met.

Symptoms.—The symptoms, in addition to those named, are progressive *wasting* and *weakness*. There is *no fever*. The abdomen is distended as by flatus, but is inelastic, doughy.

Prognosis.—It is commonly fatal. It has been likened to the *hill diarrhœa* of tropics, which affects adults.

Treatment.—This can only be symptomatic.

CHOLERA MORBUS.

Synonyms.—*Cholera nostras; Sporadic Cholera.*

Definition.—An acute gastro-intestinal catarrh, characterized by profuse vomiting, purging, and painful cramp.

Etiology.—The intensity of the symptoms and their similarity to those of true cholera justify a suspicion that a specific organism is responsible for *cholera nostras* as well as for true cholera. No single bacillus has, however, been settled upon, although there is also found in the discharges with considerable constancy the bacillus known as the Finkler and Prior bacillus, which closely resembles the "comma" bacillus of true cholera. The disease may result from toxines generated by a

variety of bacilli, but until more definite proof is brought forward, cholera morbus must be regarded as a severe form of catarrhal enteritis associated with gastritis due to some poison generated by the noxious substances causing it. Such are indigestible and decomposed articles of food, unripe fruit, especially mixtures of fish, salads, and fruit. Especially frequent are these attacks in the hot weather of July and August, though cold and dampness are also regarded as predisposing causes. So are fatigue and a debilitated state of the system. Young adults and persons in the prime of life are more frequently victims than either the very old or young.

Morbid Anatomy.—This is in no way different from that of catarrhal enteritis and visible alterations are not always apparent. The same shrunken, ashen appearance of the skin characteristic of cholera may be found in fatal cases of cholera-morbus.

Symptoms.—The victim of cholera-morbus is commonly seized suddenly, often at night, with *severe cramp*, *vomiting*, and *purging*. The first vomitus is the food last ingested, but this is rapidly succeeded by bilious matter, and still later by almost pure water. The same may be said of the bowel discharges, which follow each other in rapid succession—in fact, become at times almost continuous. They present ultimately all the physical characters of the rice-water discharges of true cholera.

The *pains* at first are confined to the abdomen, the paroxysms succeeding each attack of vomiting. Later they extend to the muscles elsewhere, especially those of the calves of the legs.

Corresponding to the loss of water is *thirst*, often intense. The patient is *restless* and *anxious*. Collapse may supervene and the *skin become cold*, *clammy*, and *ashen-hued*, the *eyes deeply sunken*, the *pulse frequent* and *feeble*. There is not often fever, though the internal temperature is higher than that of the surface. The mind remains clear even in the event of a fatal termination almost to the end, when it may become clouded.

Diagnosis.—This was fully considered when treating of cholera, to the section on which the student is referred. The symptoms caused by over-doses of arsenic, antimony, and the poisonous mushroom are similar.

Prognosis.—This is usually favorable, the gravest cases recovering as a rule. A single night commonly measures the duration of an attack. Fatal cases, however, occur, the very old and the very young being most often victims. Prompt treatment is of the utmost importance, as it will usually cut short an attack which will otherwise last twenty-four to thirty-six hours and be succeeded by a slow convalescence.

Treatment.—Opium is almost indispensable to the successful treatment of an attack of cholera morbus. The happiest method of exhibition is by the hypodermic needle, the more especially because everything given by the mouth is apt to be promptly rejected. For an adult less than a ¼ grain (0.0165 gm.) of morphine is hardly to be thought of. On the other hand, such a dose will often act magically. It should be associated with diffuse counter-irritation over the abdomen by mustard, while the hot bath may be added if the symptoms do not yield.

In the absence of the hypodermic needle, remedies must be given by the mouth. The association with morphine of the hot aromatics, such as ginger and cloves, seems to aid its retention. Hence the efficiency of the various forms of "cholera drops," the formulæ for some of which are given under cholera. Chlorodyne is an admirable remedy. Unfortunately, the preparations by different pharmacists are not of uniform strengths. On the other hand, the doses are commonly indicated on the labels, and it is safe to say they may be usually doubled without harm to the patient.

The nausea may be controlled by ice, cold carbonated waters, by pieces of ice swallowed whole, or by champagne. The latter is particularly happy when stimulants are needed, as constantly happens. Where there is a tendency to collapse, whiskey and ether may be injected under the skin, while enteroclysis and even hypodermoclysis may be needed for the same reasons as in true cholera—the restoration of the water lost from the system.

PSEUDO-MEMBRANOUS ENTERITIS.

Synonyms.—*Croupous Enteritis; Diphtheritic Enteritis.*

Definition.—A rare variety of intense inflammation affecting either bowel, and characterized by the formation of false membrane.

Etiology.—Pseudo-membranous enteritis occurs in connection with such infectious diseases as pyæmia, pneumonia, scarlet fever, and even typhoid fever; also from the toxic effect of mineral poisons, such as lead, mercury, and arsenic, and during the cachexias which develop toward the close of cancer, Bright's disease, cirrhosis of the liver, and the like.

Morbid Anatomy.—The false membrane present varies in extent and depth. It may be limited so as simply to tip the villi and valvulæ conniventes or other folds with a greyish-yellow film, or the coagulation necrosis may infiltrate a greater depth in flake-like patches, or it may invade the follicles and solitary glands, which may suppurate. To the false membrane is commonly added a hyperæmic basis. The deep-seated diphtheritic inflammation found in diphtheritic dysentery is elsewhere described.

Symptoms.—These may be so slight as to be unnoticeable. At other times there is *diarrhœa* and *abdominal pain*, but nothing distinctive.

Treatment.—This is symptomatic and that of the attending and causing disease.

Phlegmonous Enteritis.—This is a rare disease, consisting in a diffuse suppurative infiltration of the submucosa, analogous to phlegmonous inflammation of the stomach. It has been found after intussusception and strangulated hernia, and may cause symptoms of peritonitis by invasion of this coat of the bowel, but there are no symptoms by which it can be recognized before death. It has been met in the duodenum.

HEMORRHAGIC INFARCT OF THE BOWEL.

Definition.—Hemorrhagic extravasation in the wall of the small intestine, due to embolism or thrombosis of the mesenteric arteries.

Etiology.—A warty vegetation from coexisting valvular heart disease may become the embolus, or the latter may arise from the clot in an aneurism of the aorta.

Morbid Anatomy.—There is congestion, infiltration, and swelling of the jejunum and ileum, and the superior mesenteric artery will generally be found plugged with a clot, which may be preceded by an embolus. The mesentery may also be the seat of congestion and infiltration.

Symptoms.—There may be sudden *nausea*, *vomiting*, *faintness*, *abdominal tympany*, and *pain*. There may be symptoms of *obstruction*, or *diarrhœa* with *blood-stained stools*.

Diagnosis.—The condition is so rare that infarction is not apt to be thought of. But should there be valvular heart disease or aneurism, the sudden occurrence of the symptoms mentioned ought to excite suspicion of the cause.

Prognosis and Treatment.—The prognosis is invariably fatal in severe cases, and though the occlusion of a small vessel may be followed by recovery, there is no treatment which will avail further than to abate the symptoms.

ULCERATION OF THE BOWEL.

What is Meant.—Apart from ulceration symptomatic of typhoid fever, dysentery, tuberculosis, and chronic enteritis, we are not often called upon to recognize this lesion, while its presence is often unattended with any symptoms whatever. The ulceration of typhoid fever, dysentery, and follicular enteritis requires no further reference; nor the peptic duodenal ulcer, which was considered in connection with gastric ulcer; nor tubercular ulceration secondary to tuberculosis elsewhere, which may be said to be probable wherever such tuberculosis becomes associated with obstinate diarrhœa, uncontrollable or only partly controllable by medicines. Such probability may be confirmed or not by the finding of bacilli in the fecal discharges, to which end cultures should also be made. At the same time it is to be remembered that bacilli found may have been swallowed with sputum, a source more likely if the patient is known to habitually swallow sputum.

Tubercular Ulcer.—Occasionally tubercular ulcers occur primarily or without preceding symptoms of tuberculosis elsewhere, especially in children. They are seldom, if ever, below the ileum and appendix vermiformis. There is no way of discovering them during life.

In the first place, such ulceration is hardly suggested unless there is some discharge from the rectum of the nature of diarrhœa, with or without hemorrhage. Given, however, such symptoms with tenderness in the region of the ileum, decided fever, pronounced emaciation, and a tubercular history, the feces should be examined for tubercle bacilli,

the finding of which would be conclusive in the absence of the possibility of their being swallowed. On the other hand, their absence would not exclude tuberculosis. The sago-like clumps of mucus, formerly considered pathognomonic of tubercular ulceration, are no longer so regarded, since they are found in cases where autopsy has established the absence of any ulceration whatever. The presence of enlarged mesenteric glands palpable through the abdominal walls would be a further confirmation. Especially justified would be the suspicion if to the diarrhœa the symptoms of circumscribed peritonitis, viz., tenderness, impaired percussion resonance, and perhaps slight fever, are added, or if there are the symptoms of general peritonitis.

A rare but acknowledged seat of the tubercular ulcer is the appendix vermiformis, a rupture of which might cause any of the varieties of perityphlitis and peritonitis due to perforation in appendix disease, including post-peritoneal abscess invading the neighborhood of the kidney and producing one of the forms of perinephritic abscess. A similar termination may follow perforation of any form of ulcer of the bowel suitably situated, as in the posterior wall of the ascending and transverse parts of the duodenum, and ascending and descending colon. The presence of tubercular ulcer is not incompatible with cicatrization, which may even produce stenosis of the bowel.

Syphilitic Ulcer.—Syphilitic ulceration is confined almost entirely to the rectum, and is not very common here. Its possible presence in the colon and ileum is simply to be remembered, as its diagnosis is out of the question. The suspicion of its occurrence in the rectum is justified under circumstances of rectal discharge including blood and pus with symptoms of irritation prolonged and yet not due to carcinoma. These ulcers arise as primary sores and papules or from breaking down of gummy tumors. They are characterized by their serpiginous outline, an indisposition to heal, and the presence of condylomata about the anus, usually the broad but rarely also the pointed variety.

Embolic Ulcer is another possible variety of intestinal ulcer, though not recognizable before autopsy. It is a possible event where valvular heart disease exists or septic pyæmia, or it is possible that a branch of an intestinal artery may become the seat of lodgment of an embolus from the heart or from some septic focus and be followed by necrosis and solution of the area supplied by it.

Treatment.—In addition to the general treatment of tuberculosis, the diarrhœa occasioned by the ulceration should be treated by the usual remedies, among which should be included nitrate of silver and sulphate of copper with opium.

The subject of syphilitic ulceration should receive the specific treatment of syphilis, while the ulcer, if accessible, should be treated by local applications of silver nitrate in solid stick.

APPENDICITIS.

Definition.—An inflammation of the vermiform appendix, catarrhal, ulcerative, or interstitial, which commonly extends to the structures lying in contact with it, producing

(1) A peritonitis, which is plastic and limited—peri-appendicitis.

(2) Circumscribed suppuration or abscess—para-appendicitis.

(3) Septic and general peritonitis. Perforation and gangrene are often intermediate incidents.

The word appendicitis, which is now by almost unanimous consent applied to the disease under consideration, did not secure the application without a struggle. The term typhlitis, so long employed, was adopted because it was thought that the disease began in the cæcum. Modern studies go to show with almost absolute certainty that true appendicitis never begins in the cæcum, but that the appendix in all cases is the root of the evil. Inflammation and perforation of the cæcum is, however, a possible event, though it is not clinically separable from appendicitis. It also often happens that the earliest symptoms by which appendicitis is recognized are those of inflammation of the peritoneum covering the appendix and adjacent cæcum, but the existence of very positive disease of the mucous membrane of the appendix has been demonstrated over and over again where the peritoneum has not been invaded. It is, therefore, likely that the process begins in the appendicular mucous membrane each time. The term appendicular peritonitis or peri-appendicitis is a good one for the inflammation of the peritoneal covering of the appendix, while perityphlitis or para-appendicitis is equally suitable for the more extensive peritonitis about the cæcum, and the term perityphlitic abscess indicates well that the same inflammation has gone on to pus formation.

Historical.—None of the facts bearing on the nature of appendicitis are of very old date, while the correct notion of its nature may be said to be quite recently established. The first recorded case of perforation of the vermiform appendix appears to have been by Mestivier, in 1759, caused by a large pin in the appendix; another was reported by J. Parkinson, an English physician, in 1812; another by Wegeler, in 1813. In 1824 Louyer-Villermay reported a case of fatal peritonitis which he ascribed to perforation of the appendix. In 1827 Melier reported four cases, three of perforative appendicitis and one of relapsing appendicitis. Melier even suggested the possibility of curing the patient by operation provided the diagnosis could be sufficiently established. Other isolated cases of fatal inflammation of the appendix were published from time to time, but the first systematic article was prepared by Husson and Dance, in 1827, at the suggestion of Dupuytren, and the views promulgated by them were apparently those of the great surgeon himself, since they are the same as those he published six years later in his "Lectures on Clinical Surgery." He treats of irritation and inflammation of the mucous membrane of the cæcum, extending thence to the retro-cæcal tissue and thence rarely to the peritoneum. The appendix is totally ignored. In 1830 Goldbeck, at the suggestion of

Puchelt, of Heidelberg, wrote his graduation thesis, "On a Peculiar Inflammatory Tumor of the Right Iliac Region." He adopted the views of the French authors and called the disease perityphlitis. He also recorded a case of perforation of the appendix with resulting peritonitis. He says, moreover, that in fatal cases of perityphlitis the appendix has been found intact.

In 1831 J. M. Ferrall published a paper, said to have been written several years earlier, on "Phlegmonous Tumors in the Right Iliac Region," in which the cæcum is also held to be the primary seat of the phlegmon, which is described as extending thence to the connective tissue behind it, the peritoneum being accorded a minor rôle.

In 1834 James Copland, in his "Dictionary of Practical Medicine," describes what we now know as perityphlitis under the title, "Inflammation of the Cæcum." He, moreover, recognized the appendix as a possible primary seat of disease, due to foreign bodies in it and terminating in gangrene—a great advance over Dupuytren. John Burne came still nearer the truth in 1837 and again in 1839. Though he wrote on "Inflammation of the Cæcum," even in his first paper he speaks of "ulceration of the appendix" set up by foreign bodies, such as raisin seeds, cherry stones, and concretions, of possible perforation resulting in general peritonitis or local peritonitis, with abscess. In his second paper he goes farther and states his belief that all Dupuytren's cases were due to disease of the appendix. He introduced the term *tuphlo-enteritis*.

In 1838 J. F. H. Albers retrograded a little. Publishing a paper on inflammation of the cæcum, and introducing the term *typhlitis*, which he divides into acute, chronic, and stercoral typhlitis with perityphlitis, he distinguished the latter affection from typhlitis, with which he says Puchelt and others confounded it. But while recognizing the possibility of disease starting in the appendix and going on even to perforation, he regarded the phlegmon of the right iliac fossa as more frequently due to disease of the cæcum. In the next year Grisolle, appreciating correctly the rôle played by perforation of the appendix in causing the iliac phlegmon and abscess, opposed the teaching of Albers and claimed that the cæcum could not cause the grave effects ascribed to it, since dysenteric and other well-recognized forms of ulceration of the same structure show no tendency to extend into the neighboring connective tissue. Grisolle, as though under the thraldom of Dupuytren and the French school, however, still assigned an important rôle to the cæcum.

From this time, however, and, indeed, from the date of Burne's paper in 1837 to the present, appendicitis has been an acknowledged disease, but it has seemed almost impossible, even up to this day, to shake off the idea of a typhlitis as a responsible factor in the phenomena of appendicitis. Louyer-Villermay in 1840 reported some cases of rapidly fatal inflammation and gangrene of the appendix. In 1843 A. Voltz published a retrospective paper entitled "Ulceration and Perforation of the Appendix," occasioned by foreign bodies. He concluded that the appendix was the organ at fault in all cases previously published, and apparently for the first time the cæcum and retrocæcal tissues are ignored.

Simple catarrh of the appendix was first recognized by Rokitansky

in 1843 in his classic work on "Pathological Anatomy." He ascribes it to the irritation of fæcal matter and to concretions, and contrasted it with the more intense processes of gangrene and perforation. Such inflammation, he says, may become chronic or go on to ulceration. He also refers to the benign effect of inflammatory adhesions in protecting against general peritonitis in the event of subsequent perforation. He still admitted the existence of catarrhal inflammation of the cæcum, ulceration and perforation of the latter, with inflammation of the postcæcal tissue as a consequence. So G. Lewis in 1856 ascribed the less serious consequences, including, however, suppuration, to typhlitis, while the violent and fatal cases, he said, began with appendicitis, induced always by concretions. In 1858 C. Wister attached further importance to the part of the appendix in producing the symptoms in question. In this year, too, Oppolzer suggested the name *paratyphlitis* for that form of iliac phlegmon which was extraperitoneal; *i. e.*, between the iliac fascia and bone.

Samuel Wilks was one of those who appreciated the rôle of the appendix. Thus, in the treatise of Wilks and Moxon on "Pathological Anatomy" in 1875, he says, referring to the terms cæcitis, typhlitis, and perityphlitis: "It is not clear, however, that any one particular form of disease is intended by those who make use of these expressions. The cases to which these names are given frequently occur clinically and recover; but when disease in the same region with similar characters proves fatal, we find usually some prior morbid process in the appendix rather than in the cæcum itself." Also, "the suddenness of the attack of cæcitis, and the local peritonitis following, even in the large number of cases which recover, all point to the appendix as being the most frequent cause." But he says, also: "Inflammations of the cæcum itself do occur, and apparently are sometimes caused by the continuous lodgment of hard fæces in this part of the intestines. Such inflammations, by ulcerating the mucous membrane, lead to perforation and local peritonitis, forming fæcal abscess which may discharge inward, but we believe that this is comparatively rare." Dr. Wilks's most recent views are perhaps best expressed by C. Hilton Fagge, who, in his "Practice of Medicine," edition of 1886, says: "Dr. Wilks has repeatedly expressed to me the opinion that in both 'typhlitis and perityphlitis' the disease begins in the appendix, and that variations in the intensity of the morbid process are the real cause of the supposed distinction between them. And, so far as I can learn, all the evidence which morbid anatomy affords points strongly in that direction."

C. With, of Copenhagen, was, however, apparently the first to deny pointedly, in 1880, that peritonitis ever originates in typhlitis. Reginald H. Fitz, in a timely and exhaustive paper read before the Association of American Physicians in 1886, admitted, as an extreme rarity, a primary perforating inflammation of the cæcum with which appendicitis may be confounded. In a second paper in 1888 he concluded, essentially, that the conditions described as typhlitis, perityphlitis, paratyphlitis, appendicular peritonitis, and perityphlitic abscess are varieties of one and the same affection—*appendicitis.*

The text-books published prior to 1892 very generally treat of typhlitis as an important factor in producing the ultimate phenomena of

what is now known as appendicitis, unless we except that of Dr. Fagge, already quoted, who, while he uses the word typhlitis, evidently means by it disease of the appendix. Ziegler, in his "Pathological Anatomy" (1885), also uses the word typhlitis for appendicitis. William Osler, in his edition of 1892, says that the terms "perityphlitis and paratyphlitis should be altogether discarded, as the cases are, with rare exceptions, due to disease of the vermiform appendix," and says also of "typhlitis, or inflammation of the cæcum," that it is a "doubtful and uncertain malady, the pathology of which is not known, but which, clinically, is still recognized by authors." William Pepper, in the "Text-Book of Medicine by American Teachers," 1894, treats of typhlitis as an affection very much less common than formerly supposed, because "the majority of cases of acute disease in the right iliac fossa are in reality appendicitis."

Four special treatises of great value have recently been published in English: "The Pathology of the Vermiform Appendix," by T. N. Kelynack, of Manchester, England, in 1893; "Appendicitis," by George R. Fowler, of New York, in 1894; "Diseases of the Vermiform Appendix," by Herbert P. Hawkins, of London, in 1895; and "A Treatise on Appendicitis," by John B. Deaver, of Philadelphia, in 1896. Hawkins summarizes the situation in the following proposition, to the confirmation of which American surgery has largely contributed: "In fact, it will be generally allowed that a perforating ulcer of the cæcum, though it does certainly occur, is of so rare occurrence that it may be disregarded;" also, "There is ample evidence that appendicular disease is, at any rate, of frequent occurrence; and this frequency, moreover, is sufficiently frequent to justify us in regarding the appendix as the sole cause of all cases of perityphlitis, mild or severe."

Pathology and Morbid Anatomy.—The etiology of appendicitis will be more easily understood if its morbid anatomy is first considered. Modern studies establish the existence of three degrees or stages of appendicitis:—

(1) Catarrhal appendicitis.
(2) Ulcerative appendicitis.
(3) Interstitial or parietal appendicitis.

(1) *Catarrhal Appendicitis.*—Our knowledge of this is based upon the systematic minute study of cases which come to autopsy from other causes, as well as from operation. In the acute form there is a shedding of the epithelium of the mucous membrane with detachment, partial destruction, and extrusion of the follicles of Lieberkühn, with some cellular infiltration of the retiform tissue at their base. The lumen of the appendix contains mucus, leucocytes, exfoliated cells, and casts, more or less perfect, of the crypts, with granular débris from the same sources. In more advanced stages the basement membrane is broken and dislocated, the retiform tissue more closely infiltrated with leucocytes, and the internal surface ragged and uneven. A still more advanced degree shows the mucous membrane completely infiltrated with cells and thickened. The most important fact as to catarrhal appendicitis is, that all three stages offer vulnerable foci for the attacks of pathogenic bacteria and starting-points of an infectious peritonitis. On the other hand, by the union of the opposing surfaces, obliteration of the lumen

of the tube may take place, by which it is rendered immune against further attacks. A natural cure has, in a word, been effected. The obliteration may be partial, producing stricture, beyond which a cystic distention of the tube, generally near the cæcal end, is not infrequent.

(2) *Ulcerative Appendicitis.*—In this stage the mucous membrane and submucous tissue are destroyed to various depths, while it may even culminate in perforation. It is often associated with a concretion or a foreign body. The latter is now acknowledged as much more rare than was formerly supposed. The error was a natural one, owing to the close resemblance of fæcal concretions to seeds, grains of wheat, cherry stones, and even date stones, as the result of a gradual moulding of shape and loss of water. The concretions are sometimes also the seat of deposit of lime salts. They may be multiple. They may be in the appendix a long time without producing harmful effect, the patient dying of other causes. The same is true of foreign bodies, which do, of course, occur and include the objects already mentioned. Fæcal concretions are found in from 35 to 50 per cent. of cases, foreign bodies in a much smaller number, say seven to 12 per cent.

(3) *Interstitial or Parietal Appendicitis.*—This stage may succeed upon either of the two stages just described, but rarely it may arise *de novo* by infection along the lymphatics. In the former event it starts in the abraded or ulcerated surface described, in the latter in the interior of the appendix wall. It is commonly associated with necrosis or gangrene of the wall, but may prove fatal before the necrosis sets in. The appearances vary greatly. They may be limited to a mere point, scarcely visible, and between this and sphacelation of the entire organ there is every intermediate degree. The gangrenous organ is, however, usually enlarged and distorted. The virulence of the appendicular peritonitis is, however, just as great where there is no necrosis. The peritonitis, which ensues on perforation of the appendix, is virulent, resulting from the invasion of the peritoneum by myriads of bacteria in the fæcal matter set free at the time of rupture of the bowel.

The minute appearances are as varied as the macroscopic, but Hawkins's summary of three more distinctive stages or degrees of interstitial appendicitis may be accepted as nearly correct. The cases which succeed on the catarrhal or ulcerative form are, of course, characterized by the loss of tissue characteristic of them. To these succeed destructive necrotic processes in the deeper structures of the wall. In the first stage the inflammation is characterized by necrosis of the muscular coats, the second by suppuration in them, and third by their infiltration with leucocytes and inflammatory exudation. The first is, by far, the most common. In all three, bacteria are found in the mucous and muscular coats, and all three are alike followed by virulent peritonitis.

The appendix may also be the seat—indeed, is not a very infrequent seat—of tubercular ulceration also followed by perforation. I have lately seen a remarkable specimen of this kind in which no symptoms were present before death. So, too, a typhoid ulcer may form in the appendix and perforate with the formation of a tumor mass in the right iliac region. Follicular abscess may exist and occasion the usual symptoms of appendicitis. Actinomycosis has also occurred in the appendix

with the formation of retrocæcal abscess and metastatic abscess of the liver.

Superadded to these conditions is often a localized or general peritonitis, the development of which, in the majority of cases, constitutes the attack of appendicitis. In lesser degrees of the peritonitis (peri-appendicitis) the adhesions which form are limited to the appendix and adjacent serous tissues, limiting the inflammation and acting as a barrier against general peritoneal infection. In higher degrees the inflammation attacks the tissues in relation to the appendix (para-appendicitis) and forms the iliac phlegmon or tumor. This occupies the right iliac fossa, and is variously constituted. It may consist of serous and cellular exudation, which mat together coils of small intestine and the cæcum. Or there may be a massive accumulation of cells and liquid, constituting abscess. Even the latter, as well as the more solid exudate, may be absorbed. On the other hand, the appendicular or perityphlitic abscess may rupture into the peritoneum, not infrequently producing fatal general peritonitis. The amount of pus varies. There may be a drachm or two (four to eight c. c.), or a pint (a liter) or more. More commonly there are two to four ounces (60 to 120 c. c.). The pus is usually thin and very fetid; at times it is thick, yellow, and odorless. It may be mixed with fæcal matter. The pus may have escaped into the bowel, bladder, or vagina, or externally at some point in the abdominal wall, as the navel or groin, as in a case of my own, or through the obturator foramen into the hip or thigh. The iliac muscle may be destroyed and the ilium bared. The abscess, usually in the iliac region, may be in the lumbar region or perinephritic, in the true pelvis, or under the liver. These very diverse sites are commonly determined by erratic situations of the appendix. There may be secondary abscesses of the liver by pylephlebitis or portal embolism. These may have all the terminations possible to hepatic abscess.

If general peritonitis supervene, there are added the usual anatomical appearances incident to this condition—flakes of lymph scattered over the intestines, binding the latter together, with pus in the flakes in varying quantity.

Etiology.—*Exciting Causes.*—All three stages of appendicitis described are probably due to the invasion of microörganisms, while the foreign bodies, concretions, and other agencies to be mentioned are to be regarded as predisposing causes, furnishing the conditions favorable to the operation of the pathogenic bacteria.

A word as to the nature of the organisms which are responsible for the virulent forms at least. The *bacillus coli communis* is a bacterium whose natural habitat is the colon of healthy individuals, cultures of which from the normal colon prove harmless when injected. Yet cultures of this same bacillus taken from cases of virulent appendicitis produce also corresponding virulence. Whence it may be inferred that in some way virulence is engendered in an otherwise harmless bacillus. There is good reason to believe that such bacilli may pass from the intestines to the peritoneum through the lymph spaces in an intestinal wall which is simply damaged, as well as through a perforation. Thus, many cases of so-called idiopathic peritonitis, or peritonitis where macroscopic examination reveals no evident lesion, may still be due to the bacteria of

appendicitis from the interior of the tube. This has been actually demonstrated in some cases, and it is not unlikely that it will be found true of all such cases thoroughly studied.

While in most instances the bacillus coli communis has been found in pure cultures, pyogenic bacteria have been found associated with it. The most important of these is the *streptococcus pyogenes*, and after this the *staphylococcus pyogenes aureus* and the *proteus vulgaris;* so that the existence of more than one possibly infecting species may be admitted.

Predisposing Causes.—The most important predisposing cause of appendicitis is the appendix itself. An organ without use, and therefore undeveloped and feebly nourished, is correspondingly feebly resisting to all disease. Its anatomy is also such that the entrance of irritating matters is easier than their exit, while inflammatory products are not easily evacuated. As predisposing causes, too, must be considered certain influences which formerly were regarded as exciting causes, such as overeating, especially of unwholesome and indigestible food, acute indigestion from any cause, in addition to the foreign bodies and concretions already mentioned. It cannot be said that the precise mode of operation of such cause is certainly known. It may be that a hyperæmia or deranged circulation thus induced produces a condition favorable to the action of incessantly present bacteria. Similar is the effect of fatigue, cold, and traumatic causes, such as blows and contusions. Influenza should be mentioned as one of the causes in this category to which appendicitis has been apparently traced. The same may be said of typhoid fever.

Appendicitis is a disease of children and young adults. From 50 to 55 per cent. of cases occur under the age of twenty, 30 per cent. between twenty and thirty, and 15 per cent. under fifteen. What bearing the fact that the appendix is longer in children and young adults has upon this can only be surmised. Nearly 80 per cent. of all cases occur in males. It has been suggested that this is because the lumen of the appendix is larger in males, and therefore more liable to receive fæcal or foreign matters. Attacks have occurred, however, in the first year of life and as late as seventy-six. More cases occur in summer than in winter. Occupation has no effect in exciting it, but after a first attack recurring attacks of appendicitis are more frequent in men who do heavy work, such as porters and carriers, or men who stand long each day on their feet.

Symptoms.—Simple catarrhal appendicitis is often unattended by any symptoms whatever. The same is true of many cases of ulcerative appendicitis before the peritoneum is reached in the invasion. Other symptoms more or less mild and vague are on this account overlooked. The interstitial variety, consisting, as it does, in a simultaneous involvement of all the tissues, gives rise promptly to serious symptoms. In point of fact, as already stated, we know appendicitis to be present, perhaps in a majority of cases, by the symptoms of the resulting peritonitis, local or general. Though in most instances a first attack is a mild one, yet no one knows at the onset whether this is going to be the case or not. Furthermore, it is often impossible to say when suppuration has taken place. The supervention of general peritonitis is, however, usually attended by unmistakable symptoms.

The first symptom is invariably pain, *sudden pain.* Its location at first is not constant. It may be anywhere in the abdomen. Most frequently, perhaps, it is in the neighborhood of the umbilicus. At other times it is in the epigastrium ; at others, diffuse. It is intermittent, or at least remittent. Usually, within the first twenty-four hours, it settles itself in the right iliac region, where it remains. It may then be mild or severe, more frequently it is moderately severe. Even at this stage its location is not always constant. It has even settled in the left iliac fossa, under the liver, or beneath the spleen, anomalous situations for the appendix. This pain is increased by coughing or taking a long breath or turning over on the side.

As constant as pain is *tenderness* in the right iliac region, or if the appendix happens to be placed in one of the unusual situations named, it will be in that situation. Rather strong pressure may at times be necessary to elicit it, but usually moderate pressure suffices. Its extent varies. It may occupy the whole lower quadrant of the abdomen or may extend up to the costal margin and around into the flank, but the seat of maximum tenderness is oftenest a point known as McBurney's—a point at the intersection of a line drawn from the anterior superior spinous process of the ilium to the umbilicus and another along the right edge of the rectus muscle. It is one and one-half to two inches from the anterior superior spinous process of the ilium. The patient almost invariably assumes the dorsal decubitus, often with the right leg drawn up, because of the relief thus afforded.

The third cardinal symptom, if the patient come under notice sufficiently early, is *rigidity* of the right *rectus abdominis* muscle and other muscles overlying the focus of inflammation. This may be associated with a slight distention of the entire abdomen. This primary tenseness, after two or three days, may be substituted by a *tumor.* The latter varies in size and shape, but is more commonly oval and about as large as a hen's egg, with its longer axis parallel with the upper part of Poupart's ligament. It may be much larger, occupying also the whole lower left quadrant and extending upward and backward into the flank, while its shape may be quadrilateral or triangular. It varies in consistence. Its composition has been described in considering the morbid anatomy of the disease.

There is usually *impairment of resonance* to percussion over such a tumor, though less than might be at first expected. This is because we are really percussing over hollow organs, though matted together by exudation. At times, however, there is a duller note, while at others it may even be natural. In the latter event the tumor is small. Indeed, tumor may be altogether absent, but this can never be be said of tenderness.

Vomiting is a symptom of more or less frequency. It is commonly regarded as reflex and is variously severe. The matters vomited are first the gastric contents, with the evacuation of which the vomiting usually ceases, though it may recur with the event of perforation or rupture of abscess. If the symptom is more prolonged, the vomited matter becomes greenish. Many so-called "bilious attacks" of past times have really been attacks of appendicitis.

Constipation is present in a decided majority of cases from the beginning of the attack. It is due to paralysis of the bowel, and may be so obstinate as to simulate obstruction of the bowel, being even attended at times with stercoraceous vomiting. Indeed, appendicitis has often been confounded with obstruction. On the other hand, there may be diarrhœa, recurring with each successive attack. There is loss of appetite. The tongue at first may be natural, but later becomes more or less coated, and in advanced stages dry.

There is always *fever* at the outset, the temperature 102°, 103° F. (38.9°, 39.4° C.), and even 104° F. (40° C.), rarely higher, after which it gradually falls, reaching the normal in five to seven days in favorable cases, which terminate in resolution. The pulse rate corresponds with the degree of fever, but its force and volume vary with the patient's strength. Should suppuration take place, the temperature continues with but slight fall, or may even rise further. (See Fig. 17.) *Suppuration may, however, be unattended with fever.*

Nor does a sudden fall of temperature always mean the establishment of convalescence. Not very rarely the event has a widely different meaning. It means that instead of convalescence perforation has taken place. It is extremely important that this fact should be realized. More than once have I known the physician to have been misled by it. The following temperature chart illustrates such a case. (Fig. 18.) Another even more unusual explanation of sudden fall of temperature is the rupture of a small abscess into the bowel. *Finally, too much stress cannot be laid upon the fact that there may be gangrenous appendicitis in the presence of normal temperature.*

The *urine* is *scanty*, as is usual in fever, and quite frequently contains an abnormal quantity of indican. It is rarely albuminous, unless there be high fever, when there may be the small albuminuria characteristic of fever. There is often irritable bladder and frequent micturition.

The *expression* of the patient varies with the severity of the symptoms, but seldom exhibits the anxiousness characteristic of peritonitis, unless the latter actually is present in consequence of perforation or abscess rupture.

Is there any surer information of the event of *suppuration* than that furnished by the temperature as discussed? Fluctuation will, of course, be thought of, but this is rarely obtainable on account of the depth and mode of distribution of the pus. The pus may come to the surface and be thus recognized, but not often; and, furthermore, a case that has been allowed to proceed to this degree at the present day has not been properly handled. The rigor and sweat, such valuable evidence of the occurrence of suppuration under other circumstances, is, as a rule, wanting in appendicitis. Rapid growth of the tumor and the attainment of large size in a short time point to suppuration. But the most valuable sign is *the presence of exquisite tenderness over the focus of inflammation.* Continued high temperature is significant, though it may be wanting. Fully formed abscess has been found as early as the third day. More commonly six to eight days elapse before a diminished tenderness and slight decline of swelling point to this formation. Appendicitis allowed to go on to suppuration, *i. e.*, not relieved by operation, usually terminates by

rupture into the peritoneum, followed by general peritonitis and death. The event is variously delayed by the extent and toughness of the protective adhesions which may have formed about the abscess. A few abscesses rupture into the bowel, thus saving the patient's life. Two or three cases in a hundred are thus saved. The fæcal fistula, incident in

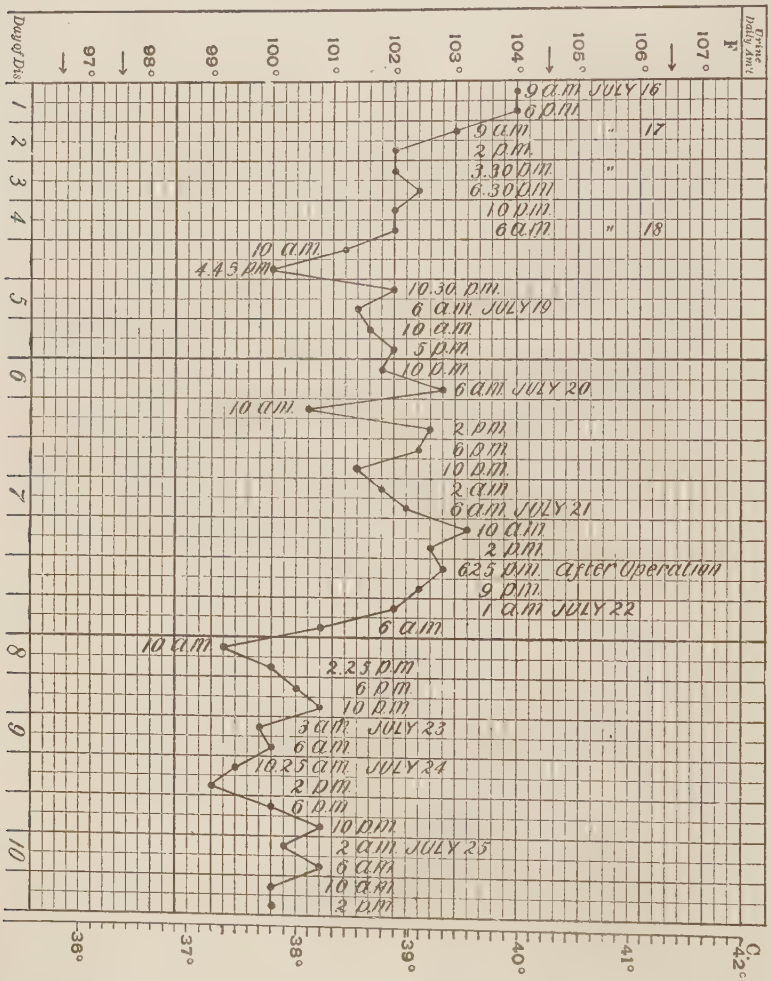


FIG. 17.—TEMPERATURE CHART SHOWING TEMPERATURE MAINTAINED BY ABSCESS AFTER PARTIAL DECLINE.

The patient was first seen by Dr. C. F. M. Leidy at 9 A. M. on July 16th, the first day of the disease, when the temperature was 104° F. (40° C.). It continued the same at 1 P. M. the same day. It then began to fall, and by 2 P. M. the next day reached 102° F. (38.9° C.). By 4.45 P. M. of the fourth day it had fallen as low as 100° F. (37.7° C.), after which it rose and fluctuated about 102° F. (38.9° C.), again rose, reaching 103.2° F. (39.5° C.) at 10 A. M. on the seventh day, when the patient was operated on by Professor John Ashhurst, M. D., and an abscess evacuated, after which the case proceeded to convalescence.

this termination, usually closes eventually, though not always. In rare instances the abscess, especially if deeply situated in the pelvis,

breaks into the bladder. The termination in these cases is less favorable, 50 per cent. being fatal. A few also break through the groin, followed by recovery. Lumbar abscess and perinephritic abscess must be mentioned as possible terminations, also infiltration of the abdominal walls and tissues of the thigh, pylephlebitis, and hepatic abscess.

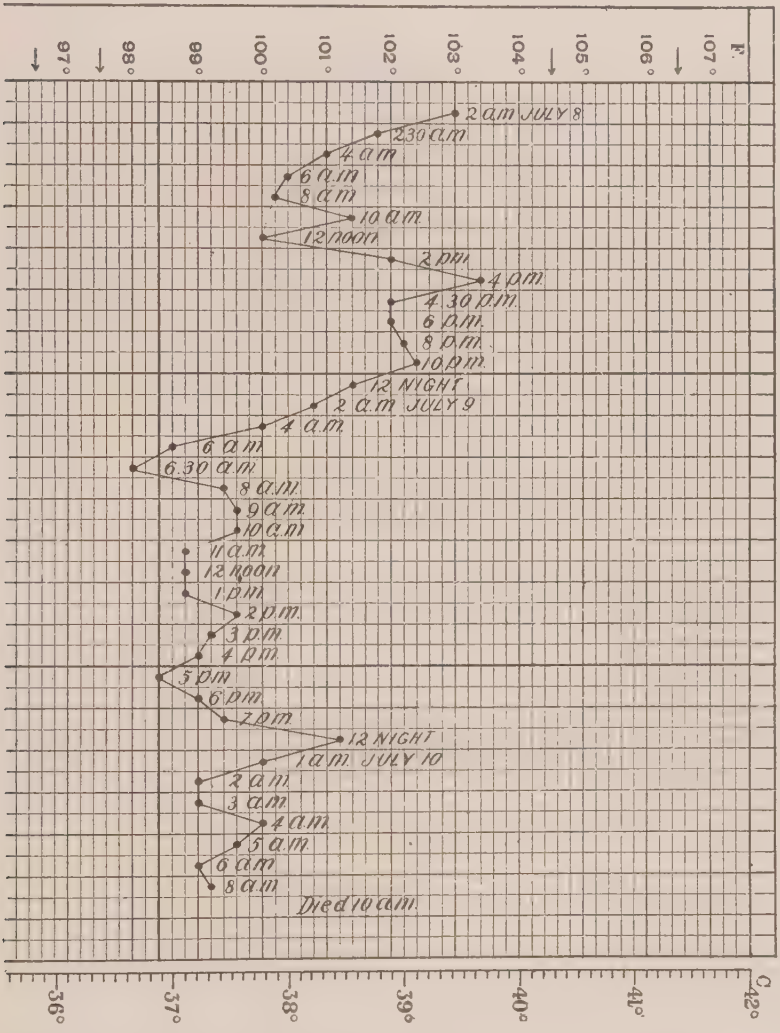


FIG. 18.—TEMPERATURE CHART SHOWING MISLEADING FALL TO NORMAL, INCIDENT TO PERFORATIONS.

General peritonitis may also ensue after perforation of the appendix. The symptoms of the resulting general peritonitis are those peculiar to this disease when suddenly induced by other causes, viz. :—

(1) Diffuse pain as contrasted with pain localized in the right iliac region ; pain of extreme severity.

(2) Generally distended and tender abdomen.

(3) Moderate fever, succeeded by normal or subnormal temperature, already alluded to as often misleading the physician.

(4) Rapid and feeble pulse.

(5) Dry and coated tongue.

(6) The phenomena of collapse, *i. e.*, cold, clammy skin, feeble pulse, anxious expression, death.

Recurring and Relapsing Appendicitis—Chronic Appendicitis.

These terms are applied to cases of appendicitis which recur after a first attack. The terms are sometimes interchangeably used, but, strictly speaking, cases are *recurring* which repeat themselves at considerable intervals, as some months or a year or more; *relapsing*, when the attacks are very close—at intervals, say, of one or two weeks, so as to make them almost continuous. In the former, to which attention was first called by William Pepper in 1883, it is reasonable to believe that the patient has recovered in the interval of the periappendicular peritonitis, while the appendicitis, catarrhal or ulcerative, has continued, or there exists a cystic appendix as an exciting cause. In the relapsing form it seems likely that there has not been complete recovery in the interval. Certain it is that one attack predisposes to another, so that, in at least 23 per cent. of cases observed, according to Hawkins, and 44 per cent., according to Fitz, it is found that there have been previous attacks. The symptoms of a recurrent attack are the same as those of a primary one. In many cases the interval between the attacks is passed in comparative comfort; in others, there is no small amount of pain or discomfort in the situation of the appendix. The term chronic appendicitis may also be applied to such cases.

Complications and Sequelæ.—The most important complication is obstruction of the bowels, by which is not intended the obstinate constipation so often met as an early symptom of appendicitis, but a true obstruction, the direct consequence of constriction by adhesions developed in the course of the peritonitis. It is an important cause of death in fatal cases, many striking cases being confirmed by autopsy, while operation frequently discloses conditions which could easily have produced obstruction.

Other complications are hepatic abscess from pylephlebitis due to thrombosis and even embolism of branches of the portal vein; also phlebitis of the right iliac vein. In abscess of the liver the diaphragm has been perforated, producing empyema and pyopericardium. Pyæmic abscesses elsewhere in the system, including the brain and lungs, have also been found in rare instances. Fæcal, bladder, and umbilical fistulæ have been referred to. Fatal hemorrhage has also resulted from necrosis of the walls of the iliac vessels. Appendicitis may occur in a hernial sac.

Diagnosis.—The diagnosis of many cases of appendicitis is easy, and becomes more so as experience increases. A certain number of cases have to be carefully weighed, and in a few diagnosis is extremely difficult. *Sudden pain*, becoming localized, *tenderness*, and *rigidity* in the right iliac region are three symptoms which, if present, point almost

unmistakably to appendicitis. The cases of difficult diagnosis are those in which these symptoms are wanting or are in unusual situations. But, in truth, these symptoms are less frequently absent than has been supposed. More frequently they are not looked for because there is very little to draw attention to them. A rule should, therefore, be made to examine carefully for them in any person subject to gastro-intestinal attacks, however induced and however manifested. It is certain that some cases of so-called catarrhal enteritis are really cases of appendicitis.

Differential Diagnosis.—Intestinal obstruction is a condition with which appendicitis has been sometimes confounded. The special symptoms of the various causes of obstruction, whether those of strangulation by bands or twists, by intussusception, or by tumor or foreign body, should be recalled. Especially characteristic of obstruction is the absence of fever, unless the patient lives long enough to permit peritonitis to be set up. The pain in obstruction is more intermittent at first, and though, like that of appendicitis, it may be anywhere in the abdomen, it is not likely to localize itself in the right iliac region. The constipation is more complete in obstruction, and even the passage of flatus is usually absent. The vomiting, also, is more severe and stubborn, and is more likely to be stercoraceous. There is more general distention of the abdomen, and limited tenderness is less easily differentiated. Intussusception occurs more frequently in children younger than those subject to appendicitis, and is often attended with bloody discharges which never occur in appendicitis, while a tumor may often be felt on examination *per rectum*. Strangulation by bands or twists is more common in adults. Malignant growths, causing obstruction, are usually in the left iliac region, although cancer of the cæcum is to be remembered as a disease of the right. Its slower development distinguishes it from appendicitis. (See also Obstruction of the Bowels.)

Typhoid fever may be confounded with appendicitis, and I have more than once been startled in the course of a typhoid fever by the thought that I might be dealing with an appendicitis, especially where there has been tympany and prolonged tenderness in the right iliac region; but one has, as a rule, only to recall the mode of beginning of the illness, the gradual development of the fever, its greater intensity and peculiar diurnal variation, the spots at the eighth day, to be reassured in the majority of instances. I recall one case of typhoid fever terminating in perforation in which was simulated very closely even the iliac tumor of appendicitis. I may add that I do not think the typical spots in typhoid fever are ever closely approached by anything similar in appendicitis, though the event of suppuration is said to be sometimes indicated by an eruption. On the other hand, there is nothing to prevent typhoid fever and appendicitis from accidentally coinciding.

A question which one would naturally expect to give rise to difficulty is that between appendicitis and the pelvic affections of women when on the right side, such as a suppurating ovarian cyst around a fallopian tube or a pyosalpinx itself, yet mistakes from this cause seem to have been rare; and when we remember the deeper situation of the latter, and their usually easy recognition *per vaginam*, it is not so surprising. The onset of suppurating ovarian cyst is much more gradual, the pain

more constant and duller. Pyosalpinx is in more intimate relation with the uterus, while the history differs from that of appendicitis.

Many cases of acute appendicitis were formerly mistaken for bilious colic and acute indigestion, but the latter are unaccompanied by tumor or tenderness, while the vomiting is more stubborn and the vomited matters differ. Entero-colitis occasions colicky pains, but there is no localization, while there is diarrhœa with mucous stools. It will be remembered, however, that these symptoms sometimes attend appendicitis.

In hepatic colic the pain is higher up, in the region of the gall-bladder, while jaundice is almost invariably present, and sometimes pain under the left shoulder. In nephritic colic the pain extends from the lumbar region into the groin and testicle. A floating kidney with twisted ureter is movable as contrasted with the iliac tumor of appendicitis; there is flattening of the corresponding lumbar region, while sudden relief to symptoms which characterizes the untwist is altogether peculiar. The presence of blood in the urine is conclusive of renal origin. In pyonephrosis there is tenderness in the region of the kidney as well as pus in the urine. Perinephritic abscess occasions tenderness in the lumbar region, while the pain radiates into the groin, as in nephritic colic. It is to be remembered that perinephritic abscess may be occasioned by suppurating perityphlitis where the position of the appendix is posterior to the cæcum.

Appendicular colic or neuralgia of the right iliac fossa is a vague condition of pain in this region, which has been ascribed to peristaltic contraction of the appendix, constituting an effort to expel fecal pellets, but of which no proof is afforded, operation in several cases failing to discover anything abnormal.

Prognosis.—It is a difficult matter to consider fairly the prognosis of appendicitis, or rather of the periappendicitis growing out of disease of the appendix. For, if we separate the cases which do not go on to suppuration, recovery is apparently the rule. Thus, out of 190 cases collected by Hawkins, none died. Again, of cases treated by section and drainage after suppuration has set in, fully 25 per cent. die; while if general peritonitis supervene, 75 per cent. die.

On the other hand, it is impossible to say of any case, however mild, that if left alone it will not terminate in suppuration, while a large number of cases still perish because of imperfect diagnosis and delayed operation.

Treatment.—As soon as the diagnosis of appendicitis is established, indeed, pending its settlement, a competent surgeon should be associated with the physician, for the reason that in the vast majority of cases operative treatment is sooner or later demanded, while the hour for such treatment is best settled by daily conference. The course of cases of appendicitis is often very delusive, and the surgeon who operates frequently is likely to have seen more cases than the physician. The diagnosis being established, operative treatment should be recommended, except in those cases where the disease is so far advanced as to make it unlikely that the patient will be saved by operation. My reason for this belief is, that while a majority of cases of simple appendicitis may subside with rest, in a very large number, at least 25 per cent., the primary

attack leaves the patient predisposed to another at once more severe and dangerous than the first, while we have no guarantee that any attack will subside without suppuration, or, what is worse, without leaving the condition referred to, in which malignant inflammation or perforation may set in at any moment without warning.

It must be admitted that it is not always easy to lay down a rule by which operation shall be determined, for it is not only that we must know when to operate to save life, but also that we must know when not to operate in cases so severe that operation will be futile, and it is due the operation that it should be saved the opprobrium of such futility. Certain it is, too, that in cases where operation is of no avail death will be hastened by it, the depressing effect of etherization co-operating to hasten the fatal end. Much difficulty is, however, removed when we decide to operate *without undue haste* in all cases so soon as the diagnosis is established, except where operation will evidently be futile. I say without undue haste. For, in many cases, it is plain that a few days' delay, if the patient is kept at rest, will make no difference in the result, while, if the inflammation is subsiding, a stage is being reached in which the operation is even less dangerous, since the united experience of surgeons goes to show that the mortality of operations between attacks is practically *nil*, while that immediately succeeding diagnosis in ordinary cases is nearly so. There can be no doubt, moreover, that excision of the appendix *after* a first attack is a safer procedure than *during* a first or any attack. Even where suppuration has set in it may be safe to delay operation for a day or two while the patient is held quiescent.

When, on the other hand, shall operation be omitted because it must be followed by a result inevitably fatal? In all cases where there is diffuse septic peritonitis, rapid pulse and leaky skin, constant vomiting and constipation, operation is generally futile. In such cases saline purgatives and stimulants, diffusible and cardiac, are indicated, and rarely, though rarely indeed, recoveries have taken place.

Whatever preparation is deemed necessary for operation when decided on must be laid down by the surgeon.

Medical Treatment.—Cases must occur, however, in which from various causes medical treatment is necessary. Operation may be declined even if urgently advised, while rarely a preparative medical treatment may be necessary previous to operation.

First of all, absolute rest in bed must be insisted upon as the first essential condition of abatement of the inflammation. Many a fatal case would have been saved had this injunction been carried out.

Next, relief of pain is demanded. For this purpose opium should be avoided except in extremest cases. Only when relief cannot be secured by the ice-bag, by poultices, by hot fomentations, by mild counter-irritants, as mustard or turpentine, or by leeching, may a minimum dose of morphia, $\frac{1}{12}$ or $\frac{1}{8}$ grain (0.005 or 0.008 gm.), be given hypodermically. The objection to opium is well founded on the ground that it masks the presence of important symptoms which should be open to observation. In cases where operation is from any cause out of the question, counter-irritation by repeated blistering may be practised, and excellent results were reported under the older treatment before operation became common.

The question of the propriety of giving an aperient is a delicate one, and must be left, for the most part, to the circumstances and the good judgment of those in attendance. The result may be very happy or mischievous. Cases may be so advanced or severe that the effect of a purgative may be to cause perforation or the rupture of an abscess, but in ordinary cases or cases of moderate severity an aperient may be useful to clear up a diagnosis, while it relieves pressure, depletes the blood-vessels, and diminishes the danger of peritonitis. On the other hand, purgatives should not be aggressive and drastics should not be used. Perhaps a safe rule would be, "if there is doubt, do not purge." The best aperient is castor oil, followed, if necessary, by salines, and of these Rochelle salts or the solution of citrate of magnesium. If the stomach is sensitive, calomel in divided doses triturated with sugar of milk is the best drug. Where there is reason to believe that suppuration has set in no purgative should be given, and, as a rule, operation should be prompt toward evacuating the pus and removing the appendix at the same time. In severe cases even enemata should be avoided as tending to favor perforation and rupture.

Nourishment should be purely liquid, and of liquid, milk is the best, though animal broths are not contra-indicated. It should not be an object to force food; indeed, only the minimum sufficient should be permitted.

INTESTINAL OBSTRUCTION.

The morbid states which cause intestinal obstruction are so various and distinct that they demand more or less separate consideration. They include strangulation, intussusception, obstruction due to abnormal contents of the bowel; twists and knots, strictures and morbid growths causing acute obstruction; strictures and morbid growths and fecal impaction causing chronic obstruction.*

I. Internal Strangulation.

Synonyms.—*Constriction of the Bowel; Hernia within the Abdomen.*

Definition.—By internal strangulation is meant stricture of the bowel by inflammatory bands or adhesions, by vitelline remains, omental or mesenteric slits, adherent appendix, and the like.

Occurrence.—This is probably the most frequent cause of acute intestinal obstruction, though closely shared with intussusception. Thus Reginald H. Fitz, in America, found it in 35 per cent. of 295 cases of obstruction, as against 32 per cent. of intussusception; Duchaussoy, in France, in 54 per cent. of 347 cases, as against 39 per cent. of intussusception; while Leichtenstern, in Germany, found it in 35 per cent. of 1134 cases, as against 39 per cent. of intussusception; and Brinton, in England, found it in 33 per cent. of 481 cases, as contrasted with 54 per cent. of

* Reginald Fitz's able paper in the Transactions of the Congress of American Physicians and Surgeons, 1889, Leichtenstern's article in Ziemssen's "Cyclopædia of Practical Medicine," and Frederick Treves's book on "Intestinal Obstruction," 1884, are important modern papers to which I am indebted for much of the matter in this section.

intussusception. The percentages of Leichtenstern, Brinton, and Fitz are astonishingly close, and cannot therefore be far astray.

Etiology.—The causes of strangulation have been carefully worked out by Fitz in his 101 cases, collected from reports since 1880. Of these, in 84 the strangulation was caused by bands and cords, of which 63 were simple inflammatory bands or adhesions and 21 were vitelline remains represented by Meckel's diverticulum, or the persistent remains of vitelline blood-vessels.* The former is usually attached by the latter to some part of the abdominal wall near the navel or to the mesentery, or it may be adherent because of peritonitis. The persistent vitelline vessels may themselves be the strangulating cord in the absence of Meckel's diverticulum. Of the remaining strangulations, six were due to adherent appendix, six to mesenteric and omental slits, three to peritoneal pouches and openings, one to adherent Fallopian tube, and one to pedunculated tumor. To these must be added diaphragmatic hernia. This was the cause of strangulation in ten per cent. of Leichtenstern's cases, but Fitz found none reported between 1880 and 1888. I reported two cases of diaphragmatic hernia in 1893,† both of some standing, the immediate causes of death being due to acute strangulation.

The seat of the strangulation is in the small bowel in a decided majority of cases, nearly 90 per cent. In 83 per cent. the strangulated part lay in the lower abdomen, and in 67 per cent. in the right iliac fossa. Seventy per cent. of cases occur in males and at least 40 per cent. between the ages of fifteen and twenty, the causes in these being inflammatory adhesions twice as often as vitelline remains. Strangulation in early youth is relatively uncommon, and when it does occur it is usually caused by vitelline remains.

II. Intussusception. Invagination.

In this condition one part of the bowel has slipped into another, always from above downward, and may be readily illustrated by slipping one part of a coat sleeve into another. The external or receiving portion, known as the *intussuscipiens*, has its mucous surface in contact with the mucous surface of the middle or intermediate portion, whose peritoneal surface is in contact with the peritoneal surface of the internal or returning portion, while the two mucous surfaces of the returning portion are apposed. The internal and middle parts are called the *intussusceptum*. The resultant is a cylindrical tumor which varies from half an inch to a foot or more in length.

Intussusceptions are named in accordance with the part of the bowel involved, viz., in the order of frequency, *ileo-colic*, or ileo-cæcal, of the small intestine into the cæcum or colon; *enteric* of the small intestine into itself; *rectal* of the rectum into itself, and *colico-rectal* of the colon into the rectum. Three-fourths of all intussusceptions are in the neigh-

* Meckel's diverticulum, a remnant of the omphalo-mesenteric duct, through which, in the early embryo, the intestine communicates with the yolk sac, is a finger-like projection from the ileum, usually within 18 inches of the ileo-cæcal valve. The length of this tube is on an average three inches, while it has attained at times a length of ten inches.

† Transactions of the Association of American Physicians, 1893.

borhood of the cæcum, while 12.5 per cent. are enteric. According to Leichtenstern, 52 per cent. only are ileo-cæcal and ileo-colic, 30 per cent. are enteric, and 18 per cent. rectal and colico-rectal.

It will be remembered that intussusception is almost, if not quite, as frequent a cause of obstruction as strangulation, under which the percentages were given.

Etiology.—As to distribution, two-thirds are found in males and one-third in females. It is especially an accident of the young, occurring in 34 per cent. under one year and 56 per cent. under ten. Diarrhœa and habitual constipation are probable exciting causes, having preceded in 13 and 12 cases respectively out of 51. Other possible causes are so infrequent as to be unworthy of mention. Experiments with faradism would seem to show, however, that spasm plays a more important rôle than relaxation.

Intussusception of the dying should be mentioned, in passing, as a form of intussusception which often takes place a short time before death, more frequently in children, and is probably caused by certain irregular peristaltic movements toward the end of life. It produces no symptoms during life.

III. Twists and Knots. Volvulus.

The majority of cases are axial twists, *i. e.*, the bowel is twisted on its mesenteric axis, this being the case in 40 out of Fitz's 42 cases, two only being knots. Eighty-seven per cent. of cases occur in the large intestine, one-half in the neighborhood of the sigmoid flexure, and nearly one-third in the ileo-cæcal and cæcal region, the remainder in the small intestine.

It is more frequent in males in the proportion of two to one. More cases occur between the ages of thirty and forty.

IV. Obstruction by Abnormal Contents or Foreign Bodies.

The majority of these are gall-stones, 23 out of 44; 19 were fecal impactions, and two enteroliths. One of the latter was shellac, found in a man who had been in the habit of drinking alcoholic solution of shellac. Usually enteroliths are made up of triple phosphate of lime and magnesia about a nucleus which may be a mass of hair or other foreign body. Obstruction by gall-stones appears to be three times as common in females as in males. They enter the bowel usually by ulcerating through the gall bladder, commonly into the small intestine, more rarely into the colon. Fecal obstruction is also more frequent in females. The ages are pretty uniformly distributed from eight to eighty. In a few instances obstruction is caused by substances introduced by the mouth, but the objects thus introduced, as pennies, buttons, pins, and the like, are, as a rule, promptly recovered in the stools. A coil of lumbricoid worms has caused obstruction. So has the accumulation of certain medicines, such as magnesia and bismuth.

The seat of obstruction by gall-stones is most frequently the ileo-cæcal region; after this, lodgments are in the small intestine, with diminishing frequency as we ascend. Fecal obstruction occurs in the large

intestine, the lower part more frequently than the upper. The fecal tumors found in appendicitis are now regarded as the result of the inflamed appendix rather than the cause of the cæcal inflammation.

V. Strictures and Morbid Growths.

A comparatively small number of obstructions occur from these causes. They are always found in adults, four-fifths after the age of forty, and are apparently twice as common in women as in men. By far the largest number is met in the large intestine and lower abdomen, the majority being in the left iliac fossa. Strictures may be

(1) Congenital, illustrated by the imperforate anus or defective union between the pylorus and duodenum.

(2) Cicatricial, from healed ulcers; or

(3) Due to malignant and benign morbid growths.

Of malignant growths, the most frequent is the cylinder-celled epithelioma, which may form a ring in the vicinity of the sigmoid flexure, where colloid cancer is also met with. Any of the varieties of benign tumors may produce obstruction, while inflammatory processes external to the bowel, especially in the pelvis, may cause obstruction by pressure from without.

VI. Fecal Obstruction.

Synonym.—*Ileus paralyticus vel nervosus.*

This has its origin primarily in an insufficiency of the forces that move the contents of the bowel onward, resulting ultimately in an absolute paralysis of a segment of the bowel, arrest of motion of contents, and finally obstruction. The plug of fecal matter grows harder and larger, and compresses and stenoses the adjacent bowel, resisting any further onward movement, and increasing the impediment to the restoration of a natural condition, culminating, finally, in paralysis and stretching of the muscular fibres. The so-called "stercoral ulcer" of the cæcum, on which the older writers laid much stress, and which was ascribed partly to gangrene, due to pressure, and partly to the irritating effect of impacted fecal matter, is to-day regarded as extremely rare.

Ileus paralyticus may affect both the small and large intestines, but is more common in the latter, especially in the cæcum, where the pressure is concentrated from above and below. A local peritonitis may also be developed about the paralyzed and distended intestine. Mention is made under chronic constipation of the enormous masses of fecal matter thus accumulated. The wall of the intestine above the accumulation may also be hypertrophied because of the propulsive efforts of the muscular coat.

The causes of fecal obstruction are various and similar to those of the constipation in which it begins. It is more frequent in females and adults, especially the aged. The tendency to it is sometimes congenital, and it is favored by sedentary habits. Nervous influence is not to be ignored; the tendency to constipation is seen in the chronic insane, in the hysterical and hypochondriacal, and in affections of the spinal cord.

Chronic enteritis and chronic peritonitis favor it; so may anatomical peculiarities of the colon.

Symptoms of Obstruction.—(*a*) *Of acute obstruction.* As most of the important symptoms are common to the different causes of obstruction of the acute type, I will first consider them from the general standpoint, emphasizing any special relation which a given symptom may bear to a special cause.

First is *abdominal pain.* This is the most constant of all symptoms, being present in a decided majority of cases of obstruction from whatever cause. The pain is one of the earliest symptoms in every form of acute obstruction. It is usually sudden and very severe, and may be intermittent, or constant with exacerbations. It may occur in any part of the abdomen, regardless of cause, though most frequent in the neighborhood of the umbilicus, so that its location has no diagnostic value.

Nausea and *vomiting* are almost as frequent. The vomitus at the onset consists of the food last taken, but soon becomes bilious, yellow, and finally fecal with varying frequency. It is relatively infrequent in strangulation, while it is relatively frequent in volvulus, stricture, and tumor. The vomitus is especially apt to become fecal when caused by strangulation—usually from the third to the fifth day.

Tympany is next in frequency. It is a symptom of later occurrence than pain and vomiting, presenting itself usually from the second to the sixth day. It varies greatly in degree, increasing as a rule with the duration of the obstruction and being sometimes enormous. It is of least importance in obstruction by intussusception, most marked in volvulus. It is sometimes, but not always, accompanied by tenderness.

Tenesmus is a frequent symptom where there is obstruction in the large bowel, as in 15 per cent. of cases of volvulus and 55 per cent. of acute intussusception. It also occurs in stricture of the large bowel.

Tumor, under which are included circumscribed visible intestinal coils as well as swelling characterized by absolute dulness, is a rare symptom except in intussusception, where it is characteristic, having been present in 69 per cent. of Fitz's cases, more particularly when in the large intestine, where it is also sometimes associated with a relaxed sphincter. It occurs sometimes in obstruction by foreign bodies. In strictures and tumors it may be found by rectal examination. Tumors with dulness are not seen in twist, though visible coils are sometimes present.

Fever is even less frequent than tumor—in fact, its absence is rather characteristic. Records of elevated temperature are, however, found in 22 per cent. to 28 per cent. of all cases, the maximum record being 102° F. (38.9° C.).

(*b*) *Symptoms of Chronic Obstruction.*—Chronic obstruction is commonly due to fecal impaction, more rarely to stricture, cancerous disease, or foreign bodies. *In fecal impaction*, what appears to be simple constipation at first is succeeded by permanent retention, which may last for weeks without causing inconvenience. Examination per rectum will often find this tube filled with hard fecal matter which may be cleaned out with the finger or a spoon-handle. There may even be *diarrhœa*, due to irritation of the bowel above the impaction, when the catarrhal

secretion will channel out the mass and carry a portion with it. Gradually, however, the impaction becomes impregnable to all remedies, natural and artificial, the abdomen *swells*, there is *fulness* and *weight* within, *pain* in the genitals or thigh from pressure on the sacro-lumbar nerves; the *appetite* fails, the *tongue* is coated, and *breath offensive;* sometimes a condition of *lethargy* and *indifference* supervenes along with *great weakness* and the patient dies of exhaustion. At any time, on the other hand, may follow with suddenness the train of symptoms already described—pain, tympany, nausea, and vomiting, ultimately of fecal matter, with collapse and death.

In all cases, sooner or later, a *fecal tumor* presents itself—a tumor formed by the mass of retained feces, chiefly in the right iliac fossa, the region of the cæcum, corresponding to the outer half of Poupart's ligament. It is sometimes hard, at others soft and yielding, and sometimes tender and painful, probably because of a little local peritonitis. In the ascending colon the tumor is soft and in the hepatic flexure it may give rise to the notion of an enlarged liver. It may move in the more loosely attached parts of the colon and may drag the transverse colon down toward the pubis. In the descending colon and sigmoid flexure it is usually harder and may be subdivided into scybala. It is, of course, easier of detection in persons with thin abdominal walls and may be obscured by flatulent distention. When recognized it is of great diagnostic value. Such tumors have been taken for tumors of the stomach, liver, spleen, and kidneys, and for pregnancy.

In fecal tumors alone, of all the causes of obstruction considered, is the prognosis favorable if the condition is recognized sufficiently early, while a considerable latitude of duration may also be allowed.

Hiccough is an occasional symptom, and appears to be more frequent in volvulus. *Jaundice* is often found in obstruction by gall-stones.

The *urine* has been irregularly studied in acute obstruction. It is not infrequently spoken of as scanty and containing an increased amount of indican, especially in obstruction in the small intestine, not, it is said, of the large. Albumin is rarely present. It is to be remembered that peritonitis causes an increased indican reaction.

Tumultuous peristalsis is not infrequent above the seat of obstruction.

Bloody stools and *tenesmus* are symptoms which especially characterize intussusception and are important in the diagnosis, the former occurring in three-fifths of cases, the latter in one-third, or as often as tympany. It may occur early or late. Blood-stained stools also occur in connection with cancer of the lower bowel.

Local peritonitis is a symptom especially of volvulus and obstruction by gall-stones, caused in the latter case rather by the destructive results incident to the passage of the stone into the bowel from the common duct.

Collapse is the terminal symptom in fatal cases, due to the profound impression on the nervous system, and presents the lowered temperature, leaking skin, and feeble pulse characteristic of collapse from other causes. Cases are reported in which operation during collapse was followed by recovery.

The same train of symptoms may succeed stricture and tumors, to

be followed at times by partial relief, which is again succeeded by similar symptoms leading to ultimate total obstruction and death. Such symptoms will, of course, be associated with the anæmic dyscrasia and emaciation which belong to the causing diseases, and which more frequently lead to death without obstruction than with it.

Diagnosis.—The importance of early and correct diagnosis is intensified at the present day by the fact that operative interference promises by far the best results, while to be effectual it must be early. The diagnosis has three principal objects—first, the existence of *obstruction per se;* second, its *seat,* and third, its *cause.* The first is by far the most important, as operation is indicated in one variety or situation almost as much as another.

First, as to the *presence of obstruction,* it has happened that a case of intense enteritis has presented all the symptoms of obstruction. Should this be the case, I do not see how a mistake can be averted. Fortunately, it must be very rare, while I can scarcely conceive a case of enteritis of such severity without decided fever and a temperature of at least 102° F. (38.9° C.), rarely obtained in obstruction.

Acute poisoning, associated with vomiting, such as is caused by poisonous mushrooms, biliary, renal, and intestinal colic, the pain caused by twisting of the ureter in a movable kidney, all present symptoms more or less like those of obstruction; but the combination of signs necessary to the picture of obstruction is still wanting.

Much more common is the mistaking of appendicitis for obstruction in which there are pain, vomiting, constipation, as well as tumor in the neighborhood of the cæcum, but the differentiation between these two conditions was considered when treating of acute appendicitis. Peritonitis itself presents symptoms common to it and obstruction, including abdominal pain, distention, constipation, and collapse, with increase of indican. But the presence of fever, the absence of tumor and of fecal vomiting, point to peritonitis.

The symptoms of hernia are also those of intestinal obstruction, and in all cases careful search should be made for a concealed hernia. Such herniæ have been found in the external ring and in the obturator foramen at autopsy by William Osler, who has also met a case of acute hemorrhagic pancreatitis presenting the symptoms of acute obstruction.

The *seat of obstruction* is more difficult to determine. Unfortunately, the situation of the pain gives little information, since it is almost always in the vicinity of the navel, wherever the actual seat of obstruction may be. In other cases it is diffuse. Rarely it may be at the seat of obstruction. Though fecal vomiting is much more frequent in obstruction of the small intestine than in the large, it still occurs in one-eighth of all cases of the latter.

When a tumor is present it gives valuable information, being commonly at the seat of obstruction. Active peristalsis, limited to one part of the bowel, indicates that the obstruction is below it.

Having excluded hernia by a careful examination for a seat of strangulation, examination *per rectum* should be made, also *per vaginam.* By either method a tumor may sometimes be recognized. Especially is this true of a tumor caused by intussusception. A stricture may also be

detected by digital examination of the rectum, as may obstruction by foreign contents. On the other hand, the rectum may be totally empty of feces and continue so, whence it is probable that the obstruction is in the small intestine, or high up in the large. The position and size of the uterus and ovaries and of the prostate will also be ascertained by vaginal examination. The rectum can be more thoroughly explored by suitable specula in the knee-elbow position, but rectal exploration by the entire hand has not been followed by the results anticipated. The hard rectal tube has produced perforation, while the flexible tube so coils itself up as to be valueless in diagnosis.

Sometimes, if the enlarged intestine is filled with hard fecal matter, it can be felt as an uneven mass in the course of the bowel. Moderate distention in the upper part of the abdomen, with flatness below and in the sides, with rapid collapse and oliguria, points to obstruction in the duodenum and jejunum. Such distention is temporarily diminished by vomiting, but is uninfluenced by fecal discharges if they be secured by enemata. Nor is the vomiting fecal in duodenal and jejunal obstruction; when the obstruction is in the ileum and cæcum the distention is more central, the region of the colon being flatter until covered in by the extending tympany, and the vomiting is more apt to be fecal.

When obstruction is seated in the colon, tympanitic distention is greatest, yet the difference between it and that of obstruction in the ileum is not so great as to possess much diagnostic value. If in the lower colon, there may be tenesmus and discharge of blood and mucus, and there is increase of indican in the urine. Measuring the capacity of the large bowel by air, gas, or water is strongly recommended as an aid to diagnosis. Reliable observation goes to show that none of these substances can be made to pass the ileo-cæcal valve. Water recommends itself so far above the others that it alone will be considered, though Frederick Treves says, in his admirable essay, that it is "absolutely worthless." The capacity of the large intestine of adults is about six quarts, or about as many liters. That of infants has been less exactly determined, but the limits of safety have been more accurately defined by the degree of pressure that can be applied without danger. These are, with absolute safety, ten feet in an infant and 20 in an adult, greater distances having been reached without harmful results. If the method is employed at all for diagnosis, it should be early, before the nutrition of the bowel has suffered, since rupture into the pelvis has taken place under light pressure. The patient, etherized, should be placed on his right side or inverted, and precaution taken to keep the fluid from returning. Close pressure of the buttocks generally suffices. The fluid is most conveniently introduced by the fountain syringe, by which pressure can be varied. Various objections, more or less valid, are made to the value of this method, such as resistance by voluntary muscles during life, valve-like obstruction, which permits ascent of fluid, but not descent, and unequal dilatation. However, the method should not be overlooked, and it is more than likely that much may be learned from future experience. Thus, since a case of obstruction at the sigmoid flexure, cited by Treves, permitted the injection of three pints of fluid (1.4 liter), it is evident that only in the event of the injection of a larger amount can the

gut be considered open at this point. Treves claims, too, that the arrival of fluid in the cæcum may be recognized by auscultation. Under favorable circumstances, as, for example, with an empty colon, a trained ear, and skilful technique, this seems quite possible.

The presence of obstruction being recognized, the *cause of the obstruction* may often be determined with some degree of probability. First to be considered is the relative frequency of the different causes. In the first place, adopting Fitz's figures, strangulation and intussusception together take up 70 per cent. of all cases, the two being nearly equal in frequency. After that comes volvulus with 15 per cent., gall-stone with eight per cent., and stricture or tumor six per cent., that is, the twists about equal obstruction from gall-stones and tumor and stricture together.

Again, if the obstruction be found in the large intestine, it is more likely to be intussusception, twist, or stricture and tumor, since of the obstructions in the large bowel, 51 per cent. are intussusception, 30 per cent. twists, and 12 per cent. stricture and tumor. If in the small intestine, it is most likely strangulation or gall-stone obstruction, since 72 per cent. of obstructions in the small intestine are strangulations and 14 per cent. gall-stones, leaving eight per cent. only for intussusception, five per cent. for twists, and one per cent. for stricture and tumor. If the attack has been preceded by one of jaundice or other liver symptoms, as hepatic colic, it is almost certain to be gall-stone, especially if the patient be over fifty years old.

Again, if the patient is under thirty, particularly if a child, it is more likely to be intussusception than twist, while if there are palpable abdominal tumor, bloody stools, and rectal tenesmus, the case is almost sure to be intussusception, rendered still more likely if the rectum has a large capacity for water, since the intussusception is found near the cæcum in 75 per cent., while twist is found near the sigmoid flexure in 50 per cent. Of all forms, intussusception presents the clearest clinical picture and is most easily recognized.

Twist, cancer, and stricture are more apt to be below the sigmoid, and the two last sometimes may be felt by the finger. In point of fact, twist in the large bowel is not often recognized. It is a disease of the adult, rarely under forty; vomiting is less early and less severe than in strangulation. Some extent of local peritonitis almost invariably results, causing rigidity of the abdomen, while meteorism appears early and is extreme, the distended intestine often displacing the solid viscera.

If there is a history of previous peritonitis, strangulation becomes more likely, since such inflammation precedes in 68 per cent., while there is also a history of previous attacks in 12 per cent. The pain in strangulation is early, sudden, and severe, and the same may be said of vomiting. It becomes stercoraceous in 60 per cent., and affords no relief. There is little or no distention unless peritonitis supervene. There is great prostration, no tenesmus or discharge of blood. The average duration is about five days. The presence of diaphragmatic hernia as a cause of internal strangulation must not be overlooked; it is almost always the result of severe injuries. The half of the thorax containing the viscera is distended and tympanitic on percussion, while breathing movement is restricted, the breath sounds feeble, the vocal fremitus and vocal

resonance diminished or absent—signs shared with pneumothorax. The pitch and intensity of the percussion note vary also with the degree of distention and the position of the viscera invading the thorax, while there may be metallic tinkling of fluid in the intestine, due to peristalsis.

Fecal obstruction is recognized by the symptoms already described under chronic obstruction, and such recognition is not very difficult, especially if the fecal tumor is found. Sometimes, however, on account of its insidiousness, fecal obstruction is overlooked when presenting only the more chronic symptoms, and the patient dies of supposedly unknown cause when accurate and careful study would have led to its discovery.

Treatment.—So soon as the diagnosis of intestinal obstruction, by any of the causes except constipation, is made, operative treatment should be considered. Shall other treatment be attempted? It is likely that, in the course of diagnosis, certain measures have been carried out which are themselves of the nature of treatment, such as distending the rectum with water, or its inflation by air after the manner described under general diagnosis of stomach and intestinal disease. This has been found effectual in 33 out of 44 cases of suspected or probable intussusception collected by Fitz, and therefore merits a trial. Irrigation with water is decidedly preferable. A long nozzle should be used, in order to carry the liquid (warm water is the best) well up into the bowel, where it should be retained some time. The lateral or knee-elbow position should be assumed. What else may be done early and without risk to the patient? Above all, give no aperients—I am speaking of acute intussusception. To relieve the excessive vomiting, the stomach may be washed out as suggested by Kussmaul, and described on page 249. This is at once a harmless measure and may be efficient for the purpose intended. It may be done three or four times a day. Opium may be administered hypodermically to allay the intense pain, and may also relieve the vomiting. Or it may be given with a view to cure, which there is reason to believe it has accomplished in cases of intussusception and even strangulation. Opium may, on the other hand, be harmful, by obscuring diagnosis and producing an appearance of relief, while the local condition of the bowel is really growing worse.

The nourishment of the cases demands careful thought. It is irrational to continue the administration of nutriment by the mouth when it is as rapidly rejected. Under these circumstances the rectum should be the only route employed, while ice should be freely administered by the mouth. On the other hand, where the obstruction is in the colon, where tenesmus and diarrhœa are symptoms, and where vomiting is a less prominent symptom, small amounts of liquid nourishment may be introduced by the mouth.

At the present day, however, where abdominal section is made with such impunity, there should not be much delay before it is done, especially in view of the fact that the operation is so much better borne early in the disease, before the strength of the patient is exhausted. Rarely should more than three days be allowed to elapse without operative interference in cases of acute obstruction from any cause except fecal impaction.

The situation is altered where a diagnosis of fecal tumor has been

correctly made. Here nothing is so efficient as repeated large injections of warm water, high up and retained ten to fifteen minutes if the patient can retain them, as he should be encouraged to do. Good results are sometimes obtained from the coincident use of small doses of calomel, 1/8 to 1/5 grain (0.008 to 0.013 gm.), given hourly. In most cases the finger or some mechanical appliance, as a spoon-handle, can be used to loosen the fecal impaction if it is low enough down in the rectum.

It is in this form, too, that electricity, massage, and metallic mercury, as used by the older practitioners, are sometimes useful. Electricity is variously used, but the most efficient application is the recto-abdominal, in which one electrode of a faradic machine is placed in the rectum, the other over the abdomen. The use of metallic mercury has been revived by M. Matignon in fecal impaction with apparently good results, the dose being 1.5 to nine ounces (50 to 280 gm.). It probably acts by insinuating itself in a state of minute subdivision through the fecal tumor and between it and the bowel, loosening and breaking up the mass so as to restore the natural passage. It is of no use where there is strangulation, intussusception, or volvulus.

In stenosis of the gut, the treatment recommended for fecal impaction may be used oftentimes with the effect of removing the obstruction so far as due to the delay of fecal matter at the narrowed point. Sooner or later, operative interference becomes necessary.

CONSTIPATION.

Synonym.—*Costiveness.*

Definition.—Unnatural retardation or delay in the natural evacuation of the bowels. Though there may be some exceptions, an evacuation of the bowels once in twenty-four hours seems to be nature's law in the case of the adult human being, and any prolongation of the natural interval may be said to constitute costiveness. A popular application of the term is, however, also to a condition in which, though there may not be infrequency of stools, the dejecta are dryer and harder than natural and are discharged with more or less difficulty and pain. The physician should appreciate this, otherwise misunderstanding may arise as to the exact meaning of the patient. Constipation is also something different from retention due to obstruction by various causes. The interval between bowel movements in constipation varies greatly, ranging between a couple of days and weeks. Many constipated persons have no dejections unless aperient medicine is taken.

Morbid Anatomy.—There are no morbid changes characteristic of constipation. Dilatation of the colon in various degrees is present, sometimes enormous, as shown in Fig. 19, and there may be found the remnants of inflammatory or other local lesions which may be responsible for the obstruction. The large accumulations of fecal matter found in these cases are known as *coprostasis.*

Etiology.—The immediate causes of this form of constipation are:—

(1) Atony of the colon, whence results a slowed peristalsis.

(2) A deficiency of the natural stimuli to peristalsis afforded by various secretions, especially the bile.

(3) Perhaps the most common cause of atony is a habit, engendered through indifference or necessity, of disregarding nature's call for relief. Repeated disregard of such call results, sooner or later, in disappearance of inclination. Sedentary habits coöperate to produce such disinclination. Atony may, however, also be the result of disease of the bowel and of general disease causing debility, such as anæmia, chlorosis, and protracted illness, like fever.

(4) A loss of muscular power in the abdominal walls from over-distention or obesity.

(5) Improper food. The foods which most stimulate peristalsis are vegetables, especially those with an insoluble residue, such as is afforded by the outer coatings of grain. Foods of an opposite kind are represented by milk and the farinacea.

(6) Finally, stricture and displaced organs, such as the uterus, tumors, and foreign bodies impinging on the bowel and delaying the descent of the feces, become causes.

Treatment.—Every case of constipation should be carefully studied with a view to determining its cause, and if such cause is found it should, of course, be removed when possible. If such cause is not found, the first injunction in the management of constipation is the observance of regularity in going to stool at a fixed hour of the day, whether inclination prompts or not. The usual hour for this purpose is immediately after breakfast, though it matters not much when it is, so it is regularly observed. Especially harmful is it to disregard any inclination which may appear at this time, or, indeed, at any time. Next is the use of food of the kind referred to under the head of etiology, such as fresh green vegetables of all kinds, with succulent fruits. Of breads, the so-called "brown" or bran bread, or, still better, gluten bread, is to be preferred. With such food should be conjoined massage of the abdomen or compression, either by the patient himself or another. A very excellent daily practice is to flex the body a number of times while in the standing position. This has the effect of compressing the bowels and stimulating peristalsis, and is one of the most useful aids. It should be practised once or twice a day; if once, in the morning on rising; if twice, at bedtime also. The free use of plain water is sometimes sufficient to overcome the milder cases. Thus, a glass of water may be taken before breakfast and another at bedtime.

Last of all should medicaments be employed. Unfortunately, these are often necessary. The simplest and least irritating should be employed. A simple tonic pill composed of $\frac{1}{3}$ grain (0.022 gm.) of the extract of nux vomica and $\frac{1}{12}$ to $\frac{1}{8}$ grain (0.005 to 0.008 gm.) of the extract of belladonna, three times a day, and kept up for some time in connection with the dietetic measures alluded to, is often sufficient.

But of actual aperients, the natural mineral waters are deserving favorites, especially Friedrichshalle and Hunyadi, and where less active waters are required, the American Saratoga waters. These are saline waters which present quite a range of proportion in their constituents at the various springs. The waters of the Bedford Springs, of Bedford, Pa.,

are also very efficient, stimulating, as does the Saratoga water also, the secretion of bile. The doses of all of these waters vary so much with circumstances that it is impossible to lay them down with definiteness. The minimum dose of the foreign aperient waters mentioned is two fluid ounces (60 c. c.) and increased to eight fluid ounces (240 c. c.). Less than the latter quantity of the American waters is seldom used at a dose.

Of drugs, cascara sagrada has become deservedly popular. The best preparation is the fluid extract, as its dose can be readily regulated. From ten to 30 minims (0.6 to 2 c. c.) may be given after the evening meal, and, if this should prove insufficient, the same dose after the mid-day meal is to be preferred before increasing the evening dose. The solid extract is, however, also efficient, and a grain or two (0.066 to 0.13 gm.) more may be added to the laxative pill already mentioned, or, if a more active aperient is desired, as many grains of extract of colocynth may be substituted.

An old favorite, a pill composed of extracts of aloes, nux vomica, and belladonna, in varying proportions, to be taken at bedtime, has been largely substituted of late by another made by the manufacturers and pharmacists, of aloin $\frac{1}{5}$ grain (0.013 gm.), strychnine $\frac{1}{60}$ grain (0.0011 gm.), and belladonna $\frac{1}{8}$ grain (0.008 gm.), of which one or two are a dose. To such a pill podophyllin in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.0165 to 0.033 gm.) may be added with advantage, or blue mass in doses of $\frac{1}{2}$ grain to two grains (0.033 to 0.132 gm.), or rhubarb one to two grains (0.066 to 0.013 gm.). The belladonna may be substituted by the extract of hyoscyamus, of which one to two grains (0.066 to 0.132 gm.) may be given. The compound licorice powder is a favorite aperient with some, but is bulky. The dose is a drachm (3.8 gm.) or more.

A glycerine suppository or $\frac{1}{2}$ drachm (2 c. c.) of glycerine injected has become a favorite means of securing an evacuation. It should be remembered as a possible remedy, but it acts by irritating the lower bowel and soon loses its effect. The enema of plain water, one to two pints (500 to 1000 c. c.), though less convenient, is to be preferred, and some persons use it regularly. None of these measures are curative. They simply empty the bowel at the time, and systematic effort should be made to reduce them gradually, while the hygienic treatment is kept up.

It sometimes happens that an impacted fecal mass becomes channelled and fecal matter may descend from above through it, and thus lead to the belief that normal passages are being secured. The physician should not be slow to explore the rectum with the finger, and by means of it, or the handle of a spoon, clear out the mass. This is often absolutely necessary before an evacuation can be secured.

Treatment of the Constipation of Infants.—I prefer to overcome this, when possible, by simple small enemata, repeated until an effect is produced, and carried out at a fixed hour each day, preferably in the evening. The child is best held on the lap of the mother, who should be properly protected, and a small quantity, say two ounces (60 c. c.), of tepid water is thrown into the rectum of the infant. If it returns unchanged, after a few minutes' delay another syringeful is thrown in, and if necessary another. Ultimately a fecal discharge is usually thus obtained. I lay much stress on the regularity of this performance also.

FIG. 19.—GIANT CONGENITAL DILATATION OF HUMAN COLON.

The more distended end is the sigmoid flexure. The narrow part taking exit from it is part of the rectum, which was normal. The narrow distal end of the preparation represents the head of the colon with the string attached to a fragment of the small intestines. The arched part of the specimen represents the transverse portion of the colon. The figure within represents a normal human colon photographed simultaneously for comparison of dimensions. Both were dried preparations.

It may be necessary to add a little soap to the hot water. Sometimes a little titillation of the anus by twisted pieces of paper answers every purpose. At the same time the belly of the child should be massaged by the mother. Small suppositories of soap or of glycerine may be used if the measures mentioned are inefficient. For simple constipation in infants it is preferable to administer nothing by the mouth, if it can be dispensed with.

Dilatation of the Colon.

This is one of the consequences of chronic constipation, though it may also occur as an acute condition, the result of sudden obstruction, as by a twist in the meso-colon. It may involve the whole colon, but the vicinity of the sigmoid flexure is its usual seat. In other cases the dilatation may precede or may even be congenital, when it becomes the direct cause of chronic constipation or coprastasis, which in turn increases the dilatation. Such is a remarkable specimen in the museum of the University of Pennsylvania, secured by the late Henry F. Formad * in the course of his work as coroner's physician, figured on p. 321. Two and a half pailfuls of fæces, weighing 40 pounds (20 kilograms), were removed at autopsy.

Symptoms.—They are the same as those of obstinate constipation, extending over weeks, in addition to enormous *distention* and *tympany* of the abdomen. Physical examination in extreme cases recognizes dislocation of the adjacent abdominal and thoracic viscera, especially the liver, spleen, heart, and lungs.

Treatment.—The treatment is that of the resulting constipation, which, in cases of this kind, is by enemata carried high up into the bowel, together with remedies which stimulate secretion into the upper bowel, of which calomel is one of the best. It should be given in doses of not less than a quarter of a grain (0.016 gm.) hourly until an effect is produced in association with that of the enemata. This condition probably results, at times, from the gradual accumulation of fecal matter while frequent small discharges are being obtained which do not clear out the bowel. Hence the rectum should unhesitatingly be explored by the finger in doubtful cases. Complete evacuation of the bowels is sometimes extremely difficult, but if the exact state of affairs is appreciated, perseverance will ultimately conquer.

NERVOUS AFFECTIONS OF THE BOWEL.

The bowel, like the stomach, is subject to deranged nervous influence, which manifests itself in

(1) Increased or diminished contractility of the muscular coat.
(2) Increased or diminished sensibility of the bowel.
(3) Increased or diminished secretory function.

I. Derangements of Motion.—These are manifested by diarrhœa, by constipation, and by cramp.

* Transactions of the Pathological Society of Philadelphia, Vol. xvi, 1891–1893, p. 23. Dr. Formad gives in his paper a summary of other cases reported.

(1) *Nervous diarrhœa* occurs in adults and children, the result of increased peristalsis due to pure nervous influence. It implies a hyper-excitability of the nerves regulating peristalsis, causing them to respond to stimuli to which they are otherwise indifferent, including the simple mechanical and chemical irritation of the natural intestinal contents. Hence we find nervous diarrhœa in nervous, hysterical, and neurasthenic persons. In these persons, too, psychical influences, such as fright, depression, and even joy, cause diarrhœa. The attacks of diarrhœa which occur in tabetic persons have a similar origin through central nervous influence. Still more does such an excitability of the nervous system respond to unnatural irritation, such as that of teething in infants, producing diarrhœa, which may be quite independent of irritating food, though the latter may coöperate.

There is no morbid change, and the bowel movements are generally watery and without blood or mucus. They vary greatly in frequency, from two to 20 or more daily, occur suddenly, and disappear often as suddenly as they come. They may last for several days.

(2) Constipation, the result of altered nervous influence, is even more common than diarrhœa.

In this condition the nerves have lost their susceptibility to impressions ordinarily sufficient to excite the automatic actions which result in bowel movements, and peristalsis is at a standstill. Associated with such condition is often an atony of the muscular coat, which permits gaseous over-distention and tympany. Thus is explained the constipation not only of the hysterical and neurasthenic, but also that which attends the various forms of nervous disease, especially of the spinal cord. The constipation associated with the passive congestion of heart and liver disease is the result of a similar lethargy of the nerves distributed to the muscular coat of the bowel. Here, too, we may infer an exhaustion of nervous excitability by over-stimulation, if, as is supposed, normal peristalsis is excited by the stimulus of the carbonic acid of the venous blood, as well as by the food present in the intestinal tube. On the other hand, a food may be too bland and unirritating to excite the normal peristalsis. Hence it is that constipation attends the use of milk and the farinacea.

The effect of paralysis of the voluntary muscles controlling the external sphincter ani results in inability to retain the fecal contents, whence involuntary evacuations take place, a frequent symptom in disease of the brain. Under such circumstances, the control of the bowels is given over altogether to the reflex nervous centre in the spinal cord, and man is reduced to the condition of the infant and the lower animals, in which defecation is a purely reflex act. Over-sensitiveness or over-stimulation of the sphincter would result in a spasmodic and painful contraction, which is, however, a very rare and anomalous condition.

(3) Nervous cramp, or excessive contraction of the intestinal muscles, is so intimately associated with pain that it will be considered in connection with deranged sensibility.

II. Derangements of Sensibility.—(1) *Enteralgia.* Sensory neuroses of the bowel are mostly in the direction of increased irritation of

sensory nerves derived from the splanchnics, which contain the sensory as well as the inhibitory and vaso-motor nerves to the bowel. Such irritation implies increased irritability of these nerves or the presence of unusual irritants. The pain thus induced, unassociated with organic lesion, is known as enteralgia or neuralgia of the bowels.

Associated with exaggerated contraction of the muscular coat, it is known as *colic*, though the terms enteralgia and colic are also interchangeably used. Characteristic of enteralgia are its suddenness of occurrence and to a less degree the suddenness of its cessation. It is often associated with crampy contraction of the abdominal walls, when the pain is augmented by pain thus produced.

Among the causes which excite pain are such foreign bodies as indigestible articles of food, intestinal worms, fecal masses, over-distention with gases, and the like. The effect of the latter is attested by the relief which attends the discharge of gas. The operation of reflex causes must also be admitted. Such may be the mode of action of cold, and of lead intoxication, which produces the well-known lead colic. Such is, possibly, gouty enteralgia, or the enteralgia succeeding an attack of gout. The enteralgia associated with certain nervous diseases, such as in the painful enteric crises of locomotor ataxia, is probably the direct result of alteration in the sensitive nerves themselves. It may be, too, that the action of lead in producing lead colic is upon the nerves themselves. The hysterical and hypochondriacal and the anæmic are subject to colic through increased sensitiveness of nerves.

The pain existing in enteralgia is diffuse and throughout the abdomen. Enteralgia is to be distinguished from enteric pain due to organic disease. Most important are the inflammatory and ulcerative states associated with enteritis, typhoid fever, peritonitis, appendicitis, and intestinal obstruction. The diagnosis is not usually difficult. There is, first of all, the absence of fever; second, the history of the ingestion of irritating foods, or some one of the causes named. Very important is the point that colicky pain is relieved by pressure, while pressure increases pain in all of the affections named. Most difficult becomes the diagnosis in those cases in which there is coincident flatulence, as is sometimes the case in typhoid fever, when the question to be answered is, Is this colic, or is it the tenderness which comes of peritonitis? Undoubtedly, in some instances, both are present, and one condition reacts upon the other.

The diagnosis from appendicitis has been considered in treating of the latter. Intestinal obstruction offers further similarity in the absence of fever, but the severity of the symptoms in obstruction is on the whole greater, their duration is longer, while constipation and vomiting are superadded. Rheumatism of the abdominal muscles sometimes very closely resembles enteralgia. In this, however, there is commonly exquisite tenderness, while the pain is superficial and more continuous. Nervous dermalgia or hyperæsthesia of the abdominal wall has similar features and is common in hysterical women. Sometimes this is associated with hysterical colic, but even here, while the skin itself is sensitive, deep-seated pressure does not bring out increased pain. Lumbo-abdominal neuralgia should be mentioned. It is, however, more lateral and toward the back.

Biliary colic and nephritic colic also resemble enteralgia, and may

at first be mistaken for it, but careful examination should soon discover the points peculiar to each, such as localized tenderness and jaundice in the former and the course of the pain into the groin and testicle and thigh in the latter. Uterine colic may also be confounded, but the pain is distinctly localized in the region of the uterus and is apt to be associated with menstruation or to precede it.

It is important also, if possible, to discover the form of enteralgia or its special cause, whether due to indigestion, to reflex causes, to constipation, to lead, to hysteria, or to central nervous causes. This is to be arrived at by close attention to the history and associated symptoms.

(2) *Neuralgia of the Rectum.*—Some special symptoms characterize the sensory neurosis of the rectum which demand separate allusion. The nerves of the hemorrhoidal plexus are those concerned. An uncomfortable aching sensation in the lower bowel and lower abdomen, extending at times to the sacrum, perineum, and genitalia, is the principal symptom. With this is associated an irresistible desire to go to stool, which is, however, fruitless.

As a reflex sensory neurosis of the rectal nerves may be considered a peculiar sensation of exhaustion and disposition to faint after a movement of the bowels complained of by some persons. Wilhelm v. Leube also calls attention to an "intestinal vertigo" excited during the passage of fæces through the anus, and capable of being excited, too, by introducing the finger into the rectum.

The sensory neuroses of the rectum are more common in nervous women and in the subjects of hemorrhoids, while tabetic patients are apt to suffer from the same symptoms.

(3) *Diminished Sensibility.*—This is manifested for the most part only in delayed peristalsis. It has already been said that constipation is one of its most constant results. In the case of the rectum, it is well known how, in health, we are informed of a desire to go to stool. Paralysis of these nerves results in anæsthesia, which is followed by the absence of this desire. The effect must be an accumulation of fæces in the rectum, which may still be evacuated if volition and the motor nerve route are intact, but which demands artificial removal if these are in abeyance. It is a constant symptom in those affections of the spinal cord associated with paralysis. To higher degrees is added the loss of the limited reflex control, and if there be also loss of the voluntary control over the sphincter ani, an involuntary stillicidium of liquid contents of the bowel results, though the solids go on accumulating unless artificially removed.

III. Secretion Neuroses.—It is difficult to separate the consideration of the *secretion* neuroses of the bowel from that of the sensory and motor neuroses. Yet it is well known that secretion into the bowel may be influenced quite independently of peristaltic motion, perhaps through the vaso-motor nerves. Thus, while it is more than likely that the saline aperients produce their effect in response to the physical laws of osmosis, the secretion into the bowel which follows the hypodermic injection of pilocarpin cannot be explained upon any other ground than that of vaso-motor nervous influence. Allusion has already been made to the responsi-

bility of the nervous system in producing the mucous discharges and casts referred to in discussing chronic enteritis.

Treatment of Neuroses of the Bowel.—This follows easily upon a correct diagnosis, which is indispensable. The primary point of attack is that of the nervous condition at fault. The removal of the causes of irritation should be coincident with measures directed to the relief of pain. If irritating ingesta are present, an emetic should be given. In children the gums should often be examined, and lanced when swollen and tender. If there is constipation, the bowels should be opened. If there is hysteria, a nervous sedative is indicated. In enteralgia the promptest means of relief is a hypodermic injection of morphine of ¼ grain (0.0165 gm.) to an adult. Less may suffice, but if the pain is extreme it is not worth while to temporize with smaller doses. On the other hand, it is not safe to give more at a single injection. Should this dose be ineffectual, associated with the local measures to be described, it may be repeated in half an hour. The combination of atropine, $\frac{1}{150}$ grain (0.00044 gm.), with the morphine will increase its efficiency.

Of local measures, massage is probably the most efficient for constipation and enteralgia. It has been mentioned that relief of the pain by pressure is characteristic of enteralgia. Especially happy results may, therefore, be expected from massage, an expectation that is realized in practice. Counter-irritation to the abdomen by mustard or turpentine stupes may be used as an adjuvant to treatment in lieu of massage.

In milder forms of enteralgia, or in combination with morphine, aromatics and carminatives have always had a justified reputation. Some of these have been mentioned in considering the treatment of cholera and cholera morbus. An especially elegant and efficient preparation is—

℞. Spiritus Ammon. Aromat.,
Tinct. Card. Comp.,
Spiritus Chloroformi,
Spiritus Vin. Gall., ... āā . . . . . ʒij (8 c. c.).

M. et S.—Teaspoonful every half hour or fifteen minutes, in cracked ice or hot water, until relieved.

Its efficiency is increased by adding to each dose a few drops of deodorized tincture of opium.

CARCINOMA OF THE BOWEL.

All parts of the bowel are subject to carcinoma, which occurs in growing frequency as the gut is descended. Thus, of all cases of bowel cancer, barely five per cent. are found in the small intestine, 15 per cent. in the cæcum and colon, while 80 per cent. are met in the rectum.

In the *upper bowel*, in the neighborhood of the orifice of the bile duct, we meet most frequently the cylinder-celled epithelioma.

In the *large intestine* there is

(1) Cylinder-celled epithelioma or adeno-carcinoma, the most common form of cancer in the cæcum and sigmoid flexure.

(2) Colloid cancer
(3) Scirrhous } in the rectum.
(4) Soft cancer

(5) Squamous epithelioma in the rectum just above the anus.

Sarcoma, including the melanotic variety, also occurs in the rectum.

Benign tumors of the bowel, which may present symptoms similar to those of malignant tumors or none at all, include mucous polyps and fibromata, more rarely lipoma, myoma, angioma, and lymphoma.

Symptoms.—There are no symptoms distinctive of cancer of the bowel. The most constant local effect is more or less obstruction of the bowel, and we have already seen in our study of obstruction how far it is contributed to by cancer. There are, however, other symptoms which, added to those of obstruction, aid in the diagnosis. Particularly is this true in the case of the rectum. The duodenum is almost the only part of the small bowel involved.

The symptoms of obstruction met with in cancer of the bowel, already considered in treating of obstruction, include, especially, *constipation*, *pain*, *tumor*, *anorexia*, *nausea*, and, more rarely, *vomiting*. The added symptoms are *cachexia* and *altered fecal discharges*, which may include pus, blood, and, in few instances, fragments of cancerous tissue.

Of the symptoms of obstruction named, *tumor* alone demands further consideration, being the most important of all the symptoms of cancer. In fact, without it, a certain diagnosis is scarcely possible. On the other hand, given a case of obstruction, the presence of tumor points more to cancer than to any other cause, except intussusception and fecal impaction. As contrasted with intussusception, it is of long duration and found in adults; as with impaction, it is tender and movable, usually harder and more irregular. While the tumor may give a dull note to light percussion, to a hard stroke it is tympanitic. It may pulsate also if it lie over one of the large blood-vessels. Fecal tumors never do this. The difficulty of distinguishing from a fecal tumor is increased when a fecal mass is added to the cancerous tumor, but some of it may be cleared up by the use of purgatives and injections.

Cachexia, added to other signs of chronic obstruction, points to cancer. Change in the shape of the formed fæces, especially a band-like flattening, is much spoken of. It may be produced by any cause which protrudes into the lumen of the large bowel, characterizes rather disease of the lower part, and, to be of value in diagnosis, it must be constant. The more or less constant presence of sanious pus, particularly of fetid character, is important evidence in favor of cancer.

Diagnosis.—The tumor of the pylorus is likewise movable, often quite as much as an intestinal tumor, and it is sometimes impossible to distinguish the two. With pyloric tumor are associated symptoms of obstruction and dilatation of the stomach. The presence of jaundice points to cancer of the duodenum, as does also the continued natural acidity of the gastric contents removed after a test meal, but neither of these symptoms is pathognomic of duodenal cancer. In cancer of the stomach dyspeptic symptoms are earlier and more serious. Cancer of the head of the pancreas also produces jaundice, but the tumor arising from it is fixed and immovable, and much more deep-seated than tumors of any portion of the bowel, being behind the pylorus and the transverse colon, between the left sternal border and parasternal line.

With the other abdominal tumors intestinal cancer is not likely to be confounded. The floating kidney is movable, but when sufficiently

so to be compared in this respect with a cancerous tumor, is more movable, and may be generally returned to its natural seat. The kidney shape may not infrequently be recognized. Compression of the kidney produces often a peculiar sickening pain, and the presence of nervous symptoms is especially characteristic of floating kidney, but there is no cachexia. A movable spleen is even less likely to be confounded for similar reasons. It is, moreover, less sensitive. A laced-off lobe of the liver, often quite movable, can generally be traced to its normal attachment.

An actual tumor of the kidney, being behind the peritoneum, pushes the bowel and the ascending or descending colon before it, and must attain considerable size before it shows itself to the usual examination from the front. It very rarely compresses the bowel so as to produce symptoms of obstruction. The same may be said of tumors of post-peritoneal lymphatic glands. An ovarian tumor is characterized by its deep-seated origin, its ascending development, and its relation to the uterus as determined by joint vaginal and abdominal examination.

A circumscribed peritoneal exudate might be mistaken for a cancer of the bowel, but the history of its development, its flat percussion note, and the presence of some temperature which characterizes it are wanting in cancer of the bowel.

Cancer of the bowel is not likely to be mistaken for appendicitis, the acuteness of symptoms marking the grave form of the latter, while the absence of serious constitutional and cachectic symptoms is characteristic of the more chronic form of appendicitis.

Chronic inflammatory thickening of the bowel may, however, be a serious stumbling block. Especially apt to occur about the sigmoid flexure, it produces also obstructive symptoms, and careful and prolonged study may be necessary to the making of a correct diagnosis. Cachexia remains absent in simple inflammatory stenosis for a longer time at least than cancer.

Diagnosis of the Part of Bowel Involved.—As to the part of the bowel involved, once assured that the tumor is of the bowel, some indication of its more exact location may be obtained by noting its position, which, if in the right upper abdominal region, suggests the duodenum; in the vicinity of the umbilicus, the transverse colon; in the right iliac fossa, the cæcum, and the left, the sigmoid flexure. It should be remembered, however, that serious dislocation of the tumor from its natural site may occur as the result of inflammatory adhesions formed while the tumor is temporarily in a position remote from its natural site. Commonly, too, a cancer of the sigmoid flexure gives no indication of its presence to abdominal examination. Distention of the bowel with water or gas and the application of the principles laid down from this standpoint when treating of obstruction may be availed of in settling this question (see p. 317). Allusion has been made to the presence of jaundice as characteristic of duodenal cancer; also to the retained natural acidity of the gastric contents removed after a test meal as compared with gastric cancer.

As to *the rectum*, it is subject to the same forms of cancer as the pylorus, much in the same order of frequency, the columnar-celled epithelioma being most frequent.

The early symptoms are those of *irritation*, including *pain*, *tenesmus*, the *discharge of mucus and blood*, and, probably, most cases of carcinoma of the rectum are mistaken at first for dysentery. In the cases of colloid cancer, the colloid material may be discharged from the bowel and reasonably mistaken for mucus. Fortunate is the clinician if it occurs to him to make an early examination of the rectum by the finger; for generally the disease can be felt, either as an ulcerated mass infiltrating the wall of the bowel, thus intruding upon the lumen, or as one or more nodular growths under the mucous membrane and adherent to it. If ulceration has occurred, there is apt to adhere to the finger bloody and mucoid matter characterized by extreme and persistent fetor. V. Leube especially calls attention to hemorrhoids as a symptom of cancer of the rectum, and says they are seldom absent, because of the resistance opposed to the return of the venous blood. He claims he has discovered rectal cancer in examination suggested by hemorrhoids when no other symptoms were present. So, too, the presence of secondary cancer of the liver should suggest examination of the rectum, since marked instances of the former have been found associated with cancer of the rectum, otherwise latent.

Almost all morbid growths affecting the rectum are cancerous. Polypi, mucous and fibromatous, are occasionally found in children, produce dysenteric symptoms, including bloody discharges, while they may project from the rectum during stool. Lipomata and other histioid tumors have been found at autopsy without having caused symptoms.

Prognosis and Treatment of Cancer of the Bowel.—The prognosis of cancer of the bowel is always unfavorable. Occasionally operative procedures have prolonged the life of the patient at the expense of an artificial anus in the lumbar or abdominal region, while resection has even been made with some degree of success. Especially happy have been the results in some cases of exsection of the rectum.

The propriety of operation should, therefore, always be considered. Should it be decided against, the patient must be nourished by easily assimilable foods, such as peptonoids and peptonized milk, by the mouth or bowel, as circumstances may determine. A regular and sufficient evacuation of the bowels should be carefully looked after lest impaction add its inconveniences to the others present.

DISEASES OF THE LIVER.

ABNORMALITIES IN THE SHAPE AND POSITION OF THE LIVER.

Altered Shape.—The only abnormality in the shape of the liver requiring special mention is the laced-off or corset liver. In this the right lobe is divided by a transverse furrow, more or less deep, into two nearly equal parts. In extreme cases the connecting furrow is a mere fibrous band, and the liver can be folded on itself; in others it contains more or less liver parenchyma. It is usually caused by the pressure of a tight waist-band or corset, and accordingly is more frequent in women, but it occurs also in men.

It seldom gives rise to any symptoms, but sometimes leads to confusion in diagnosis, being, especially, frequently taken for a movable kidney or abdominal tumor, for the inferior portion sometimes extends as low as the crest of the ilium. This confusion is increased if, as sometimes happens, a loop of intestine lies in the furrow and gives a tympanitic note on percussion, whence the inference that the lower portion is a separate object. Skilful palpation is a valuable means for determining the true nature of such a condition. The edge of the liver should be followed around from the epigastrium into the right lumbar and iliac regions. If the continuity with the supposed tumor is uninterrupted the latter must be a portion of liver laced off. It is not unlikely that such a condition may occasion symptoms of dragging and weight, with the nervous strain frequently incident to such condition, like that which is so characteristic of floating kidney. Such a condition of the liver is said to be one of the favoring causes of cholelithiasis by reason of its interference with the natural onward movement of the bile.

Abnormality of Position.—The liver in cases of transposed viscera is found on the left side. More frequently it is simply turned downward or upward, anteverted or retroverted as it may be on its transverse axis, chiefly as a consequence of tight lacing in women. It may be pushed upward above its normal site by ascitic fluid or abdominal tumors, and downward by pleuritic effusion on the right side or by emphysema of the right lung.

The *floating liver* is by far the most interesting of these conditions. When it occurs the natural seat of the liver is vacant, especially when the patient is in the upright position, occupied usually by hollow viscera, or, in rare instances, by morbid growths. The condition of such movableness is a long suspensory ligament and a coronary ligament so stretched as to form a sort of mesohepar, which permit the liver to fall out of its normal position. It occurs usually in women past middle life, with loose abdominal walls, and is favored by tight lacing. It has been met with in men. It is sometimes responsible for the condition known as the pendent belly. It is a rare condition.

The organ itself is usually easily recognized as a large, hard, but movable tumor below its normal place, and having also the shape and size of the liver, while the normal site is tympanitic on percussion or occu-

pied by organs which do not give the same outline on percussion. The suspensory ligament may also be felt. The organ may generally be restored to its normal position when the patient is recumbent.

The same dragging symptoms mentioned as characteristic of the constricted liver with the usual contingent of nervous symptoms which succeed upon it and the movable kidney may be here present.

Treatment.—The treatment for both of these conditions, the constricted and the displaced liver, must consist in some instrumental means by which the organ or constricted portion can be held in position.

Diseases of the Bile Passages and Gall Bladder.

JAUNDICE, OR ICTERUS.

Definition.—Jaundice is not a disease, but a symptom, consisting in a yellowish coloration of the skin and other tissues by coloring-matters ultimately derived from the bile. The shades of coloring range from a very pale, scarcely appreciable, yellow to a brown olive hue. It is a symptom present in so many different diseases of the liver, and so associated with other symptoms more or less constant, that its separate consideration is justified.

Etiology.—As intimated, its immediate cause is a deposit of pigment in the skin, which, in the majority of cases, is reabsorbed bile pigment. In other instances the pigment represents the coloring-matter arising from a rapid disintegration—hæmolysis—of red blood discs, so rapid that the liver, spleen, and kidneys all combined are unable to eliminate it. That jaundice can also be due to a suppressed secretion, the result of extensive destruction of liver cells, has been rendered very unlikely by the experiments of Stein, who found that jaundice did not occur when the entire blood supply of the pigeon's liver was cut off. The jaundice due to bile absorption has received the name *hepatogenous* jaundice because of its purer hepatic origin, the latter *hæmatogenous*, because the disintegrated blood is its direct source.

Resorption of bile takes place when there is obstruction to its onward movement, caused, for example, by impaction of a gall-stone in some one of the biliary passages; closure of the duodenal end of the common bile duct by inflamed and swollen intestinal mucous membrane; complete or partial obliteration of the duct by adhesive inflammation; and pressure from without by morbid growths. These growths may be enlarged glands in the fissure of the liver, or tumor in the gall bladder, in the liver itself, in the pancreas, stomach, and especially cancer of the pylorus and duodenum. More rarely, tumors of the kidney or omentum, abdominal aneurism of the cœliac axis or aorta, or enlargement of the uterus may occasion obstruction. So may fecal accumulation. The morbid states in the liver which may produce jaundice are cancer, abscess, hydatid cysts, and cicatrices, all of which will be referred to again. Reduced pressure in the blood-vessels of the liver, as contrasted with that in the biliary vessels, also favors resorption

of bile from the latter. Such explanation is speculative, but thus have been explained those interesting cases of jaundice brought about by emotion. Those who have read the charming story of "Put Yourself in His Place," by Charles Reade, will recall the case of Henry Little, whose attack of jaundice is described with the skill of an expert physician.

It should be mentioned, also, that the hæmatogenous form of jaundice has recently been denied by Stadelmann, who holds that all jaundice is hepatogenous in origin, and that the needed condition of obstruction is secured in the so-called hæmatogenous form by a plugging of the smaller bile ducts by viscid bile or catarrhal secretion, or by compression of these ducts by swollen adjacent liver cells, or by leucocytic infiltration of the interstitial tissue. I do not as yet feel justified in discarding the heretofore accepted classification.

Associated Symptoms.—(*a*) *Of Hepatogenous or Obstructive Jaundice.*—This is the usual form of jaundice. Young adults are most frequently affected, but all ages are subject to it. In addition to the coloration described, there is often an annoying *itching of the skin*, due to irritation of the deposited bile-pigment. Further evidence of the irritation thus caused is seen in occasional *eruptions*, such as urticaria, lichen, and even furuncles. A *bright yellow coloration of the sclerotic coat of the eye* is as constant as the staining of the skin, while the mucous membranes are often similarly tinged.

After the skin, the *urine* exhibits the most conspicuous alteration, even in mild cases. Indeed, "bilious urine" is sometimes the first symptom. The color may be slightly yellow, or deep brown like that of porter. The presence of bile-pigment in the urine is readily shown by Gmelin's nitrous acid test, though ordinary nitric acid answers nearly as well. A few drops of the urine and half as many of the acid are placed on a porcelain plate and gradually allowed to approach and fuse, when a brilliant play of colors appears, in which green, yellow, red, and violet are most easily recognized. The reaction is due to the oxidation of the bilirubin by the acid. The demonstration of the biliary acids by Pettenkoffer's test with cane-sugar and sulphuric acid is impossible unless the bile acids be first separated by a tedious process. One of the most reliable ways of recognizing bile in the urine is by the stained cellular elements which it contains. Under no other circumstances are the bright yellow-stained cells found, and they are even met with when the quantity of coloring matter is insufficient to react by Gmelin's test. In a few cases the bilirubin reaction is not obtainable when the urine contains in increased amount its normal coloring-matters, urobilin or hydrobilirubin, *i. e.*, reduced bilirubin. Of the remaining *secretions*, the perspiration is often stained, the milk rarely, the tears, saliva, and mucus not at all. There is sometimes a bitter taste in the mouth, showing an elimination of some constituent of the bile by the buccal glands, probably the salivary.

On the other hand, the *fæces* are devoid of biliary coloring-matter, and their pale grey or pipe-clay color has long been significant of the absence of bile. For the same reason the bowels are usually constipated and the discharges pasty, ill smelling, and acid. Occasionally there is *diarrhœa*, which may be caused by the irritating effect of the fæces dis-

posed to rapid decomposition, because of the absence of their natural antiseptic ingredient. For the same reason, too, the absorption of fats is hindered. There may be other signs of gastro-intestinal derangement, such as *loss of appetite, nausea, fetid breath, fulness in the epigastrium* after eating. *Gastro-intestinal hemorrhages* have been noted in grave cases. In cases of long standing there may be *albuminuria* as well, with bile-stained tube casts.

Very characteristic of simple obstructive jaundice is a *slow pulse*, which may be as infrequent as 50, 40, or 30. It must be due to some stimulating effect on the inhibitory action of the pneumogastric nerve. The breathing rate, on the other hand, is normal.

The chief subjective symptom of jaundice is *depression of spirits*, which may even amount to melancholia. *Irritability* is also prominent. *Headache and vertigo* are frequent. *Vision* is variously affected. To some, objects appear yellow. Some see better by obscure light—nyctalopia; to others, the approach of darkness is associated with more than usually difficult vision—hemeralopia. Grave nervous symptoms, rarely manifested, are *sudden coma*, *acute delirium*, and *convulsions*. These usually supervene in cases of long standing, and are attended by *fever*, *rapid pulse*, and *dry tongue*, the symptoms, in a word, of the typhoid state. The term cholæmia is applied to the sum of these symptoms, and the condition is regarded as due to the presence in the blood of the constituents of bile, of which cholesterin is the most important, whence also the name cholesteræmia.

The *liver* remains usually of normal size, but there may be slight enlargement. On the other hand, if the obstruction be long continued, there may be over-growth of interstitial tissue, hardening, and slight reduction in size. It is also bile-stained, as are other internal organs, especially the kidneys.

The duration of this form of jaundice is variable, from two weeks to a year. In chronic cases remission and exacerbations occur, but the longer the duration the more likely is there to be some organic change in the liver.

(*b*) *Of Hæmatogenous Jaundice.*—The symptoms of this form are those of the diseases which are responsible for the hæmolysis, viz., acute yellow atrophy, phosphorus poisoning, yellow fever, bilious fever, typhoid, typhus, and relapsing fevers, pyæmia, pernicious anæmia, snake poison, chloroform, and other poisons. In all of these there is some toxic agent working destruction of the blood. It should be added, that in this form of jaundice the stools are not clay-colored. The urine also is less bile-stained, though the true urinary pigments, notably urobilin, are often very much increased.

Diagnosis.—One of the most frequent mistakes of the beginner, and a constant one of the laity, is to mistake for jaundice a dirty, yellowish discoloration of the skin, known as sallowness, which is symptomatic of general ill health, especially uterine disease in women, and of malarial poisoning. It is probably an anæmia, and may be distinguished from jaundice by the fact that it is not associated with staining of the conjunctiva and secretions. It is, moreover, not a yellow, but a dirty brown. One needs only to have his attention aroused to avoid error.

Much more closely does the discoloration of the skin in Addison's disease resemble that of some cases of jaundice. In the former there is no discoloration of the sclerotic coat nor of the urine, while the fæces remain natural. In Addison's disease the exposed portion of the body and its flexures are more deeply stained.

The purpose of diagnosis includes the discovery of the cause and seat of obstruction. In the first place, most cases of acute jaundice are due to catarrhal inflammation of the common bile duct. If associated with fever, it may be assumed that the smaller ducts are involved. If the gall bladder is distended and can be felt at the edge of the liver, there is probably obstruction of the common duct, and if there is pain and tenderness the obstructive agent is probably a gall-stone. The paler the fæces the more complete must be the obstruction, and the more likely is it to be in the common duct, for with obstruction of the cystic duct there may still be a discharge of bile into the intestine; also with obstruction of the hepatic duct until the gall bladder is empty, which, however, soon happens. Obstruction of the hepatic duct is unassociated with distention of the gall bladder, while there will be jaundice. Obstruction of the cystic duct may still be associated with distention of the gall bladder, either through transudation or pus formation, but there will be no jaundice. If the jaundice is chronic or permanent, we must look for some organic change in the liver or external permanent cause of obstruction outside of its condition. In the hæmatogenous form, the jaundice is usually so plainly secondary to other symptoms that there is little difficulty in recognizing its cause.

Prognosis.—This depends on the cause producing it. Ordinary catarrhal jaundice invariably recovers in from two to six weeks, and jaundice from impacted calculus usually sooner or later. When due to other causes, its duration depends on them. In the hæmatogenous form the duration is brief, because the termination of the disease causing it is usually early and fatal.

Treatment.—The treatment of both forms of jaundice must be directed to the conditions causing it, and will be appropriately considered in treating them.

ICTERUS NEONATORUM.

Synonym.—*The Jaundice of the New-born.*

Jaundice occurs in new-born children in a simple and harmless form, with symptoms comparable to obstructive jaundice, and in a grave form comparable to hæmatogenous jaundice. The first is probably a form of obstructive jaundice due to like causes, though it has been assigned a hæmatogenous origin. It is much the more frequent, and disappears in from a few days to a couple of weeks. The latter is usually fatal.

A patulous ductus venosus has been suggested as an avenue through which the portal blood which contains bile enters the circulation.

The grave form has been found associated with absence of the hepatic duct or common duct, with congenital syphilitic hepatitis and with septic phlebitis of the umbilical vein.

Treatment.—The simple form of jaundice of new-born infants demands no treatment. In the graver forms, treatment is of no avail un-

less the condition be traceable to syphilis, when it demands the treatment of that disease in its tertiary form.

SIMPLE CATARRHAL JAUNDICE.

SYNONYMS.—*Duodeno-cholangitis; Inflammation of the Common Bile Duct.*

Definition.—The term catarrhal jaundice is applied to jaundice due to any inflammation of the common duct not the result of impacted gall-stone.

Etiology.—The most frequent cause of such inflammation is the extension of a gastro-duodenitis into the common duct. To the same cause is ascribed the jaundice sometimes occurring with passive congestion of the liver due to mitral valvular heart disease, also that found in association with the infectious diseases, especially pneumonia, or with mental emotion. Catarrhal jaundice may also be epidemic. The jaundice in hypertrophic cirrhosis is probably also due to cholangitis.

Morbid Anatomy.—Opportunities of studying post-mortem conditions after catarrhal jaundice are not often afforded, but when occurring, the duodenal end of the duct, the *pars intestinalis*, has been mostly involved. In it the mucous membrane is swollen, while its orifice and the diverticulum of Vater may be filled with mucus. The inflammation may extend up into the common and cystic duct and even higher, into the hepatic duct. Suppuration does not take place in this form of cholangitis.

Symptoms.—Excepting the jaundice, there may be no symptoms. There is no pain, but there may be *tenderness*, due to gastro-intestinal derangement rather than to the hepatic state, while such derangement may also lead to *general malaise*, *loss of appetite*, *nausea*, *vomiting*, a sense of *fulness*, *constipation*, or irregular action of the bowels. There may also be slight *fever*.

Diagnosis.—The presence of jaundice without pain or other symptoms points almost invariably to catarrhal jaundice. The same diagnosis is justified by the presence of the symptoms of gastro-intestinal catarrh, of associated mitral disease, or any of the infectious diseases.

Prognosis.—Unless associated with infectious diseases or with hypertrophic cirrhosis, the prognosis of catarrhal jaundice is favorable. In the diseases referred to the danger is not from the jaundice but from the diseases with which it is associated.

Treatment.—The treatment of catarrhal jaundice resolves itself into two parts—first, that for the catarrhal state; second, that demanded by the absence of bile in the small intestine.

For the catarrhal inflammation, either of the duodenum adjacent to the duct or of the duct itself, local depletion is indicated. This is accomplished by the use of saline aperients and the natural mineral waters which act similarly, *i. e.*, produce watery stools. Of the former, Rochelle salts, Epsom salts, or the solution of the citrate of magnesium are representative; while the Saratoga, Hunyadi Janos, Friedrichshalle, or Rubinat and Carlsbad waters represent the latter. These should be taken daily in aperient doses. In this country the Saratoga mineral

waters, particularly those of the Hathorn Spring, are especially valuable, and no better course can be pursued by those who can afford it than to spend a couple of weeks or more at Saratoga. The Bedford Springs waters, near Bedford, Pa., are also useful, but not nearly so efficient as the Saratoga waters. Of foreign waters, those of Carlsbad are especially valuable, and in Europe these springs may be resorted to. Their use may also be associated between meals with that of the alkaline mineral waters, of which those of Vichy and Vals are the type. These waters are largely employed in this country, and may be availed of at home. There is no indication for the use of calomel, which is so often prescribed, as it is not reasonable to believe the secretion of the bile can be so stimulated by it as to force onward any obstruction, whether by calculus or swollen mucous membrane, until the latter is depleted. After the flow into the intestine is resumed, calomel may be given to stimulate it further. Podophyllin and colocynth may be used for the same purpose. Sodium salicylate has also a reputation to this end. Irrigation of the large bowel with cold water has been recommended as a means of stimulating the descent of the stone.

The second indication should be met by the use of such food as does not require the bile to facilitate its digestion or absorption or to prevent its decomposition. Fats and oils should, therefore, be avoided. Hence, skimmed milk, animal broths, and egg albumin, with an abundance of liquids, are indicated. The liquids may be some one or more of the mineral waters above named, or, in their absence, plain water. Warm bathing is especially indicated, as it causes elimination by the skin and relieves the itching. Lotions of carbolic acid and glycerine are also useful to this end.

IMPACTED GALL-STONE.

Synonyms.—*Hepatic Colic; Cholelithiasis.*

Etiology.—Since the great bulk of the gall-stone is cholesterin, an evident condition of its formation is a precipitation of this substance from the bile, of which it is the chief constituent. The thicker the bile the more likely it is to deposit. What other conditions coöperate is not precisely known. If, as is supposed, the cholate salts of sodium hold in solution cholesterin, it is plain that their decomposition or destruction may cause precipitation, which has also been ascribed to the action by microörganisms in the bile.

Gall-stones are found in adults only, and their tendency to form appears to increase from the age of thirty onward. Most patients who consult us for their effects are over thirty and under fifty. Cholelithiasis is also very much more frequent in women than in men; according to Naunyn, four times as frequent, and especially so in women who have borne children or have had abdominal tumors.

Naunyn says, too, that 25 per cent. of all women who die have calculi in the gall bladder. Sedentary habit and tight lacing are held partly responsible for this, and with some reason, as both of these conditions are calculated to impede the movement of bile. Cholelithiasis has been found associated with the habit of free eating of starchy and saccharine

foods and in stout persons; yet I recall striking cases among the lean also. The movable liver and the movable right kidney are also said to predispose to cholelithiasis. Constipation and a tendency to depression of spirits are apt to be associated, probably as effects rather than causes.

Morbid Anatomy.—The gall-stone itself is a brown object, nearly spherical, oval or faceted, and even polygonal in shape, usually the size of a pea, or as small as a millet-seed, producing in aggregation "gall sand." The faceted shape is produced by close packing of a large number of stones in a gall bladder, as frequently happens. More rarely the stone is irregular, mulberry-shaped. In addition to cholesterin, which makes up from 70 to 80 per cent. of most stones, they contain in various but still small amounts bile pigment, calcic carbonate, and organic matter. A few are made up almost entirely of bilirubin and lime. On section the stone exhibits either a concentric or homogeneous appearance, with or without a nucleus of bile pigment or organic matter, and very rarely of some foreign body. The cholesterin stones are almost completely soluble in etherized alcohol, whence beautiful crystals of cholesterin may be obtained after evaporation.

In addition to their enormous accumulation in the gall bladder, where they may be counted sometimes by hundreds, they are found anywhere in the biliary tract between the duodenal end of the common duct and the ultimate ramification of the bile vessels. Outside of the gall bladder, the cystic duct and the common duct are naturally the situations in which lodgment most frequently occurs. If in the common duct, it is usually at the orifice of the papilla in the diverticulum of Vater, and from the duodenal side the stone feels as though it was directly under the mucous membrane. Two or even more stones may be found in the duct. The common duct under these circumstances may attain a diameter of an inch (2.5 cm.) or more. Permanent obstruction of the cystic duct causes dilatation of the gall bladder—*hydrops vesicæ felleæ*. Such dilatation may be enormous, filling the entire abdominal cavity; usually it is more moderate, but the contents frequently amount to a pint (500 c. c.) or more. The contents are a colorless, viscid, or watery fluid, more or less albuminous, neutral or alkaline in reaction; the greater the dilatation the more aqueous and unlike bile do its contents become. In any situation the stone may produce ulceration and even suppuration, with perforation into the peritoneal cavity or adjacent organs, the duodenum, stomach, transverse colon, right renal pelvis, ureter, through the diaphragm into a bronchus and into the abdominal wall.

Symptoms of Acute Impaction.—The characteristic symptom of impacted gall-stone is *biliary colic*, but biliary colic is by no means always present with every case of cholelithiasis. The gall bladder is often found full of calculi without the suggestion of a symptom. Small stones even pass into the duodenum without producing symptoms. Commonly, however, they lodge while in this transit and give rise to attacks of *pain* which are known as biliary colic. This pain is usually sudden, very severe, often excruciating, and the patient writhes in agony and sometimes faints. It is usually referred to the epigastrium, whence it radiates in all directions over the abdomen and at times into the right shoulder and arm. As a rule, however, it is localized on the right side

under the liver. It is a sharp and cutting pain. There is always *tenderness* in this region, which varies in degree. It is sometimes associated with a more or less rigid state of the abdominal muscles of that side. The duration of the pain is that of the lodgment of the stone, and it may be from a few hours to weeks, ceasing rather suddenly when the stone is discharged into the bowel. There may, however, be remissions. *Nausea* and *vomiting* are almost invariable symptoms of biliary colic. They often bring temporary relief through the resulting relaxation. *Fever* is soon added to the pain, while a *chill* is not infrequent. The temperature, usually 102° F. to 103° F. (38.8° to 39.5° C.), may even reach 105° F. (40.5° C.). It may be intermittent, but such intermission is more apt to be associated with prolonged obstruction, constituting with a chill a part of the symptoms of so-called *hepatic fever*, to be next considered.

If the attack last long enough, *jaundice* almost always supervenes, whence we infer, too, that the stone is likely to be in the common duct, having probably started in the cystic duct. Three or four days may elapse between the beginning of obstruction and the supervention of jaundice, the degree of which increases with the completeness and duration of obstruction. The entrance of the stone into the common duct may be attended by one of the remissions alluded to, though the jaundice grows even deeper on account of the more thorough obstruction to the descent of the bile.

The liver is sometimes slightly enlarged, as determined by percussion. A rare symptom is *collapse* with fatal syncope, due to perforation at the seat of lodgment, with consequent *peritonitis* and *shock*.

Diagnosis of Acute Impaction.—This is commonly easy. While the pain may be more or less diffuse, it is for the most part localized in the right lower thoracic and upper abdominal regions, and the tenderness is always there, while if jaundice and biliary urine are present, all doubt is removed. Nephritic colic and biliary colic are confounded with surprising and unjustified frequency. In the former condition, the pain starts in the lumbar region and radiates downward into the groin, the testicle, and the inside of the thigh. Such error is fortified by the fact that bilious urine is too often confounded with blood urine. It should be necessary only to mention this to guard against error.

Our growing knowledge of appendicitis has led to the discovery that the pain characteristic of this disease is sometimes localized in the right hypochondrium, where, indeed, the appendix has been found at operation. Jaundice and bile-stained urine do not, however, attend appendicitis. Gastralgia has been confounded with biliary colic, but attention to the symptoms described when treating that affection should secure against error. The term hepatic neuralgia has been applied to an apparently causeless pain, sometimes felt in the neighborhood of the liver, but it is less severe than the biliary colic and unaccompanied by any of the other symptoms.

It is very important, immediately after an attack of supposed biliary colic, to search for a stone in the fecal discharges. For this purpose the mass should be placed on a sieve and water passed over it until all soluble parts are run out. Such examination should be kept up for

several days after the attack, for the stone is not always immediately passed.

A *pseudo-biliary* colic is to be remembered as a possible event in nervous women. It is characterized by the absence of jaundice.

Prognosis.—The termination of an ordinary attack of biliary colic is, in the vast majority of instances, favorable. It is only in the rare cases, where perforation takes place, that a fatal ending follows. More frequently the escape of the stone is long delayed, producing the symptoms of chronic impaction, to be next described.

Chronic Impacted Gall-stone.

Synonyms.—*Chronic Cholangitis; Hepatic Fever.*

Symptoms.—These vary a good deal with the seat of the impaction and its duration. From this standpoint they may be divided into certain groups.

(1) *Symptoms Due to Chronic Calculous Obstruction of the Cystic Duct.*—The immediate result of such obstruction is dilatation of the gall bladder, or hydrops vesicæ felleæ, already referred to. The occasional enormous dilatation has more than once been mistaken for ovarian disease, a mistake the more to be excused when we remember that jaundice is often absent. More frequently the dilatation is moderate, and can be felt below the edge of the liver as a round or ovoid elastic tumor, in which fluctuation may sometimes be obtained.

A cancer of the gall bladder, which will form a tumor in the same locality, is much more tender; it is harder and more uneven, and jaundice is invariably associated with it, while the patient is much more seriously ill and declines more rapidly. There should be no confusion with a movable kidney, which furnishes a different physical condition. An aspirator needle may be used to confirm the diagnosis.

(2) *Symptoms Due to Chronic Calculous Obstruction of the Common Duct.*—If the common duct is obstructed, dilatation of the gall bladder does not necessarily follow, and if it does occur, the dilatation is moderate. On the other hand, when the common duct itself, the hepatic duct, and the branches uniting to form it become dilated, there is added a set of symptoms of greater interest, for the special study of which we are indebted to Charcot and Murchison abroad and William Osler in this country.* The symptoms in general may be said to be those of a chronic cholangitis, which may be catarrhal or suppurative.

Chronic *catarrhal cholangitis*, in addition to the persistent jaundice and pain, which is paroxysmal, is characterized by *ague-like attacks*, consisting in chills, fever, and sweats. These occur at surprisingly regular intervals, resembling in this respect the quotidian, tertian, and quartan spells of intermittent fever, with which the condition has been confounded. They may occur for weeks at a time and then remit. *Pain* is commonly associated with the ague-like spells, but is not always present. The chills may be extremely severe, the sweats also, and the fever cor-

* On "Fever of Hepatic Origin, Particularly Intermittent Pyrexia, Associated with Gall-stone."—Johns Hopkins Hospital Reports, Vol. II, No. 1, 1890.

respondingly high, the temperature reaching sometimes 105° F. (40.5° C.). The *jaundice* usually deepens after an attack. There may be *nausea* and *vomiting*. The duration may be indefinite, from a few months to years, and the patient may yet recover. Or he may perish, although the exhaustion is extremely slow and the effect on the general health barely appreciable from week to week. The fever is probably irritative, although it has been ascribed to the omnipresent organism—bacterium coli commune. There is rarely slight enlargement of the liver appreciable to physical examination, and in long-protracted cases some fibroid induration may be expected to take place.

Suppurative cholangitis is marked symptomatically by a fever which is more of the septic type, with remissions rather than intermissions. The jaundice is less marked, the liver is tender and enlarged, the duration shorter, and termination fatal. The inflammation involves more or less of the ducts of the liver, whence it may extend into the liver substance or gall bladder.

Diagnosis.—There may be some difficulty at first in the diagnosis of hepatic fever, but the persistent jaundice, the ague-like paroxysms of chills, fever, sweats, and pain are a combination of symptoms belonging to no other condition. The suppurative form is characterized by the more continuous fever and the more serious aspect of the septic state, its shorter course, and ultimate fatal termination. The catarrhal form is less serious and quite often terminates favorably.

Other Remote Results of Gall-stone Impaction.—Rarer terminations of impacted gall-stones are the various forms and situations of biliary fistulæ, alluded to when treating of the morbid anatomy; phlegmonous cholecystitis associated with high fever, intense prostration, and death from fatal peritonitis; empyema of the gall bladder, already alluded to as a result of suppurative cholangitis; perforation and abscess formation in adjacent organs; calcification of the gall bladder, and atrophy of the organ. The latter is not infrequent. I have seen many gall bladders which did not hold more than a drachm (four c. c.) or two of bile, and sometimes there is a mere remnant left in the shape of a fibroid mass; at other times the shrunken bladder closely embraces a gall-stone of large size. Suppurative phlebitis and abscess of the liver may also be due to gall-stone, causing a puriform thrombus in an adjacent branch of the portal vein.

In other instances the gall-stone is of such a size as to obstruct the bowel when discharged into it, although it may have been discharged through the natural channel, as evidenced by dilatation of the common duct. For the most part, such discharge is by ulceration into the intestinal tract. This subject has been sufficiently considered when treating of obstruction of the bowels.

Treatment of Impacted Gall-stone and its Complications.—The first indication is the relief of pain. This is best accomplished by the hypodermic injection of morphine, the action of which is favored by combination with atropine. Scarcely less than $\frac{1}{4}$ grain (0.0165 gm.), with $\frac{1}{150}$ grain (0.00044 gm.) of atropine suffices, and this has often to be repeated. The use of anodynes must be kept up as long

as needed. The atropine favors the relaxation needed to release the calculus.

Whether anything else can be done toward releasing the stone is not established. The nausea and vomiting, which are so often symptoms, sometimes relieve the pain by the relaxation they produce, such relaxation being at times sufficient to favor the onward movement of the stone. Anæsthesia by ether or chloroform may act similarly, and it is sometimes necessary to exhibit these cautiously by inhalation, while waiting for the morphine to act. Hot baths or fomentations applied to the region of the liver may also be similarly effective. Some solvent for the stone is constantly inquired after. Ether, turpentine, and sweet oil, although lauded for this purpose, have been tried and found wanting. Durande's remedy consists of turpentine one part, ether four parts; dose, 15 drops three times a day.

The free use of alkaline mineral waters does seem to favor the dislodgment of the stone, especially if the authorities at Carlsbad are to be relied on, who claim the discharge of immense numbers of biliary calculi under the use of Carlsbad water. Certainly no harm can attend its use, and when within the power of the patient to get it, it may be freely taken. In this country, however, the Saratoga waters may be used in its place. These waters are saline and not alkaline waters, but they seem to fulfil much the same indications. Those containing the largest proportion of alkaline carbonate are to be preferred. The imported Vichy and Vals waters—true alkaline waters—are to be recommended for the same purpose.

Surgical procedures have of late been availed of even with a view to exploration, and, if done by competent surgeons with due antiseptic precautions, cannot be regarded as more dangerous than most abdominal sections. The curative measure consists in removing the impacted stone and emptying the gall bladder of others. Under such procedure is included aspiration of dilated gall bladder, which is justified in the event of a certain diagnosis. A carefully conducted exploratory operation is little more dangerous, but, on the other hand, should not be done until the case has assumed some chronicity, or repeated attacks endanger the life of the patient.

The preventive treatment is important, and although an attack of biliary colic very commonly does not take place until a number of stones have accumulated in the gall bladder, so that the descent of one is apt to be followed by that of another, prophylaxis should still be availed of. To this end diet is important. The patient should eat sparingly of hydrocarbons and carbo-hydrates, omitting every form of fat, alcohol, sugar, and starch. Meat, cheese, and glutens, on the other hand, are allowable.

The alkaline and saline mineral waters are even more indicated between the attacks than during them, and their more or less continued use is advisable, especially in the morning, when their efficiency is also increased by their being taken hot. The sodium salts have considerable reputation for their efficiency in preventing the concentration of bile and formation of gall-stones, having been long ago recommended by Prout. The phosphate is the modern favorite, in drachm doses in the morning,

or more frequently, but the sulphate is more constant and more potent in its results, and little if any more unpleasant. The sodium salicylate has also a similar reputation, and may be used where no effect on the bowels is desired. By either of the former or by the aperient mineral waters a daily action of the bowels should be secured, while a proper hygiene of the body, in which daily exercise, bathing, and frictions should play a conspicuous part, should be constantly maintained.

I have been in the habit of placing my patients, between attacks, on the succinate of sodium, in doses of five grains (0.32 gm.) three times a day, and it has so happened that I have never met a recurrence in one of these cases, although many of them passed out of my observation and may have had attacks without my knowledge.

OTHER AFFECTIONS OF THE BILE DUCTS.

Carcinoma may invade the bile ducts from the gall bladder, and possibly from primary or secondary cancers in the parenchyma of the organ. Or it may start in the common or hepatic ducts and invade adjacent structures. The symptoms, which always include those of obstruction, are not otherwise differentiable from those of the two forms whence it may start.

Stenosis, or complete occlusion of the common duct by inflammatory adhesion, has been referred to when treating of obstructive jaundice. Sometimes it follows the ulceration attending the passage of a gall-stone. Stenosis also results from external pressure, due to morbid growths and other causes, alluded to on p. 331.

Parasites may enter the larger biliary passages and produce obstruction. Such are echinococci, which may enter the ducts primarily in the larval state and develop there the *hydatid* cyst with resulting obstruction; or, as is more frequent, the sac perforates or compresses a duct in the course of its growth. The other symptoms of echinococcus disease are added to those of obstruction thus produced, or the cysts may appear in the stools, vomited matter, or expectoration. Cases are reported in which the *distoma hepaticum* has been found lodged in the hepatic duct and *round-worms* in the common and hepatic ducts. A remarkable specimen, containing a number of lumbricoids lodged in these ducts, is in the Wistar and Horner Museum of the University of Pennsylvania. The symptoms of these last conditions would be indistinguishable from hepatic obstruction from other causes.

Cicatricial contraction the result of perihepatitis, syphilitic disease, duodenal ulcer, and cholelithiasis should also be mentioned as a cause of external compression of biliary passages, to be recognized, if at all, by the aid of the associated symptoms of the diseases causing it. In the first there may be a peritoneal friction in the neighborhood of the liver, audible and palpable.

CANCER OF THE GALL BLADDER.

Etiology and Morbid Anatomy.—Though rare, this affection has excited much interest and has been thoroughly studied, with widely different results in some points. Thus John H. Mussen, in a study of 100 cases, found it three times as frequent in women as in men, while Courvoisier, in a study of an equal number, found it five times as frequent among men.

It is sometimes primary, when it begins usually in the fundus. At others it occurs by contiguous invasion, either from the liver or adjacent abdominal organs. Cancer may also extend from the gall bladder to adjacent parts. The primary form is associated in at least 87 per cent. of all cases with biliary calculi, the ulcerative and cicatricial tissue due to which may be the starting point, as suggested by Zenker, resembling in this respect cancer of the stomach. This, too, may account for the greater frequency of the disease in women, who are much more commonly the subjects of gall-stone. It seems to me much more reasonable to regard the biliary calculi as secondary to the cancerous disease which may produce changes in the composition of the bile. A more or less solid and irregular mass is the ultimate result.

Symptoms.—These are, first, *jaundice*, present in 69 per cent., great *tenderness* with *pain; vomiting*, sometimes of blood, *bloody stools*, and *dropsy*, at times succeeded by the *cancerous cachexia*. But none of these are distinctive, being found in cancer of the pylorus, duodenum, and transverse colon. The presence of a hard, uneven, and tender tumor in the neighborhood of the gall bladder and which moves with the liver in respiration confirms the suspicion. This has, in fact, been found in about 69 per cent.

Treatment.—The treatment can only be palliative.

HYPERÆMIA OF THE LIVER.

Passive Hyperæmia—Red Atrophy.

The hyperæmia of the liver which is of chief clinical importance is passive hyperæmia.

Etiology.—It is always due to obstruction to the movement of the blood toward or through the heart. Valvular heart disease is the most frequent cause, though diseases of the lungs, such as emphysema or cirrhosis, intrathoracic growths, disease of the pleura, compression of the vena cava, or other cause resisting the movement of blood through the organ are all competent to produce passive hyperæmia of the liver. On the other hand, such hyperæmia cannot always be inferred without the presence of some one of the causes named, though there are a few symptoms which are quite characteristic of it.

Morbid Anatomy.—The appearances of the organ are governed by the duration of the congestion If it has been of short duration the

liver rapidly resumes its natural size and appearance after death. Even in long-continued passive congestion the liver after death becomes very much smaller than during life by reason of the emptying of the blood-vessels, which rapidly succeeds death. In other respects, however, after prolonged hyperæmia it presents decided changes. It is dark in color and the vessels still contain an excess of blood, but the intralobular vein, *i. e.*, the central vein of each lobule, and its adjacent capillaries contain most blood, contrasting strongly with the peripheral or interlobular vessel and its adjacent capillaries. There is thus produced in one way that alternation of dark and light tint which constitutes the *nutmeg* liver and which is particularly conspicuous on section. It becomes even more marked at a later stage when the organ, in its ultimate atrophy, becomes reduced in size, constituting the so-called *red* or *cyanotic atrophy* of the liver—the atrophied nutmeg liver—the histology of which exhibits a destruction of the cells and capillaries in the centre of each lobule and a deposit of dark pigment in their places with an appearance of increased connective tissue. In the liver thus atrophied the blood-vessels also share in the destruction and short cuts are established between the branches of the portal vein and hepatic vein, while the latter may also become dilated. The exterior of the liver is smooth and the organ differs in this respect from the cirrhotic liver, though there is sometimes a slight overgrowth of the interlobular connective tissue.

Symptoms.—The liver at first is *enlarged* and *tender*, sometimes very much enlarged and exquisitely tender. The lower border, as determined by percussion, may be as low as the umbilicus and even lower. It may be the seat of pulsation, due to regurgitation of blood into it from the right heart. This pulsation is to be distinguished from a motion communicated to the liver by the action of the heart. In the true pulsation, the whole liver seems to dilate, and does dilate as the blood flows back into it, as contrasted with the downward movement communicated by the heart. Very characteristic of this enlargement is its changing size, which varies with the degree of congestion, diminishing as it diminishes, whether spontaneously or as the result of action of medicines producing cardiac compensation. *Ascites* is also a symptom. It does not occur, however, until a marked degree of passive hyperæmia or *secondary contraction* is attained. The ascites is partly the result of the general stagnation always present, and partly of the congestion of the portal system due to the backing of the blood of the hepatic vein into it. *Jaundice* is another rather rare symptom. It is due to the compression exerted on the fine interlobular gall-ducts by the over-distended interlobular capillaries into which the blood of the intralobular vessels is backed, thus producing an obstructive jaundice.

Scanty urine of *high specific gravity* is also a symptom, while *hyperæmia with enlargement of the spleen* and *hyperæmia of the mucous membrane of the stomach* are constant, as a result of the same cause.

Treatment.—The treatment of passive hyperæmia is the treatment of the condition causing it. Most frequently the cause is heart disease, and where the latter is amenable to digitalis or other heart tonics, the passive hyperæmia disappears with the restoration of compensation. Simultaneously the urine is increased, the general dropsy, ascites, and hydro-

thorax disappear. Such treatment is aided also by depletion from the portal side by purgatives. Blue mass is the type of these, but colocynth, elaterium, and compound jalap powder, or the simple salts, are also efficient. It sometimes happens that the general dropsy in these cases is dispersed by treatment, but the ascites remains, in which event we must suppose the simple passive congestion to be combined with some degree of atrophy, when the dropsy is more likely to remain. Treatment should now be supplemented by hydragogue cathartics or, still better, by tapping, followed up by dry diet and the hydragogues. A drachm (four gm.) or more of compound jalap powder may be given each morning fasting, or elaterium, one-sixth grain (0.01 gm.), every three hours until the bowels are moved.

It was suggested by George Harley to deplete the liver under these circumstances, and it has been put into practice by East Indian physicians, it is said, with good results.

Active Hyperæmia.

Definition.—This is a much less important condition than passive hyperæmia, and, indeed, is rarely recognized. A physiological hyperæmia of the liver takes place after each meal, which may be exaggerated and even continuous in those who over-eat and over-drink habitually. Such hyperæmia may lead to structural change, consisting ultimately in interstitial overgrowth. Such is also the hyperæmia which is associated with diabetes mellitus, and which is the associated condition of all glycosurias, whether experimental or the result of disease affecting the diabetic centre. Such is a vicarious hyperæmia said to take place during suppressed menstruation and after cutting off a hemorrhoidal flux.

Active hyperæmia does not, however, present any symptoms referable to it, unless it be that the dull ache and full feeling sometimes felt in the right hypochondrium be caused by such condition.

Treatment.—The treatment of fluxion to the liver must consist in measures which tend to diminish this, mainly the substitution of a scanty for an over-abundant diet, simple and easily-digested foods, dilute milk, and thin broths, the avoidance of fats, alcohols, and sugar.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER.

The portal vein is the seat of thrombosis and of inflammation constituting pylethrombosis and pylephlebitis, while the hepatic artery also becomes rarely the seat of aneurisms.

Pylethrombosis.—Thrombosis takes place in the smaller branches of the portal vein, which are constantly being obliterated in the course of a cirrhosis of the liver. Larger branches are sometimes invaded by cancer, or a gall-stone may be admitted into one of them by ulceration, or the lodgment of a parasite may be the focus about which a coagulum may form, while thrombosis may also be favored by the narrowing effect of pressure incident to the encroachment of a neighboring tumor.

Symptoms.—These are, of course, those already detailed when treating of cirrhosis, viz., ascites, hyperæmia, with their consequences induced more or less suddenly. And it is mainly by such suddenness and the intensity of the symptoms that we suspect the cause, especially if it be associated with any of the above-named conditions capable of producing it. In such an event the symptoms would come about in the course of a few days, instead of weeks and months. A caput Medusæ thus rapidly produced would mean that the thrombus had formed, not in the portal vein itself, but more peripherally, causing the para-umbilical veins to be filled from the more peripheral branches, which leave the portal vein in the suspensory ligament, and pass out to the neighborhood of the navel by two branches which communicate with the epigastric and internal mammary vein.

When pylethrombosis occurs, it sometimes happens that a complete collateral circulation is thus established, the thrombus undergoing the usual changes, while the portal vein may be ultimately converted into a fibrous cord. Osler reports such a case, in which compensation ultimately failed, and the usual symptoms, including hæmatemesis, supervened, and the patient died.

Pylephlebitis.—Trifling grades of pylephlebitis probably succeed the thrombosis referred to, but they are of no consequence. Much more serious is suppurative phlebitis, the result of septic embolism, or thrombosis arising from an inflammatory focus somewhere in the portal area or in the territory of the umbilical vein of the newborn child. Pylephlebitis is one of the causes of abscess of the liver. It is associated with the usual signs of septic infection, viz., chills, remittent fever, and sweats, while the symptoms which point to the liver are pain in that neighborhood, jaundice in most cases, and the signs of portal-vein obstruction more or less pronounced. Suppurative peritonitis is also sometimes added. Such phlebitis does not always proceed to the degree of abscess formation before death supervenes, perhaps because of want of time. The symptoms of abscess will be considered when treating of that subject, when, too, attention will be called to the diagnosis between it and suppurative phlebitis, so far as it can be made out.

Hemorrhagic infarct does not usually succeed the lodgment of an embolus in a branch of the portal vein, because of the free anastomosis of its branches with those of the hepatic artery, by which the lobular capillaries are supplied. Hemorrhagic infarct does, however, sometimes occur.

Changes in the Hepatic Artery and Vein.—The artery is sometimes dilated in cirrhosis of the liver; it may be the seat of endarteritis and sclerosis, and rarely of aneurism. Aneurism of the artery is a rare condition. The signs are a pulsating tumor, which may be the seat of a murmur. In the cases reported there have been hæmatemesis, bloody stools, jaundice from compression of the biliary ducts, and pain in the neighborhood of the liver due to compression of adjacent nerves.

The hepatic vein is subject to dilatation, alluded to in treating of passive hyperæmia, to stenosis, and to thrombosis extending backward from the right auricle.

FATTY LIVER.

Definition.—The term fatty liver is applied to a condition in which the cells of the liver are more or less completely converted into fat. This is accomplished, however, by two distinct processes. In one, there is an infiltration of the liver cells with fat drops, which simply push aside the protoplasm and cause its ultimate disappearance by interfering with its nutrition. In the other, there is a disintegration or metamorphosis of the protoplasm of the cell into various products, of which one is oil. In the former, fatty infiltration, the cell maintains its integrity, being simply filled with the fat drops ; in the latter, the cell disintegrates, and leaves a residue of which fat is the chief representative. It should be mentioned that some use the term "fatty liver" as synonymous with "fatty metamorphosis."

Fatty Infiltration.

Etiology.—Abnormal fatty infiltration occurs in two ways :—

(1) In case of over-ingestion of fat-producing substances, resulting in obesity, of which it is a part, and as the result of which the liver becomes a storehouse for fat.

(2) In a series of cachectic states, in which oxidation is interfered with and the fat which is ingested is not oxidized but accumulates in the liver. Such a condition is tubercular consumption, which is the most common cause of fattily infiltrated liver, except alcoholism. Excessive consumption of alcohol is attended by fatty infiltration because more carbohydrate is introduced than can be burned up. It is, therefore, stored in the liver cells.

Morbid Anatomy.—The fattily infiltrated liver is uniformly large, soft, and smooth. It exhibits an appearance somewhat different at different stages. Since the infiltration begins at the periphery of the lobule, we have, in the first stage, a simple distinctness of the line of demarcation between the adjacent acini. In the second stage this has become more marked, contrasting strongly with the darker color of the centre of the lobule, and producing one form of *nutmeg* liver—the liver of red atrophy, one of the results of passive congestion already described, being the other. In the third stage, the entire acinus is infiltrated, and the whole organ assumes a uniform yellow or brownish-yellow appearance, from complete fatty infiltration of the cells. The organ is also anæmic. In this latter stage it is that we have the macroscopic changes complete, the softness, the edges broadened, the increase of size, with, however, a decided reduction in the specific gravity, so that the whole organ floats when placed in water.

Symptoms.—Outside the physical condition, determined by palpation and percussion and the causative disease or state, there are no distinctive symptoms. There is no jaundice, and the bile-forming function of the liver seems little interfered with, though the stools are pale. There is no obstruction to the portal circulation and, therefore, no abdominal dropsy. Percussion recognizes *enlargement of the liver*, which is, however, moderate compared with that of amyloid liver and cancer, extending, as it does, but a short distance below the normal site, where its edge can be felt

even through abdominal walls of some thickness. There is no enlargement of the spleen.

Diagnosis.—It becomes necessary to differentiate the enlarged fatty liver from the amyloid liver, which is harder and larger and associated with enlarged spleen and albuminuria. With the hyperæmic enlargement of the first stage of cirrhosis it is not likely to be confounded. Such enlargement would be trifling accompanied by tenderness, and sooner or later succeeded by contraction, while the fatty liver keeps on enlarging. From the enlargement due to the cloudy swelling of the infectious diseases, typhoid and typhus, it is distinguished by the absence of fever and the other symptoms of these diseases.

Prognosis.—This depends upon that of the causative disease. The liver of fatty infiltration can be completely restored to its natural condition with the removal of the cause.

Treatment.—This treatment is also that of the causing condition.

Fatty Metamorphosis.

Definition.—This is a much more serious condition, in which the cell-protoplasm is directly converted into fat, or rather, perhaps, into a number of products, of which fat is one, and the cell undergoes disintegration. It is the result of the operation of some poison, which has its type in phosphorus poisoning and the cause, whatever it may be, of the disease known as acute yellow atrophy of the liver.

Morbid Anatomy.—The liver, instead of enlarging, undergoes rapid reduction in size, or at least, if there is enlargement, it is of such short duration that it is never recognized. The appearance and condition of the liver, to be described under acute yellow atrophy, are those of the liver which is the seat of rapidly fatty metamorphosis.

Symptoms.—They are those of the diseases causing it, which will be described under acute yellow atrophy.

The **prognosis** is fatal and **treatment** is unavailing.

THE AMYLOID LIVER.

Synonyms.—*Lardaceous Liver; Waxy Liver; Albuminoid Liver.*

Definition.—In the amyloid liver there is an infiltration in various degrees of all the tissues of the organ by the so-called amyloid substance. The blood-vessel walls are the first affected, and by preference those of the intermediate area of the lobule, *i. e.*, that supplied by the hepatic artery, then the central or hepatic vein zone, and finally the peripheral or portal zone. The infiltration begins in the smaller arteries, then invades the cells and capillaries, and in the extreme cases pervades everything, including connective tissue.

Etiology.—The most usual cause of amyloid liver is prolonged suppuration, especially in connection with tubercular disease of the bones. Hence it is found in children who have had hip disease. For the same reason it is found associated, though less frequently than might be expected, with prolonged tuberculosis of the lungs. Syphilis is one

of the recognized causes, whence it may arise as a tertiary manifestion or as the result of bone disease incident to it. Rickets also produces some cases, and it is also associated, though rarely, with leukæmia, the cancerous cachexia, and the infectious diseases, the etiological relation of which is not, however, established.

Morbid Anatomy.—The liver is much enlarged, reaching sometimes enormous dimensions, scarcely exceeded by the largest cancers. Its appearance is waxy or bacony, especially in thin sections. This appearance is partly due also to the anæmic state of the blood-vessels, whose lumen is encroached upon by the infiltrated walls. The amyloid parts strike a mahogany red color with weak solutions of iodine. In addition to the change in size and its translucency, the amyloid liver is hard and smooth, its border usually rounded, though not always, and its fissure exaggerated. In certain syphilitic forms its surface is beset with nodules. Instead of being general, the amyloid change is sometimes circumscribed, when it may be associated with red atrophy. It is sometimes associated with fatty infiltration.

Symptoms.—Beyond the *enlargement*, which is usually manifest, the organ extending sometimes as low as the umbilicus, and the symptoms of its causing state, there are none peculiar to the amyloid liver. There is no pain unless it be the result of an associated syphilitic hepatitis, but there may be a *dragging sensation*, induced by the weight of the organ. There is no jaundice, though the stools may be light-hued, because the secretion of bile is diminished. There is no ascites, except in extreme cases, when it is a consequence of the general hydræmia and not of obstruction in the portal circulation. It is usually associated with amyloid spleen, which is enlarged, and with the amyloid kidney, which secretes albuminous urine.

Diagnosis.—This is usually easy. The large, smooth, hard organ, the history of the presence of the causing disease, the absence of jaundice and of dropsy, the association of enlarged spleen and albuminuria, admit of scarcely any other interpretation. It is to be remembered, however, that amyloid spleen is not invariably present, and, when present, may be overshadowed and compressed by the large liver. The enlarged liver of leukæmia, the result of white-cell infiltration, is not likely to be confounded, because the other symptoms of this affection are so evident. The nodular amyloid spleen, due to syphilis, must be remembered as a possibility, and will be referred to again in considering the diagnosis of cancer of the liver.

Prognosis and Treatment.—They are those of the causing disease. I have never seen an amyloid liver reduced to the normal size, yet the absence of symptoms growing out of moderate degrees of it makes practical recovery not impossible.

CIRRHOSIS OF THE LIVER.

SYNONYMS.—*Chronic Interstitial Hepatitis; Gin Liver; Granular Liver; Hob-nail Liver.*

SPECIAL VARIETY.—*Hypertrophic Cirrhosis.*

Definition.—Cirrhosis of the liver is a disease characterized by an overgrowth of the interstitial connective tissue with destruction of the parenchyma of the organ, commonly attended by a reduction in size and increase in consistence.

Etiology and Pathology.—Alcoholism is the commonly recognized cause of cirrhosis of the liver, though by no means all alcoholics, even the most confirmed, have cirrhosis. Indeed, a large number of drunkards, watched to their death and examined with special reference to this subject, have been found to have normal livers at the autopsy. Hence, some authors, notably Anstie, and later Henry F. Formad, were disposed to deny that the abuse of alcohol ever produces cirrhosis. The large, fatty liver is probably as frequent a consequence of alcoholism as is cirrhosis. Even W. H. Dickinson's observations, which were made with the definite purpose of settling the question, were not as conclusive as might have been expected. Thus he noted, in 149 autopsies upon people connected with the liquor traffic, 22, or only seven per cent., had cirrhosis; while out of 149 otherwise engaged, eight, or five and one-half per cent., were thus affected. R. Palmer Howard's * studies, noted below, seem to reaffirm the long-acknowledged dictum.

Long-continued malarial intoxication and congenital syphilis are considered causes of cirrhosis. Syphilis produces, however, quite a special form of interstitial hepatitis. In his able study of that very interesting class of cases—cirrhosis in children—the late Dr. Howard, of Montreal, found 11 per cent. due to syphilis, chiefly hereditary, while alcohol was still responsible in 15.8 per cent., even in children. Passive congestion, due to heart disease or pulmonary obstruction, causes some cases. Other causes mentioned are stimulating diet and irritation of the gall ducts by such agencies as obstructing calculus. Finally, a certain number of cases of cirrhosis are altogether inexplicable.

It has heretofore been thought that most of the causes act through the blood of the portal vein, irritating the connective tissue of Glisson's capsule, which accompanies everywhere the branches of that vessel, causing first a hyperplasia of connective tissue cells. Thus the first stage of the disease would be one of enlargement, accompanied often by tenderness. That subsequently this embryonic connective tissue undergoes organization and contraction, gradually compressing the cells within its grasp and ultimately destroying immense numbers of them. That the reduction in size goes *pari passu* with a hardening of the organ, which is a conspicuous feature of advanced degrees of the disease. Such, at least, has been the commonly received theory of the formation of the cirrhotic contracted liver. But while cases are met with representing both ends, so to speak, of the process, the initial stage of enlargement and tender-

* R. Palmer Howard, Transactions of the Association of American Physicians, Vol. II, 1887.

ness and the terminal one of smallness and hardness, few can attest that they have had the opportunity of tracing the one stage into the other in the same patient, though the celebrated Dr. Bright, as far back as 1827, claimed to have traced cirrhosis from the incipient enlargement to the smallness of the later stage.

Much more reasonable appears Weigert's conclusion, based on experiment, that the death of the cells is primary and the overgrowth of connective tissue secondary. Acknowledging that the majority of causes which produce the disease, such as alcohol, for example, operate through the portal circulation, it is only natural that the cells whose business it is to eliminate the poison should receive the first sting and perish in the attempt, and that their place should be supplied by a reactive overgrowth of connective tissue, as Weigert has shown by experiment. We may also admit a reactive contracting effect of the new connective tissue on remaining cells, producing thus the death of a greater number.

Though occurring often in children, it is still a comparatively rare disease among them, being rarely met before the age of thirty-five. It is also a disease of men rather than women.

Morbid Anatomy.—At least two well-defined varieties of interstitial hepatitis are met, known as atrophic and hypertrophic cirrhosis. (*a*) Of *Atrophic Cirrhosis.*—In addition to the hardness and reduced size of the liver, which may fall from its normal weight of four or five pounds (1.8 to 2.2 kilograms) to two pounds (0.9 kilogram) and less, the surface of the organ is rough and uneven. In the formation of these inequalities, circlets of parenchyma are destroyed and replaced by connective tissue, within which the parenchyma remains intact and appears raised. According as the elevations vary in size, the liver is described as a *granular* liver, a *hob-nail* liver, or a *lobular* liver. If the cells are fatty, as is sometimes the case, they are yellow; at others they are natural in hue; at others paler. It was on the color of the nodules that Laennec based the name of *cirrhosis* from the Greek *κιρρος*, reddish-yellow, applied by him. In some instances the cirrhotic liver is quite smooth, showing a uniform distribution of the connective tissue through the parenchyma of the organ, appreciable only in thin sections examined by microscope. As the process extends, it involves branches of the portal vein itself in its destruction, and even bile ducts are obliterated. Amyloid and fatty infiltration may be associated with cirrhosis. The new connective tissue, on the other hand, is richly supplied with blood-vessels from the hepatic artery, and Rindfleisch has suggested that the bile is secreted from this blood rather than that of the portal vein. The atrophic liver is very commonly associated with fatty infiltration, which enlarges the liver to a degree which may outbalance the contraction.

(*b*) *Hypertrophic Cirrhosis.—Elephantiasis of the Liver.*—We owe our knowledge of this form chiefly to the French clinicians, headed by Charcot and Henoch. The liver is enlarged, and it is not unlikely that what has been characterized as the first stage of atrophic cirrhosis has sometimes been represented by this form of disease. An important difference between the two forms is that, while in both there is an overgrowth of connective tissue, in hypertrophic cirrhosis the new-formed tissue exhibits little disposition to contraction. Nor is there any com-

pression of the branches of the portal vein. On the other hand, there is obstruction of the biliary channels, producing the jaundice which is so characteristic a symptom, whence the French investigators start the disease as an inflammation of these passages,—a cholangitis,—and call it *cirrhose hypertrophique avec ictère*. It is claimed also by Henoch that there is a new formation of biliary capillaries. Others hold that this absence of contraction in the connective tissue is exaggerated; that while it is much less marked than in atrophic cirrhosis, it does occur sooner or later if the patient lives long enough. It is further characteristic of the development of connective tissue in hypertrophic cirrhosis that it is more active *within* the lobules.

However this may be, the liver, thus enlarged, may weigh eight or ten pounds (16 to 20 kilograms). Its color is greenish-yellow or green.

Biliary Cirrhosis.—This term is considered by some as synonymous with hypertrophic cirrhosis. But the French clinicians also describe a liver of increased size, in which the enlargement is ascribed to an overgrowth of the interstitial connective tissue; an overgrowth, moreover, which is to replace gaps in the parenchyma due to the destruction of cells which have perished on account of the noxious influence of bile retained in the small and medium-sized bile ducts. This is followed by a deposit of pigment granules in the interlobular connective tissue and within the acini themselves. It is, therefore, "secondary" to the obstruction of the gall ducts by any prolonged cause, as a gall-stone, tumor, or the like. In such case the liver is larger and harder. The above reasoning seems to be sustained by the experiment, since ligature of the common bile duct in animals has been followed by such cirrhosis.

Symptoms.—(*a*) *Of Atrophic Cirrhosis.*—It must be admitted that cirrhosis of the liver sometimes fails to give rise to any symptoms. The early subjective symptoms of this affection are rather the result of secondary conditions caused by it. Among these are, preëminently, those of *chronic gastric catarrh*, *anorexia*, *nausea*, *distention*, and *discomfort*. The gastric catarrh is the result of a chronic passive hyperæmia, due to the obstruction of the movement of the portal blood through the liver. As a consequence of the hyperæmia, the mucous membrane of the stomach is more or less constantly covered with mucus, which excites the nausea and prevents the secretion of gastric juice. A similar state of affairs exists in the small intestine, causing constipation, which is increased by the defective biliary secretion. This is further shown by the paleness of the stools. The well-known comforting effect of the early morning dram upon the inebriate may be due to some action of the alcohol upon this mucus.

The remaining symptoms are also mainly the result of the ligature-like effect of the connective tissue on the portal vessels. *Nasal hemorrhage*, often very obstinate, is one of these. So are *gastric* and *intestinal* and more rarely *œsophageal hemorrhages*, the former often enormous and alarming, but really beneficial, by removing the gastro-intestinal congestion. Either one of these forms of hemorrhage may be the very first symptom to attract attention. Similarly caused is the *abdominal dropsy*, which is often enormous. Four gallons (20 liters) are not infrequently removed at one tapping, and sometimes the fluid, from its weight, bursts

through the feeble barrier at the abdominal ring, distending the tunica vaginalis with the dropsical fluid. The navel is often pushed out by the enormous distention.

The surface of the upper abdomen and lower thorax, anteriorly, is marked by over-distended veins. This is directly due to the backing of the blood into these veins, rendered possible by the anastomotic communication between the portal and caval circulations. Such anastomosis between the rudimentary veins in the round ligament (branches of the portal vein) and the epigastric and mammary veins leads to enlargement of the superficial branches of the latter and in extreme cases to the formation of a *caput Medusæ* about the navel. Communication between the superior hemorrhoidal vein (a branch of the portal vein) and the middle and inferior hemorrhoidal, and through them with the hypogastric veins and vena cava, produces *hemorrhoids*, a characteristic symptom of cirrhosis. Anastomosis between the superior gastric vein (a branch of the portal) and the inferior œsophageal, whose blood goes to the cava through the azygos and hemiazygos, causes a varicose condition of the veins of the lower end of the œsophagus which has resulted in fatal hemorrhage. The over-filling of the œsophageal and azygos veins also obstructs the movement of the blood through the intercostal and pleural vessels of the right side, causing right-sided hydrothorax. These dilatations, which have been characterized as "attempts at compensation," are to be distinguished from the more diffuse dilatations of the abdominal veins seen in the flanks, which are due to the pressure of a large abdominal dropsy on the cava, preventing the return of the blood of the lower extremities to these veins. *Œdema of the legs* is, however, much more uncommon than abdominal dropsy, and when present depends upon the further pressure exercised by the enormous accumulation of fluid in the abdominal sac upon the returning blood of the lower extremities.

Jaundice is a rare symptom in atrophic cirrhosis, though the constricting effect of the interstitial tissue upon the gall ducts would lead us to expect it to be not uncommon. It is probably because comparatively little bile is secreted by the compressed cells and none at all by the destroyed ones. Fagge * reports 34 cases of jaundice out of 130 examined in the post-mortem room of Guy's Hospital, rather more than might be expected. A *sallowness of complexion* is also sometimes present, while a ruddiness of face is not uncommon.

Physical examination by palpation and percussion discovers a diminished area of hepatic dulness. On the other hand, splenic dulness is often enlarged, the latter because of obstructed return of its blood through the liver. According to Frerichs, the spleen is enlarged in about one-half of the cases. It is often impossible to outline either of these organs because of the extreme abdominal distention, and tapping must first be resorted to before physical exploration is satisfactory.

The *urine* in atrophic cirrhosis of the liver is generally scanty, of high specific gravity, high-colored, and often loaded with biliary coloring-matters and urates, the latter subsiding on standing, forming a bulky

* "Practice of Medicine," 1886, Vol. II, p. 306.

sediment. The proportion of urea is often diminished, a natural result of the deranged function of the liver, to which modern physiology assigns an important rôle in urea formation.

Drowsiness and coma and even delirium are sometimes terminal symptoms, especially in cases where there is jaundice, but also where there is ascites without jaundice. They are ascribed to cholesteræmia.

(*b*) *Symptoms of Hypertrophic Cirrhosis.*—The symptoms which distinguish this form from the atrophic variety are—

(1) The jaundice, which begins with the first vague symptoms of the disease and gradually deepens as the disease progresses. The explanations suggested of this feature of the disease, as contrasted with its absence in atrophic cirrhosis, cannot be said to be satisfactory. It is simply true that in some way there is produced obstruction in the biliary vessels, perhaps by a cholangitis.

(2) The absence of hyperæmia of the stomach and bowels, of hemorrhoids, enlargement of the spleen, and preëminently of ascites; or the presence at least of only mild degrees of these symptoms.

(3) The presence of tenderness in the liver, in addition to its evident enlargement and smoothness.

(4) Certain differences in the urine in the two forms.

It is a well-recognized fact that where there is jaundice the urine is also jaundiced. In *atrophic* cirrhosis jaundice is exceedingly rare, and when present, say in about one-sixth the cases, it is very slight. The same is true of the urine, for while the latter is scanty and high-colored, it rarely contains bile pigment. In *hypertrophic* cirrhosis, on the other hand, bile-stained urine is very common. Blood is never found in the urine, while in atrophic cirrhosis it sometimes is, as is also albumin in advanced stages. In atrophic cirrhosis the urea is diminished, in hypertrophic it is normal. In hypertrophic cirrhosis the fæces are sometimes devoid of bile, sometimes not.

Rosenstein has made a study of the blood in hypertrophic cirrhosis, and has found the red corpuscles diminished one-half and the leucocytes relatively increased. He also found it to coincide in certain cases with the hemorrhagic diathesis. Alcohol is even a more important factor in causing hypertrophic cirrhosis than atrophic.

The course of hypertrophic cirrhosis is usually more rapid than that of the atrophic. It may be put down at one or two years. Yet in some cases it is very short. Osler mentions a case which proved fatal in ten days, another in three weeks. It may be questioned whether these were not cases of acute yellow atrophy. All cases terminate more or less acutely. Delirium sets in, the tongue becomes dry, the pulse rapid, the temperature rises to 102° F. and 104° F. (38.9° C. and 40° C.).

Diagnosis.—(*a*) *Of Atrophic Cirrhosis.*—The diagnosis of cirrhosis of the liver is not usually difficult. If one is satisfied that there is a reduction in the size of the organ, and there are associated with this no symptoms of acute disease and no history of starvation, we may infer scarcely anything else but cirrhosis. And if to this is added ascites, without dropsy elsewhere, the diagnosis is absolute.

Tubercular peritonitis, with its liquid effusion, has been mistaken for cirrhosis, and the wasting which attends advanced stages of the for-

mer affection closely resembles that in the latter, but the abdominal tenderness in peritonitis is characteristic, there is fever, and the effusion is never very large.

(*b*) *Of Hypertrophic Cirrhosis.*—Hypertrophic cirrhosis is to be distinguished from cancer of the liver, amyloid liver, multilocular echinococcus disease, and the liver of obstructive jaundice or biliary cirrhosis. In cancer there is no splenic enlargement, ascites is more frequent, the liver is more uneven, and the patient is older, while in hypertrophic cirrhosis we have also the history of alcoholism.

In amyloid liver there is also splenic enlargement, but there is no jaundice, and we have the etiological history peculiar to amyloid disease.

Multilocular hydatid disease in the liver presents almost identical symptoms, including splenic tumor, but in addition there are the nodules on its surface which soften with time.

The liver associated with chronic biliary obstruction, secondary cirrhosis, while somewhat enlarged, is not nearly so much so as in hypertrophic cirrhosis. It is also hard and accompanied by marked jaundice, as well as by evidence of hepatic obstruction, or causes leading to obstruction. Its course, while slow, is more rapid as a rule than that of hypertrophic cirrhosis, while the liver also, after a while, diminishes in size.

Prognosis.—The prognosis of cirrhosis of the liver is unfavorable if restoration of the normal organ be the object. A liver once the seat of interstitial hepatitis can probably never resume its normal histology. Yet the liver has a good deal of elasticity of function, and if the cause of the condition, supposing it to be alcoholism, is removed and the contraction be not too far advanced, the patient may be restored to comparative health. Generally, however, the course of cirrhosis is from bad to worse, although it may be a slow course, and the patient finally dies of exhaustion.

It only rarely happens that death is caused by the copious hemorrhages from the stomach and bowels which sometimes occur. On the other hand, they are more frequently a relief to the portal congestion, giving to the patient thus a new lease of life. He may live many years in comparative comfort.

Treatment.—The treatment of cirrhosis of the liver resolves itself into two parts—first, the relief of the symptoms, and, second, the restoration of the organ to its normal state.

Toward the first result the removal of the cause is indispensable. The alcoholic must stop drinking. This, after some temporary inconvenience, of itself brings alleviation. But the effect of gastric congestion remains in part, and sufficiently to cause want of appetite, nausea, unpleasant taste in the mouth, and a general disgust of one's self and everyone else. The mucous membrane of the stomach is swollen and probably bathed with mucus. The latter can be removed by free drinking of alkaline mineral waters before meals, such as those of Vichy, Vals, and Carlsbad, the effect of all of which is increased when hot. Here, too, as in gastric catarrh—it is really gastric catarrh we are treating—the hot-water treatment is often highly useful by ridding the stomach of mucus. A full tumbler, as hot as it can be borne, is taken slowly before breakfast or before each meal. Its effect is often highly beneficial. I know no additional ex-

planation of its action unless it be that it may also stimulate the secretion of gastric juice.

But the congestion which is responsible for this secretion must also be removed. This is best done by the saline and mercurial purgatives. Two to five grains of blue mass at bedtime, followed by a dose of sulphate of magnesium in the morning or of Hunyadi or Friedrichshalle water, will deplete the engorged veins and relieve the symptoms for the time being. The mineral waters of Saratoga in this country, some of which are also purgative, are very useful for the same purpose. A course at Saratoga is greatly appreciated by the confirmed free drinker and he is always better for some time after it.

Finally, foods which make the least demand upon the stomach are to be used. Fatty matters are especially contra-indicated. In advanced stages, milk and Vichy, peptonized milk, and beef peptonoids may be assimilated when other foods cannot be managed by the feeble digestion, but even these are absorbed with difficulty as long as the mucous membrane of the bowels is so much congested.

The abdominal effusion is combated by the purgatives alluded to, and diuretics may be added, and of these the acetate of potassium seems more efficient than the bicarbonates and citrates where dropsy is due to hepatic affections. Perhaps this is because in large doses it has also some laxative effect. When the abdominal effusion becomes large it must be removed by tapping, although the re-accumulation may be very rapid and it may have to be repeated many times.

Can anything be done to remove the growth of the connective tissue and promote the re-development of the destroyed parenchyma? Presumably, if the former could be accomplished the latter may take place, for there is evidence to show that the liver structure may be reproduced. Theoretically iodide of potassium is a remedy which should melt away the overgrown connective tissue. Practically, it is extremely doubtful whether it does. I have never seen such effect, nor can I point to any reliable observations that affirm it. There may be, however, and to such an end it is right to use the drug in small doses, which, to produce any effect, should be long continued. There is reason to believe that the iodide is more efficient when taken freely diluted and on an empty stomach than in larger doses after meals. Thus administered, three to ten grains (0.2 to 0.66 gm.) may be regarded as a sufficient dose.

SUPPURATIVE HEPATITIS.

SYNONYM.—*Abscess of the Liver.*

Etiology.—The vast majority of abscesses of the liver, some would say all of them, are traceable to causes which, in one way or other, are associated with microbic origin. Even traumatic abscess, which is of admitted occurrence, is ascribed to an associated infectious agent. The possibility of abscess excited by simple chemical, as contrasted with bacterial, cause should be at least mentioned. Most abscesses of the liver arise by infection from the portal area. These are either thrombotic, embolic, or amœbic. The thrombotic are caused by infectious throm-

bus, which, starting in the venules of an area drained by the portal vein, extends thence to the branches of the portal vein in the liver, where it starts a suppurative pylephlebitis. Such an area is the colon, which is the seat of dysentery, the rectum by its hemorrhoidal veins, or the neck of the bladder. More frequently a fragment of such a thrombus lodges in a branch of the portal vein and starts an abscess, constituting the embolic origin. Or the *amœba coli*, which is the cause of amœbic dysentery, is transferred from its primary state in the intestine into the liver. A similar mode of origin of abscess is by an umbilical phlebitis in the new-born infant.

Abscesses of the liver may also arise from emboli arising in the left heart, the pulmonic or systemic circulation, reaching the liver *viâ* the hepatic artery. These mostly originate in the lungs or left heart, but may arise beyond, the condition being that they are small enough to pass through the capillaries of the pulmonary artery. Such would be the abscesses caused by injuries to the scalp, or to bones of the skull, or from seats of osteomyelitis anywhere, all of which are acknowledged to be rare causes of abscess of the liver. Septic emboli, producing abscess of the liver, may arise from the left heart in cases of mycotic endocarditis. These are among the rare causes of abscess of the liver. Even a non-infectious embolus may excite an abscess if brought into association with pyogenic organisms entering the liver in another way. Such organisms may enter the liver through the common duct from the alimentary canal. This is probably the route of the organism causing suppurative cholangitis, and of that causing the abscess often associated with hydatid cyst of the liver. Finally, regurgitant embolism of the hepatic vein is a possible cause of hepatic abscess. In the vast majority of cases, however, abscess of the liver is preceded by dysentery, whence arises an infectious thrombus, embolus, or an amœba coli.

Morbid Anatomy.—The right lobe of the liver in its thickest part is the most frequent seat of abscess—in two-thirds of all cases. The abscess varies in size from that of a mere point to that of a child's head, the whole right lobe being sometimes converted into one abscess cavity. It may be single or multiple. Rarely, the abscesses intercommunicate. The liver is, of course, proportionately enlarged. Notwithstanding this, the external appearance of the organ may not be changed. On the other hand, if the abscess is near the surface there may be a prominence under which fluctuation may be recognized, or the liver may become adherent to the abdominal wall or adjacent viscera. The abscess cavity, if of any size, is usually ragged, and not sharply defined from the surrounding hyperæmic liver tissue. Such hyperæmia may involve two or three rows of acini. In chronic cases, however, there may be a tolerably firm pyogenic membrane.

The contents of the abscess may be pus, or a puriform fluid consisting of the granular débris of cells, oil drops, a few leucocytes, cholesterin and other fat crystals, and numerous crystals of bilirubin. The amœba coli has been found among the contents of the abscess, but recognizable liver cells are rarely found. Rarely the pus may become inspissated, caseous, or even calcified or cystic. Should the abscess accompany hydatid disease, echinococcus hooklets may be found. The contents of

these abscesses is generally a true pus. Any form of abscess may perforate the diaphragm and lung, producing interstitial emphysema; or the pus, with echinococcus hooklets, may be expectorated; or the abscess may burrow into the peritoneum, setting up fatal peritonitis, or into the pericardium, causing fatal pericarditis; into any adjacent hollow organs or into the abdominal wall, discharging externally by fistulous openings.

The thrombotic and embolic forms of abscess always begin as a phlebitis, which rapidly invades the adjacent tissue. Contrary to what is usual in embolism elsewhere, the lodgment of an embolus in the liver is not followed by hemorrhagic infarct.

Symptoms.—There may be latent liver abscess, even when the abscess is of considerable size, though this is a very rare event. Abscess of the liver is generally associated with *pain* in the hepatic region, with *fever*, very often with *chills*, *sweats*, and sometimes with *jaundice*. The pain is almost invariably accompanied with *tenderness*. It may be deep or superficial, and in the latter event it may be sharp and cutting, because involving the peritoneum. The characteristic *shoulder pain* of hepatic disease may also be present.

Fever is, perhaps, the most invariable symptom, and in no other affection of the liver does it rise so high. Indeed, except acute yellow atrophy and the so-called hepatic fever, there are no diseases of the liver associated with fever. In the former it is of comparatively short duration, and in the latter it is moderate. The temperature reached in abscess is very high, 104° to 105° F. (40° to 40.5° C.), and may be preceded by chills of corresponding severity, while the fever in turn is succeeded by sweats, profuse and exhausting. Jaundice is not usually marked, but varies somewhat in intensity.

Physical examination easily recognizes an enlargement of the organ upward in the mammary and mid-axillary regions rather than downward, as is usual with other diseases of the liver. Yet the liver is by no means always enlarged, even if there be multiple abscess. The enlargement is due not merely to the presence of pus, but is also contributed to by the hyperæmia and the swelling of cells. The lung being thus encroached upon, the movement of the liver consequent on respiration is less marked than in health. The hepatic region is at first hard to palpation, but ultimately fluctuation may be recognized, while a doughy or œdematous condition of the abdominal wall is sometimes present and quite characteristic.

Diagnosis.—This may be difficult at first, but as time passes doubts clear up. Intermittent fever very naturally is first thought of in many instances, but it will not be long before this disease can be eliminated. There is no enlargement of the spleen, no history of malarial exposure, no malarial organism is found in the blood, and, above all, antiperiodic therapeutics, so efficient in malarial disease, fails of its purpose. In the absence of malaria, and in the presence of the causes usually responsible for abscess of the liver, there is little else left to mistake for it. A pleuritic effusion on the right side gives on percussion dulness in the same locality, but along with this are the diminished fremitus and diminished vocal resonance characteristic of fluid in the pleural sac, while there may also be the bronchial breathing peculiar to compressed lung. The dul-

ness to percussion of the pleuritic effusion maintains itself posteriorly below the angle of the scapula, while that of abscess of the liver fades away as the angle of the scapula is reached. A suppurating echinococcus cyst may give rise to similar symptoms, and, in view of its rarity in this country, is scarcely likely to be recognized until aspiration discovers the elements characteristic of it. The needle should be early tried if abscess be suspected, yet it is evident that in so large an organ an abscess of moderate size may easily elude it.

Hepatic intermittent fever, due to chronically impacted calculus, resembles abscess by the fever, chills, and sweats, and in tenderness over the liver, but the history of hepatic colic is present, jaundice is more marked and obstinate, and the condition is evidently not so serious.

Prognosis.—This is generally unfavorable. Even in cases where the abscess happens to point to the surface and is properly opened, death usually supervenes after long and tedious illness, say in six weeks to three months, and, where surgical interference is not possible, death is even more speedy. Cases do, however, recover, not so much by the aid of the physician as through nature's irresistible tendency. It is said that with surgical interference 30 per cent. recover, and where this is impossible 20 per cent. still survive, but this has not been my experience. The hydatid abscess is more apt to terminate favorably if opened than is the infectious abscess.

Treatment.—This is palliative and supporting, except in those cases where surgical interference is possible. The usual measures to relieve pain, nourishing and easily assimilable foods, quinine, iron, and stimulants are indicated.

ACUTE YELLOW ATROPHY OF THE LIVER.

SYNONYMS.—*Icterus gravis; Acute Parenchymatous Hepatitis; Malignant Jaundice.*

Etiology.—This remarkable and fortunately rare disease is probably due to the action of some virulent poison, autogenetic perhaps, but whose nature is as yet undiscovered. Pregnancy is one of the conditions acknowledged to produce it, and more cases occur among women than men. It occurs in the second half of pregnancy. It has occurred in the course of the infectious diseases, and the usual microbic origin has been held responsible for it, as have been alcoholism and mental excitement. Bacteria have been found in the organ after death. Beyond this we know nothing of its cause.

Pathology and Morbid Anatomy.—The destructive process in the liver is almost identical with that of phosphorus poisoning, and consists essentially in a very rapid destruction of liver cells. Opinions are divided as to whether this is the result of an acute inflammatory process, or whether the cells are destroyed by some solvent action. Frerichs and Demme held the view that it is an acute parenchymatous inflammation, of which the chief seat is the peripheral zone of the lobule, whose swelling causes obstruction in the biliary capillaries, and the reabsorption of bile. Henoch and v. Dusch consider retention of bile the starting

point, and that the liver cells are dissolved by this retained bile. Munk regarded all cases as the result of phosphorus-poisoning.

The liver, at necropsy, is found very much reduced in size, often to half and even quarter its normal volume. This may take place in three or four days, and even less. A stage of primary enlargement is said to be sometimes present, but is never seen at autopsy. The organ is flattened, flabby, and can be folded over on itself, and the usual lobular markings are either very indistinct or altogether absent. The capsule is loose and wrinkled, and the organ of a dirty yellow color.

On section, the surface is either uniformly yellow, or it exhibits an alternation of yellow and red. The yellow appears, for the most part, in islets, which are surrounded by the red. The yellow represents an earlier stage of the disease. It is soft and spongy, and rises cushion-like above the surface. The red is tougher, more leathery, and sinks below the level of the cut surface. When the organ is uniformly yellow, this later stage, represented by the red, has not been reached before death.

Histologically, the yellow areas exhibit softening and apparent solution of the cell network, very few liver cells remaining which retain their own contour. Instead, are found disintegrating cells with fat drops of all sizes, the cells being in places still united by their connecting substance, so as to maintain the original network. Sometimes crystals of bilirubin, leucin, and tyrosin are met with. The red areas consist of a loose connective tissue, whose meshes contain fat drops and biliary coloring-matter, representing the softened liver parenchyma bereft of its cells. In places there may be seen a slight degree of cell infiltration of the interstitial tissue, in others irregular branching bands and apparently blind-ending tubes of cells, resembling biliary epithelium. These Waldeyer says are the result of an attempt at repair. The atrophy usually takes place more rapidly in the left lobe.

The skin and organs generally are intensely bile-stained. There may be small extravasations of blood in various parts. The spleen is enlarged and hyperplastic, the renal epithelium and heart-muscle are fatty, while the serous cavities contain more than the normal amount of fluid.

Symptoms.—There are no symptoms distinctive of the beginning of acute yellow atrophy. For several days there may be signs of gastro-intestinal catarrh, promptly followed by jaundice. The former include *headache*, *malaise*, *loss of appetite*, *nausea*, *vomiting*, *eructations*, *epigastric discomfort*. Then there supervene suddenly serious symptoms—*delirium*, *abdominal pain*, *convulsions*, local or general, *drowsiness*, *coma*. Sometimes the symptoms of this stage are delayed, in extreme cases as much as three weeks.

The liver rapidly diminishes in size. Three or four days may see its disappearance to percussion and palpation, favored by further obscuration by distended air-holding viscera. W. v. Leube calls attention to a symptom elicited by palpation which he thinks may be of diagnostic value—a more or less permanent "*pitting*" *to pressure in the epigastric region*. He ascribes this to an impression made upon the relaxed liver, to which the abdominal wall fits itself. The *spleen*, on the other hand, is enlarged, the jaundice is intense, the vomiting obstinate, while there may be epistaxis, hæmatemesis, hæmaturia, menorrhagia, and hemorrhagic

extravasations, while the stools are devoid of bile. The pregnant woman aborts. There is little fever, and in the worst stage there is but moderate rise of temperature, rarely above 104° F. (40° C.). The pulse, at first infrequent, increases toward the end to 120 or more.

The *changes in the urine* are very characteristic and have been thoroughly studied. It is deeply bile-stained, is concentrated, the specific gravity often reaching 1030. It is slightly albuminous and may contain the bile acids, bile-stained fatty casts, and bile-stained renal epithelium. The urea is greatly lowered, even totally absent. The characteristic feature is the presence of leucin spheres and tyrosin needles in most cases. These crystals may appear without treatment of the urine or they may come down after slight concentration. In addition are found also aromatic oxyacids, especially oxymandel acid, all representing products of albumin disintegration.

Diagnosis.—The symptoms of acute yellow atrophy in the first stage do not admit of a diagnosis. This is the more true because there is no symptom, even atrophy, which may not be wanting. Thus cases have perished from hemorrhage before the disease was recognized. This is true even of jaundice in the rapidly-terminating cases. In the second stage, on the other hand, the symptoms are so distinctive that it seems almost impossible for one familiar with them to fail to recognize them. It is, however, such a rare disease in this country that the opportunity does not often present itself. Hence it is sometimes overlooked because not suspected, the more excusably because grave nervous symptoms may occur even in catarrhal jaundice and in the infectious diseases, as, for example, in pneumonia, where jaundice is sometimes a symptom. Acute phosphorus-poisoning so closely resembles acute yellow atrophy that the diagnosis depends largely upon the possible recognition of the cause. There are, however, some differences. The reduction in size of the liver is not as rapid, the nervous symptoms are not as grave, and leucin and tyrosin are not usually found in the urine of phosphorus-poisoning. Hypertrophic cirrhosis also sometimes resembles acute yellow atrophy clinically, but the enlarged liver is the distinctive feature of the former.

Prognosis.—This is so unfavorable that recovery may be said to imply an error of diagnosis.

Treatment.—There is no curative treatment. Symptoms should be relieved by the usual palliatives. Headache should be treated by phenacetin and acetanilide, rather than morphine. An ice-bag may give great relief.

MORBID GROWTHS OF THE LIVER.

The only morbid growths of the liver which are of clinical importance are cancer and sarcoma. An angeioma is an interesting new formation of small size which presents no recognizable symptoms before death. It is composed of vascular tissue and is distinctly capsulated. The large sizes may be as large as a walnut, more rarely still larger. Some pathologists describe an adenoma which others class among the cancers as a trabecular variety. Myoma is another form of histioid tumor rarely found in the liver.

Carcinoma of the Liver.

Etiology.—Cancer of the liver is a comparatively common disease, next in frequency after that of the uterus and stomach of internal organs. It is, moreover, in the vast majority of cases secondary, in fully three-fourths of cases, and of these two-thirds are secondary to primary cancer of the portal area, one-third to primary cancer elsewhere. The stomach is, naturally, the most frequent primary focus. Cancer of the liver is more common in male adults between the fortieth and sixtieth year, yet it does occur occasionally in children.

Morbid Anatomy.—There are two chief forms in which cancer of the liver presents itself, the nodular and massive. Rare forms are radiating, colloid, and cancer with cirrhosis.

(1) In the *nodular form* nodules of various sizes are scattered throughout the organ. The nodules vary in diameter from one-fifth of an inch to two inches (0.5 cm. to five cm.). They are usually opaque, white, or yellowish-white, and may be very numerous. The superficial nodules project above the surface, and may even be felt through the abdominal wall in the emaciated subject, giving rise to the oft-described "bosselated" feel. These superficial nodules are often umbilicated, because of the disintegration and absorption of the older central cells, leaving a residue of connective tissue and partially obliterated blood-vessels. The umbilication is confined to the superficial nodules, which also received the name of Farre's tubercles. This variety of nodular cancer may be both primary and secondary. The nodules usually reach a larger size in the secondary, and are apt to be more numerous.

(2) The *massive* form, in which there is one large cancerous mass, greatly increasing the bulk of the organ. It is greyish-white in color, and may reach four or six inches (ten or 15 cm.) in diameter. This form is primary.

(3) The radiating form, usually pigmented, in which the nodules may also be multiple, but smaller and less numerous than in the nodular form. It is a form of secondary cancer.

(4) A colloid form, rare, and only secondary.

(5) A rare form is cancer with cirrhosis, in which the liver is but slightly enlarged, weighing 4.5 to 6.5 pounds (*circa* two or three kilograms), and presents a greenish-yellow appearance, studded over with small white nodules, not unlike those of the hob-nail liver, the same appearing in large numbers when the organ is cut.

All varieties of cancer are subject to degeneration, but the secondary forms more rapidly. The change is a fatty metamorphosis of the cells, associated sometimes with rupture of blood-vessels and large extravasations of blood, which may even burst into the peritoneum and gall bladder. There may be occasional suppuration around the nodule.

As to the histological origin of cancer, the primary forms start in the liver cells; they are true epitheliomata, the capillary network forming the primary stroma, to which an independent growth of stroma is subsequently added. The secondary forms are embolic in origin, chiefly through the branches of the portal vein, but possibly by the hepatic ar-

tery, with or without intermediate involvement of the lung, the first new cancer cell being an infected cell of the capillary wall, whence the parenchymal liver cells are in turn affected. The stamp of the pigmented radiating cancer is, perhaps, thus derived, and illustrates this mode of invasion. The secondary forms repeat the type of the primary varieties. The cells are mainly epithelioid, but may be polygonal, and even cylindrical. They exhibit various grades of fatty degeneration.

The liver is variously enlarged by these different forms of cancer, the maximum product being the largest produced by any disease of the liver. (See Fig. 20.)

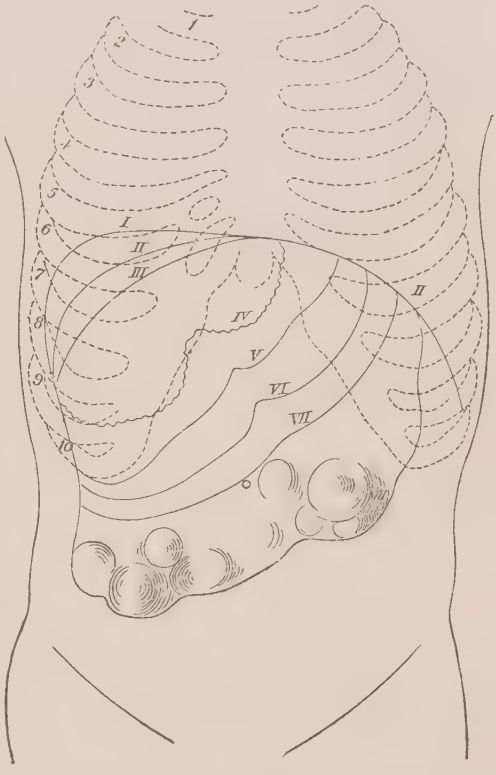


FIG. 20.—SHOWING APPROXIMATE ENLARGEMENT OF THE LIVER, CORRESPONDING TO THE DIFFERENT DISEASES DESCRIBED IN THE TEXT.

I. Position of the diaphragm to the maximum enlargement (carcinoma). *II, II.* Normal situation of the diaphragm. *II, III.* Relative dulness. *IV.* Border of the liver in cirrhosis. *V.* Border in health. *VI.* Lower border of the fatty liver. *VII.* Of the amyloid liver. *VIII.* Of cancer, leukæmia, and adenoma. (*After Rindfleisch.*)

SARCOMA.—Of the remaining morbid growths of the liver, sarcoma alone demands a few words. It is almost invariably secondary, very few cases of primary sarcoma of the liver having ever been found. Secondary sarcoma of the liver includes melano-sarcoma, lympho-sarcoma, and myxo-sarcoma. The melano-sarcoma is the most frequent and interesting. It is always secondary and usually multiple, though a diffusely infiltrated variety exists, giving the liver on section a granitic appearance. Melanotic sarcoma of the orbit often precedes it and it is sometimes a part of a general melanotic distribution over the body, including the skin. Sarcoma of the liver is said to be never associated with ascites.

Symptoms.—Very rarely cancer of the liver may be latent, except as to a vague ill health explained by the findings of the autopsy. In most instances such ill health grows worse more or less rapidly, and examination of the liver discovers *enlargement*, to which may or may not be added recognizable nodules. The enlargement may extend beyond the umbilicus, but it is not usually so great, and at times there is none whatever. To inspection the enlargement is first seen in the upper zone and produces a change

of configuration which involves commonly the whole upper abdomen. Rarely the nodules may be seen. The superficial veins are enlarged.

The other signs of ill health alluded to, apart from those of a primary cancer elsewhere, are *loss of appetite*, *nausea*, a *sense of epigastric fulness*, *pain* in the epigastric or hypochondriac region or in both simultaneously. The pain may be lancinating and extend to the right shoulder. To this *tenderness* is sooner or later added. Indeed, perhaps tenderness precedes. Emaciation may have preceded the more striking degree of these symptoms and increases rapidly, while the characteristic *cachexia* develops *pari passu*. An examination of the blood shows a reduction of hæmoglobin and corpuscles, and as the blood becomes thin *œdema* develops. In some cases there is *fever*, especially toward the end, with a temperature of 100° to 102° F. (37.8° to 38.9° C.), more or less intermittent but rarely associated with rigors.

Obstructive *jaundice* is a frequent symptom in carcinoma hepatis, it may be said in fully half the cases. It is due to compression of the smaller biliary passages, and does not usually reach a high degree. Nor are the fæces usually devoid of bile. If the latter event occurs, and the jaundice is intense, it means that some of the larger ducts are obstructed, while involvement of the gall bladder may be suspected, or the portal lymphatics. *Jaundiced urine* is about as constant as jaundice itself. The presence of melanin is said to point especially to the presence of the pigmented varieties of cancer. Albuminuria is, on the other hand, unusual.

Ascites is a rather infrequent symptom, and can only occur when the portal vein or branches become involved either by compression or invasion. Should, however, a bloody fluid be obtained by tapping, and there is a tumor of the liver, the indications are that the tumor is cancer. Enlargement of the spleen is rarely present in cancer of the liver.

The duration of the disease ranges from three to fifteen months.

Diagnosis.—This is not usually difficult if there is enlargement, but becomes quite so if there is not. Especially easy is it if the nodules can be felt, or if there is recognized primary cancer elsewhere.

The smooth, enlarged liver of cancer is distinguished from that of the more benignant conditions of fatty liver and amyloid liver by the absence in the latter of grave symptoms and jaundice. The fatty liver is softer than the liver of cancer, the amyloid is harder, more often smoother, while its rounded border can sometimes be felt. It is also accompanied by enlarged spleen. In abscess of the liver the organ may be soft or doughy in consistence, and the same may be true of the abdominal walls over it. There are also the causes of abscess of the liver and among symptoms the characteristic chills, high fever, and sweats.

Multiple echinococcus cysts may furnish similar local signs, even the "bosselated" feel, but hydatid disease is rare in temperate climes; the nodules are softer, the disease is of longer duration, and is less rapidly followed by wasting. Enlargement of the spleen is quite common in hydatid disease, present, it is said, in nine-tenths of all cases. Jaundice is even more frequent in this disease than in cancer—in four-fifths, as contrasted with a little more than half. Aspiration may aid in the solution. Of other affections attended by uneven surface of the liver, the amyloid organ beset with gummy nodules offers difficulties, but the lesser gravity,

the longer duration, and, especially, the syphilitic history solve the question. Both are associated with enlarged spleen, but the rapid enlargement of the liver in amyloid disease sometimes obscures the enlarged spleen, and even interferes with its development.

Doubt sometimes arises, in the presence of certain stubborn forms of jaundice, as to whether they may not have cancer at bottom, especially as in some of these there is rather rapid loss of weight. If there is enlargement of the liver, the solution is less difficult, because in simple jaundice there is no enlargement, but in its absence I know of no other way than to wait. For stubborn as these rare cases of jaundice are, they are less so than cancer, while, even if they are not followed by ultimate recovery, their course is much longer than that of cancer. Should ascites arise, the question is settled in favor of cancer. It may sometimes be difficult to decide between cancer and hypertrophic cirrhosis, which also furnishes an enlarged, hard, sometimes nodular liver with jaundice. A possible cause in either case must be sought, primary cancer elsewhere pointing to cancer, and the alcoholic habit to cirrhosis, to which also the enlarged spleen and the absence of cachexia point.

There is no special reason why cancer of the liver should be distinguished from sarcoma or adenoma, as the clinical significance of the various conditions is about the same. But if along with a primary sarcoma elsewhere, as in the orbit, there appears enlargement of the liver, then the inference is reasonable that a secondary sarcoma is there established.

There is no evidence by which secondary cancer can be distinguished from primary except the presence of primary cancer elsewhere, notably in the stomach, breast, large intestine, uterus and appendages, and the presumption based on the fact that the majority of all cases of cancer of the liver are secondary. Careful search should, however, be made for cancer in all organs in which primary cancer is likely to occur. The gastric secretion should be investigated chemically, the rectum explored by the finger and speculum, the uterus by the finger, speculum, and sound. Such investigation is further useful in the settlement of the diagnosis of cancer of the liver, for a doubtful case becomes confirmed if a primary focus can be found.

Prognosis.—This is invariably fatal, usually in from three to fifteen months.

Treatment.—This must consist in attempts to relieve the discomfort and prolong the life of the patient.

SYPHILIS OF THE LIVER.

Definition.—Syphilis of the liver includes several morbid conditions due to this specific poison, which are best considered under a single title.

Etiology.—Syphilis of the liver may be the result of acquired or inherited syphilis.

Morbid Anatomy.—The product in the liver of *inherited* syphilis is always a cellular infiltrate which may be diffuse or localized. The first effect is an enlargement and hardening of the organ, which gives place to a reduction in size and unevenness due to contraction of the

new-formed connective tissue. More rarely the gummy tumor of syphilis has been found in the liver of hereditary syphilis.

The changes in the liver due to *acquired* syphilis are regarded as one of its tertiary manifestations and do not show themselves until some time after the primary infection, it may be not for several years. They are represented by the syphilitic gumma or syphiloma, by an interstitial hepatitis, and by amyloid disease. Interstitial hepatitis does not differ essentially from the more usual forms of non-specific cirrhosis. The gumma is the most characteristic lesion of tertiary syphilis. It is a nodular growth which may be as small as a pea or smaller and as large as an orange—from $\frac{1}{5}$ to four inches (0.5 meter to ten meters) in diameter. A favorite seat is the convexity of the organ near the suspensory ligament, another on the under surface in the connective tissue embracing the portal vessels, while it is also found in the substance of the organ. Its tendency is to central cheesy change, and to produce contraction, which distorts the liver and reduces its size, with the formation of cicatricial markings and furrows. These cicatrix-like puckerings and fibrous bands are found also on section of the liver. Endarteritis sometimes invades the smaller and even larger branches of the hepatic artery and portal vein.

Symptoms.—Syphilitic changes in the liver are often first discovered at autopsy. When symptoms are produced during life they are commonly those due to portal obstruction, as already detailed in treating ordinary cirrhosis. *Jaundice* is not a frequent symptom, yet it was early made a matter of record. Thus Paracelsus (1789) is said to have noted the complication of syphilis with a jaundice which could not be cured until the venereal disorder was overcome. Portal (1813) also speaks of jaundice as one of the evils following on syphilis, curable only by the use of mercury. Ricord (1851) noted two cases of jaundice complicated by syphilis; but Gubler (1854) first pointed out that jaundice commonly comes on at the beginning of the secondary stage, and also treated of the relations of jaundice to the general infective process. He collected seven cases in which the jaundice followed syphilitic infection. It accompanied a syphilitic exanthem and was also preceded by *digestive troubles, loss of appetite, nausea, diarrhœa, bitter taste in the mouth*, and *pain in the epigastrium.* I have recently had under my care one case precisely fulfilling these conditions pointed out by Gubler. The jaundice may be slight, moderate, or severe. It rapidly attains its maximum intensity, lasts a variable time, seldom more than a fortnight. Though the explanation may not be immediately easy, Gubler gives sufficient reasons for justifying a relation of cause and effect. It is possible that the poison may act like certain other poisons which produce grave icterus, as phosphorus. On the other hand, it is quite as likely that it may be the result of a duodenal and biliary catarrh, the result of the general disturbance, especially as it is so often associated with other symptoms of this condition, viz., loss of appetite and nausea.

Enlargement of the spleen is an associated symptom where there is amyloid disease, to which ascites may also be added. Sometimes the larger nodules of gummy growth can be felt through the abdominal walls, when the diagnosis has to be made between syphilis of the liver and carcinoma, a differentiation greatly aided by the history of the case.

Diagnosis.—This depends most largely upon the history of the

case, which must be carefully sought. Nor should the physician be satisfied with a negative history, in view of the fact that it is so common for syphilitic subjects to deny infection, even though they know it is their interest to tell the truth. Careful examination should, therefore, be made for secondary symptoms, such as glandular enlargement, or cicatrices and markings left by syphilides.

Prognosis and Treatment.—Patients should be subject to the usual syphilitic treatment by the iodide of potassium and bichloride of mercury so soon as the diagnosis is established and even when it is doubtful. For while early treatment may be efficient in preventing new growths, it is less certain that when present they can be removed by antisyphilitic treatment.

PARASITES OF THE LIVER.

Echinococcus Disease or Hydatid Cyst of the Liver.

The most important of the parasitic diseases of the liver is the echinococcus, or hydatid disease, due to an invasion by the embryo or larva of the tænia echinococcus, a minute tapeworm, consisting of three or four links, and about $\frac{1}{5}$ inch (four to five mm.) long, whose natural habitat is the upper part of the intestine of the dog. It is very rare in this country. This rarity may, however, be more apparent than real, as the parasites are so minute (see Fig. 21) as to be easily overlooked, forming, as they do, minute, thread-like bodies, adhering to the villi of the intestine, while hydatid disease, though not very common, is still, nevertheless, more so than would be expected from the rarity of the worm. There are few hospital physicians of much experience who have not met one or more cases, though they are mostly in foreigners. In Australia and Iceland, where the intercourse between men and dogs is more intimate, it is a comparatively common disease.

Fig. 21.—Tænia Echinococcus.
At *a* natural size; at *b* magnified. (*After Heller.*)

The larva, entering the human intestine with food or drink, has its eggshell dissolved off by the digestive fluids, and bores its way by its stilettos and hooklets into a branch of the portal vein, through which it is carried to the liver. Lodging there, the hooklets disappear, and the embryo becomes a small cyst, possessed of two layers, an external cuticle of laminated structure, and an internal parenchymatous layer, the endocyst. Within the cyst is a clear fluid. Surrounding the cyst is gradually developed a capsule of connective tissue, due to reactive inflammation. The cyst continues to grow, and when it has reached a certain size buds develop from the inner or parenchymatous layer, which are gradually converted into cysts identical in structure with the original cyst. These daughter-cysts, as they

are called, of which there may be a dozen or more, are at first attached to the lining membrane of the mother-cyst, but are soon detached, and from their inner wall other granddaughter-cysts appear. After the capsule has grown from four to sixteen months, there arise from the inner layer of the parent and daughter-cysts, buds which develop into what are known as brood capsules. These outgrowths develop into bodies, which are really the heads of the tænia echinococcus and known as *scolices*. Each presents four sucking discs and a circle of hooklets (see Fig. 21). Each scolex carried into the intestine of a dog may develop into a separate tænia echinococcus.* In man, the development of the echinococcus is usually as above described, producing *echinococcus hydatidosus*.

In animals, the buds from the primary cyst may penetrate outward between the layers and develop outside of the parent cyst, constituting a second or *exogenous* variety,—*echinococcus veterinorum*. In a third form, called by Virchow *echinococcus multilocularis*, from the primary cysts develop buds which are cut off completely, and, becoming surrounded and joined together by thick capsules of connective tissue, form a hard tumor composed of vesicles the size of a pea, often resembling, *en masse*, colloid cancer. In the spaces are found remnants of the echinococcus cyst, at times hooklets or scolices ; at others they are barren. Most cases of this form of disease have been met in Bavaria and Switzerland, but one case being reported in this country, by Delafield and Prudden in their "Pathological Anatomy," third edition, page 372. The subject was, however, a German, who had been in the country five years.

The fluid contained in the young cyst is clear and limpid, has a specific gravity of 1005 to 1009, is non-albuminous, but contains a little chloride of sodium, occasionally a trace of sugar, succinic acid, or hæmatoidin. Scolices and hooklets are almost always present and are of great diagnostic value.

The hydatid cyst ranges in size from that of a pin's head to a child's head. It grows very slowly, and may be in the liver for many years, some say as many as twenty. Ultimately it dies, the walls contract, their contents become inspissated and calcified, as do the walls also. Sometimes they suppurate, the cysts forming large abscesses ; or they may rupture in various directions with corresponding mischief, including sudden death from collapse. Seats of rupture have been the bile passages and inferior cava.

Symptoms.—Small cysts may give rise to no symptoms, being often unexpectedly found at necropsy, and under any circumstances the failure of health is very gradual at first. As cysts become large they produce a *sense of weight or dragging* in the region of the liver and other symptoms, depending on their size and situation : *jaundice*, if they cause obstruction of the biliary passages ; *dyspnœa* and *cardiac disturbance*, if they encroach on the lungs or heart ; *pyæmic symptoms*, that is, fever, sweat, and sometimes chills, with rapid exhaustion, if they suppu-

* It may aid the understanding to contrast here the difference in the mode of development of this little tænia with that of an ordinary tapeworm, say the tænia medio-canellata. In the latter, this ovum develops into a single larva, the cysticercus cellulosæ, while the egg of the tænia echinococcus develops into a cyst, which may produce countless larval tapeworms.

rate. The liver may become very much enlarged, demonstrable by inspection, palpation, and percussion. If there is a single superficial cyst, either in the right or left lobe, it may be felt as an elastic or even fluctuating tumor ; or there may be the distinct feel of a nodular growth over the liver. If posterior in the right lobe, it may encroach on the inferior lung and pleural space, causing dulness on percussion posteriorly and postero-laterally and other signs of pleuritic effusion. *Hydatid thrill* or *fremitus* is always to be sought for. It may be found if the cyst is superficial by placing one hand over the tumor and tapping lightly with the fingers of the other. The result is a vibrating or trembling movement felt for a short time. It is not often obtainable, and is possible only with superficial cysts. It has been ascribed by Briançon to the collision of the daughter-cysts.

If rupture occurs, other symptoms are added. The pleural cavity is often invaded, or the lungs, as evidenced by the expectoration of cysts and hooklets ; the bile passages by the production of jaundice or increased jaundice, and the subsequent appearance of hooklets and cysts in the fecal discharges. Rupture into the stomach is shown by vomiting of hooklets and cysts, into the vena cava by embarrassment of right cardiac action and pulmonary thrombosis from lodgment of cysts, into the pericardium by fatal pericarditis, into the peritoneum by fatal peritonitis, and into the abdominal wall by outward discharge.

Diagnosis.—The differential diagnosis depends on the recognition of hydatid fremitus or on some one of the pathognomonic features just mentioned, and the history of the case in connection with the slowness of development of the symptoms. The resemblance to cancer is sometimes very close in consequence of the presence of nodular swellings over the liver, and to syphilis of the liver for the same reason. In the cancer the health suffers very much more rapidly, but in syphilis scarcely more so, and the history must here again come to our assistance. When suppuration takes place we have the symptoms of abscess of the liver. The recognition of sugar in the fluid obtained by tapping is presumptive evidence of its hydatid nature.

Prognosis.—Where the disease develops sufficiently to manifest symptoms, the chance of spontaneous recovery is very slight. It is possible when external rupture takes place, but this should be anticipated by operative interference, which is often successful.

Treatment.—No medicinal treatment avails, while spontaneous cure is not infrequent by reason of the death of the parasite before the development of the disease to a recognizable degree. A surgeon should he consulted as soon as the diagnosis is made. A preliminary tapping is justified under strict antiseptic precautions, and, in fact, has been succeeded by permanent recovery. Australian surgeons have had the largest experience, and it appears to justify the bolder course of incision and evacuation of the cysts rather than the more conservative method of first securing adhesion of the sac to the abdominal walls and then laying open the cyst and evacuating the contents. The former practice of injecting the sac with iodine has also been discontinued. Should suppuration take place the treatment becomes that of abscess of the liver.

Other Parasites of the Liver.

The remaining parasites of the liver are of pathological rather than clinical interest.

The arthropoda are represented by the *pentastomes*, of which the *pentastomum denticulatum*, the larval form of the *pentastomum* or *linguatula tænioides*, has been found in the liver. The adult worm is lancet-shaped and marked with numerous rings. The female is three to five inches (eight to 13 cm.) long, the male little less than one inch (1.8 to 2.5 cm.). The adult worm has been found in the nostril of man.

The *cysticercus cellulosæ* and *psorosperma* are rare parasites. Of the latter the coccidum oviforme, which is very common in the liver of the rabbit, produces whitish nodules in the liver, as in other organs, ranging in size from a pin to a split pea and even larger. They may produce fever of an intermittent type, diarrhœa, nausea, and tenderness over the liver or other organ invaded with enlargement. (See also parasites at end of volume.)

PERIHEPATITIS.

Definition.—An inflammation of the peritoneal covering of the liver.

Etiology.—Perihepatitis occurs in a circumscribed area, as the result of extension by continuity from some one of the various diseases of the liver, such as abscess or hydatid cyst; as a part of a general peritonitis, and rarely by the spread of a pleurisy through the diaphragm. It may also be caused by direct violence, as by a blow, or be the result of a perforation of an ulcer of the stomach or duodenum or gall bladder.

Morbid Anatomy.—In the more acute forms there is a fibrinous or puriform product with more or less adhesion. These adhesions may lace off areas between the liver and the diaphragm which may be filled with pus, sometimes large quantities, constituting subphrenic abscess, or if there be perforation of the diaphragm, subphrenic pyopneumothorax, more common over the right lobe. In the more chronic form the capsule of the liver is thickened, especially near the portal fissure, and adhesions may take place with adjacent organs, as the diaphragm, stomach, colon, or abdominal wall. The organ may be shrunken and lobulated, and the portal or hepatic vein and bile ducts may be stenosed. The capsule of the liver is often found thickened at autopsies when no symptoms were present during life to indicate it.

Symptoms.—The *pain* and *tenderness* which naturally are attached to this condition, while often exceedingly severe, like those of peritonitis from other cause, are not distinctive of it. Nor is the *jaundice* resulting from compression of the bile ducts, nor the symptoms of portal engorgement, due to compression of the portal vein by the inflammatory products. Physical examination sometimes gives more definite results. Thus a *friction rub* may sometimes be heard in the mammillary line from the 7th rib downward and in the axillary line from the 9th rib downward; also sometimes in the epigastrium. It is, however, of short duration. If there is a purulent collection, the right hypochon-

drium may be distended and the intercostal spaces motionless. The dulness on percussion may extend as high as the angle of the scapula and all the signs of a pleuritic effusion may be present. On the other hand, the lower border of the liver may be much lowered, as far down as the navel.

The course of perihepatitis may be acute, or it may be much prolonged, when all the symptoms of chronic suppurative processes are added,—fever, high temperature, sweats, fistulous communications with other organs, including the lungs, intestines, and abdominal wall.

Diagnosis.—This lies chiefly between that form of the condition under consideration, attended with pus accumulation between the liver and diaphragm, and an empyema or pneumothorax. The physical signs and later symptoms are very similar, and it is chiefly in the initial symptoms that the two conditions differ, the one beginning with cough, pleuritic pain associated with cardiac displacement; the other with symptoms more abdominal in situation. The liver in pleuritic effusion and empyema is never as much lowered in situation as in the hepatic disease. Aspiration may also be availed of in diagnosis. The trocar is to be introduced in the mid-axillary line in the 7th or 8th interspace. It was pointed out by Pfuhl that in subphrenic abscess the spurting occurs with inspiration as the diaphragm moves downward, and in empyema with expiration as the diaphragm moves upward. The atrophied results of perihepatitis are rarely recognized before death.

Prognosis.—This in the severer forms, terminating in suppuration, is grave. A protracted illness, with gradual exhaustion of the patient's strength, is prone to occur, which skilful surgical measures may nevertheless turn to recovery. Milder attacks terminate favorably in a few days.

Treatment.—Treatment in the early stage must consist in measures to relieve pain, local and general. Counter-irritation by cupping operates to check the disease and also to shorten the attack. Sinapisms and fomentations contribute in a less degree to the same end. If suppuration occur, the counsel and aid of a surgeon should be early sought, as it is by his efforts that a cure becomes possible.

Glissonian Cirrhosis.—This is a term applied to a form of perihepatitis in which the capsule is thickened, assuming a semi-cartilaginous appearance. It is associated with reduction in size and some degree of interstitial overgrowth and distortion. The capsule may attain a thickness of 0.4 to 0.6 inch (ten to 15 cm.).

DISEASES OF THE PANCREAS.

Almost the only disease of the pancreas which possesses much clinical interest is cancer. It is true that Reginal H. Fitz has invested the subject of pancreatitis with increased interest by his masterly Middleton Goldsmith lecture, but I note that few cases more are now recognized ante-mortem than previous to its publication. The remaining diseases are of great pathological interest.

ACUTE PANCREATITIS.

This is a rare affection. Fitz divides it into hemorrhagic, suppurative, and gangrenous.

Hemorrhagic Pancreatitis.

It may begin in hemorrhage, which may be traumatic. Most subjects are over thirty. A few are alcoholic. Many have been previously subject to gastric and gastro-intestinal derangements often inflammatory. The causative gastro-duodenitis extends probably from the bowel to the pancreatic duct.

Morbid Anatomy.—The pancreas is enlarged throughout or at one extremity, its head, and is infiltrated with blood, which imparts its color in different shades and which may invade the pancreatic duct. The hemorrhagic foci may alternate with white spots of fat necrosis. The hemorrhage may extend into the peri-pancreatic tissue or the mesentery, meso-colon, omentum, and beyond to the brim of the pelvis. On minute examination round cells and red blood discs are found in the ducts and acini. Many lobules are in a state of coagulation necrosis, while bacteria are present in large numbers.

Symptoms.—The disease begins suddenly with *abdominal pain*, sometimes succeeding attacks of indigestion. It is severe and in the upper left quadrant of the abdomen and in the course of the pancreas, but it may extend throughout the abdomen. It is ascribed to stretching of the cœliac plexus of nerves. There is also *tenderness*. The pain is usually followed by *vomiting*, rarely by nausea only. The vomited matters may be bilious or black. There is usually *constipation*. The upper abdomen becomes swollen and tympanitic or the tympany may be general. The temperature is subnormal or slightly elevated. Death occurs usually within three days, but may be delayed a week. If the patient lives longer the case becomes one of gangrenous pancreatitis. Recovery may occur, though rarely.

Diagnosis.—This is based upon the above symptoms and their suddenness, especially the circumscribed tympany. The disease is to be differentiated from the effects of irritant poison, perforation of the stomach or biliary tract, and acute intestinal obstruction. The history eliminates the former. Perforation of the stomach is preceded by symptoms of ulcer, and of the biliary passages by symptoms of gall-stones. There is no tenderness localized in the region of the pancreas in intestinal ob-

struction, which is rare in the upper part of the small intestine. Obstruction in the large intestine must be eliminated by measures calculated to determine the patulousness of the bowel. Laparatomy has been done for intestinal obstruction and pancreatitis was found.

Prognosis and Treatment.—The former is almost always bad. If recovery takes place, it is accidental rather than the result of treatment, which can be only palliative and such as is demanded by peritonitis.

Gangrenous Pancreatitis.

Etiology.—This is one of the terminations of hemorrhagic pancreatitis. Its causation is, therefore, the same.

Morbid Anatomy.—Gangrene often supervenes upon hemorrhagic pancreatitis about the fourth day, when the tip or the entire gland may be converted into an offensive, dark, slate-colored mass which softens and becomes shreddy. Or hemorrhagic pancreatitis and gangrene may be associated. The organ may become completely sequestrated in the smaller omental cavity, attached only by a few shreds. The adjacent parts exhibit the appearance of peritonitis with dirty, purulent extravasate. Disseminated fat necrosis occurs. The spleen may be enlarged and its veins thrombosed, as may also be the portal vein.

Symptoms.—To those of the hemorrhagic form first present succeed *chills*, *fever*, elevated temperature, general tympanitic, *abdominal swelling*, *tenderness*, and sometimes *jaundice*, *collapse*, and death.

Diagnosis.—This depends upon that of the hemorrhagic form and the symptoms of deep-seated peritonitis.

Prognosis and Treatment.—The former is very unfavorable, while the latter is of a surgical nature, if the condition of the patient justify it.

Suppurative Pancreatitis.

Etiology.—This has the same causes as the acute hemorrhagic form, of which it is the termination when gangrenous pancreatitis does not supervene or recovery take place.

Morbid Anatomy.—The organ is enlarged and contains numerous small abscesses, intervening parts being hyperæmic. There may be peritonitis of adjacent areas of the peritoneum. There may be diffuse suppuration or small abscesses may be disseminated throughout the organ. In the chronic form there may be solitary abscess as large as a hen's egg with cheesy contents. The lesser omental cavity may be invaded and the peripancreatic tissue; rarely also the liver. Fat necrosis is here a rare condition, while thrombosis of the splenic and portal veins may still occur.

Symptoms.—These are prolongation of life for three or four weeks after the symptoms of acute hemorrhagic pancreatitis and those of the gangrenous form set in; with the addition of *irregular chills* and *high temperature*, *exacerbations* and *remissions*, and the signs of deep-seated epigastric peritonitis.

Prognosis and Treatment.—The former is absolutely fatal. There is no treatment but palliation.

Chronic Pancreatitis.—This consists in an interstitial overgrowth by which the organ is hardened and slightly enlarged. The secreting structure is compressed and degenerated. It has frequently been found in diabetes. There may be pigmentary deposits, and pancreatic calculi may be found in the ducts.

CYSTS OF THE PANCREAS.

These are retention cysts, due to closure of Wirsung's duct by concretions or cicatricial contraction. They may become very large and even occupy the entire abdominal cavity. They may be slow or rapid in development.

Symptoms.—In none of the 53 cases thus far collected, 35 by W. W. Johnston and 18 by N. Senn, was there fatty diarrhœa, a condition regarded as symptomatic of suspended function of the pancreas. On the other hand, the stools may be clay-colored and putrescent, probably because there is a simultaneous obstruction to the descent of bile. A resulting tumor presents itself usually in the left part of the epigastrium between the costal cartilages and the median line. More rarely it is in the neighborhood of the navel. It is globular, resisting, and inelastic, changes its position slightly with the movements of the diaphragm, and possesses some lateral motion.

The differentiation of such a tumor, in the absence of more definite symptoms, cannot be said to be easy, yet the diagnosis was made in seven out of Senn's 18 cases. Aspiration should be made. The fluid is usually brown or chocolate-colored, but sometimes it is transparent. It presents some of the characteristics of pancreatic fluid, emulsifying fats and converting starch into sugar.

Treatment.—After exploratory aspiration, the treatment is surgical.

CANCER OF THE PANCREAS.

Morbid Anatomy.—Though a rare disease, it is not infrequently correctly diagnosticated. It is usually primary and situated in the head of the organ. It is commonly scirrhous, but it may also be colloid. It is especially apt to invade adjacent parts by contiguity, and more distant ones by metastasis, especially the liver and lymph-glands. It may arise by contiguity from cancer of the stomach or intestines. It occurs in those past middle life.

Symptoms.—These are not distinctive. The most valuable is *jaundice*, which occurs when the head of the organ is involved. It is caused by the obstruction of the common bile-duct. A tumor may be felt in the pancreatic region, and if it be associated with jaundice, the pancreas may be justly suspected. If we add to these symptoms *fatty* or *greasy stools*, the suspicion is fortified. There are symptoms of *indigestion* and a *dull pain* in the epigastrium, but these are not distinctive. Emaciation and loss of strength proceed irresistibly. As the former advances, the aortic pulse is transmitted with great distinctness through the transverse colon and pancreas. There may be *ascites* and *diabetes mellitus*.

Diagnosis.—Cancer of the pancreas has to be differentiated from cancer of the pylorus, of the transverse colon, of the glands in the hilus of the liver, and from aortic aneurism. The first should not give much difficulty, for the pyloric tumor is movable in a decided majority of cases, the pancreatic fixed; the pyloric cancer is rarely associated with jaundice, the pancreatic is almost always; pyloric cancer produces dilatation of the stomach, pancreatic cancer does not.

Cancer of the transverse colon is rare. It is also more movable than pancreatic cancer, and sooner or later obstruction of the bowel results. Cancer of the hepatic fissure is more difficult to distinguish, but it is higher up and more superficial. The tumor is also more tender. Both are accompanied by jaundice.

The pulsation communicated to the pancreas is very different from the expansile dilatation of aneurism. Fatty stools are a great assistance to diagnosis, but they are by no means always present.

Sarcoma is a possible tumor of the pancreas, but it is not distinguishable from cancer, tuberculosis, and syphiloma.

The **prognosis** is unfavorable, and the **treatment** only symptomatic.

DISEASES OF THE SPLEEN.

Most of the morbid states of the spleen which possess clinical interest are considered in connection with diseases of the blood and with malaria.

Splenitis.—Splenitis occurs rarely as the result of extension of inflammation from a neighboring organ, such as the stomach, perinephritic tissue, the diaphragm and lungs, or as the consequence of injury.

The symptoms are *tenderness* and *enlargement* in connection with the inflammatory conditions of adjacent organs referred to, and it is upon the association of such symptoms with those in the spleen itself that the diagnosis depends.

Perisplenitis.—This may occur as the result of the same causes and may be recognized by the presence of palpable friction.

Abscess of the Spleen.—Abscess of the spleen occurs along with pyæmic processes elsewhere in the presence of the usual causes of pyæmia. Such abscess may break into the stomach, bowel, or lungs, as well as in the peritoneal cavity.

Rupture of the Spleen.—This arises from severe injury, also from extreme and rapidly occurring acute hyperæmia, due to malignant malaria, and from rapidly growing splenic tumors. The symptoms are sudden pain in the region of the spleen, collapse, pallor, and death associated with the causes named.

The Amyloid Spleen.—This appears as a hard, smooth, and enlarged organ associated with amyloid disease of other organs, such as

the liver and kidneys, especially when there has been long-continued suppuration, as in hip-disease, osteomyelitis, tubercular consumption, or syphilis.

Atrophy of the Spleen.—On the other hand, the spleen may be reduced in size by fibroid overgrowth and contraction due to syphilis.

Hemorrhagic Infarct of the Spleen.—Infectious hemorrhagic infarct results in abscess of the spleen. The non-infectious is the result of embolism by a non-infectious embolus, such as arises from the cardiac valves in endocarditis, acute or chronic, from clots in the cavities of the left ventricle, or from clots in aneurisms in the large arteries. After the kidney the spleen is the most frequent seat of such lodgment.

Symptoms.—The event is sometimes ushered in by chills, vomiting, and painful enlargement, the true nature of which can only be inferred if the causes named are present or the symptoms of embolism elsewhere occur simultaneously.

Neoplasms of the Spleen.—These are represented most frequently by gummy tumors which are almost never recognized before death. Carcinoma, sarcoma, and tuberculosis occur, but are not recognizable by special characters. A nodular and uneven spleen may be regarded as due to cancer when associated with cancer elsewhere, sarcomatous when there is general sarcoma, tuberculous if there is tuberculosis elsewhere, and syphilitic if associated with the history of syphilis, especially the congenital form.

Echinococcus of the Spleen.—The spleen may present a fluctuating tumor the nature of which can only be determined by the certain knowledge that a tumor of the same kind exists elsewhere or by the recognition of hooklets in the aspirated fluid. Should the fluctuating tumor be associated with chills and fever it is more likely to be abscess, which, it is to be remembered, may also start as echinococcus disease which later takes on suppuration.

Wandering Spleen.—This is a term applied to a condition of the spleen analogous to the movable kidney and liver. It is the direct result of an elongation of the gastro-splenic ligament and splenic artery and vein. Under these circumstances the usual splenic dulness in the mid-axillary line between the 9th and 11th ribs has disappeared and the spleen can usually be felt somewhere else in the abdominal cavity, usually, however, on the side below its normal site, whence it can be pushed into the natural situation, to leave it immediately as the upright position is assumed. Rarely it is found in more distant situations, even in the pelvis. At times it may form attachments by inflammatory adhesion in the new situations, making its restoration difficult or impossible.

Symptoms.—The symptoms are not invariable. The most constant is a *dragging sensation*, while there may also be the *effects of pressure*, which vary with the situation. There may be pressure on the ureter or bladder, causing difficulty in micturition; upon the bowel, caus-

ing partial obstruction or pain by the compression of sensitive parts. The same train of nervous symptoms which attends floating kidney may also be present.

Diagnosis.—Some difficulty of diagnosis may result in consequence of such vagueness of symptoms. There may be a question between the existence of wandering spleen and fecal tumor. With the former, the normal splenic dulness is wanting, though the well-known fact that the latter in health is sometimes very small may give rise to error. A highly movable cancer of the pylorus, a tumor so movable that it may be felt in the left hypochondrium, may occasion similar difficulty, which must be settled in the same way. And so with other abdominal tumors of movable nature, the normal splenic dulness remains. The question as to whether a movable organ is the spleen or kidney is not likely to be a knotty one, even if the movable kidney be the left, if the same guide be availed of. The difference in outline of the two organs may be recognized in persons with thin abdominal walls and, in rare instances, by the splenic notch. The possible co-existence of a movable spleen and a movable kidney is to be remembered.

Treatment.—The treatment must consist in mechanical measures to keep the spleen in place, measures which must be determined by the requirements of each case. They are variously successful.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneal membrane.

Etiology.—(1) *Of Primary Peritonitis.*—Primary peritonitis, or that form which originates independently of inflammation of adjacent structures, is spoken of as idiopathic in origin. It is a disease of such rarity that its existence may reasonably be questioned, and there are those who deny its occurrence *in toto*. Its reputed cause is exposure to cold. Formerly operations involving the peritoneum were fruitful causes of peritonitis, but since aseptic surgery has become general, such operations are done with an impunity formerly undreamed of.

(2) *Of Secondary Peritonitis.*—By this is meant an inflammation the result of invasion of the peritoneum from a primary focus of disease somewhere in the vicinity, or traumatic agencies like blows or punctures involving the peritoneum. There are two chief foci whence such inflammation originates. One of these is the digestive tract, the other the genito-urinary system, more particularly of women. Inflammation may also invade the peritoneum from the liver, gall bladder, spleen, or perinephritic region, Pott's disease, or psoas abscess. Perforation of the stomach in ulcer or cancer, of the intestine in typhoid fever, appendicitis, and dysentery, are the commonest causes originating in the gastro-intestinal tract. The second focus is purulent inflammation of the fallopian tubes and the genito-urinary tract. Endometritis and metritis may be the

starting-point of such inflammation, which may extend up the fallopian tube, or there may be parametritis with suppuration, the abscess arising from which may rupture into the peritoneal cavity. All of the different forms of secondary peritonitis are infectious, and caused either by organisms responsible for the primary diseases, or by such as are set free by the perforation with the gastric or intestinal contents. The organisms found under these circumstances are the streptococcus pyogenes, the staphylococcus pyogenes aureus or albus, and the bacterium coli commune, the latter especially after perforation of the appendix. The amœba coli has been found in the peritoneal fluid in amœbic dysentery. Peritonitis may also occur from infection from more distant foci of suppuration, when it is also called pyæmic peritonitis.

Finally, peritonitis not infrequently becomes a complication of pleurisy, articular rheumatism, and nephritis by a process not thoroughly determined. The first is probably the result of extension by continuity, since the two cavities communicate by the lymph vessels of the diaphragm. The poison of rheumatism, whatever it is, may be the cause of the peritonitis, while the retained excreta which accumulate in the blood in Bright's disease may act similarly.

Morbid Anatomy.—This varies somewhat with the extent of the peritonitis and the duration of the attack. First there may be a "general" or "diffuse" peritonitis, or it may be "circumscribed." In general peritonitis the peritoneal surface of the intestinal coils is hyperæmic and covered more or less continuously with flakes of yellow lymph made up of fibrin and leucocytes. This is especially abundant in the sulci between the coils, while it also covers the convexity. In an earlier stage, before the exudate appears, the surface of the peritoneum is dull and uneven, from a desquamation of the epithelium. In the flanks is found a variable amount of fluid, which may be serous, sero-fibrinous, or purulent, which, increasing, produces an appreciable ascites. In prolonged cases organization and vascularization from the capillaries of the peritoneum take place, the solid contingent being formed from the epithelium or wandering cells, resulting in adhesions between the coils of intestines and adjacent organs. These are at first soft and easily ruptured, but later become firm bands. These latter are, however, more common in the circumscribed form.

In circumscribed peritonitis limited areas of lymph formation occur and adhesions are more pronounced. Copious fibrino-serous exudate is less abundant though sometimes quite large circumscribed collections of pus occur, laced off from the remainder of the peritoneal cavity by organized tissue. Such abscesses sometimes rupture into the general peritoneal cavity, producing general inflammation, collapse, and death.

Symptoms.—(*a*) *Of an Acute General Peritonitis.*—The most decided symptom is *pain*, usually of extreme severity, which is commensurate in extent with that of the inflammation. There is also extreme *tenderness*, which is similarly limited. So great is this that any tension on the abdominal walls excites pain. Hence the legs are drawn up to relieve this, and we have the well-known position almost characteristic of general peritonitis,—dorsal decubitus, with the thighs flexed on the abdomen. Any motion, such as straining, even the act of breathing and the empty-

ing of the bladder, increases pain. From the nature of the causes this pain is usually sudden in occurrence, succeeding, as it does, on perforation, abscess-rupture, and the like. Sometimes, indeed, it is the first intimation of any illness whatever. *Abdominal distention* is a third characteristic symptom of peritonitis, ascribed to a paralysis of the muscular coat of the bowel, and continues throughout the attack. Rarely, however, the abdomen is flat, hard, and board-like. Rarely, too, pain is altogether absent.

Among the symptoms which may usher in the attack is *vomiting*. It is regarded as reflex in origin, excited by the inflammation of the peritoneum. The effort is sometimes ineffectual, and sometimes a perforation of the stomach permits the more ready discharge of its contents into the abdominal cavity. The vomitus consists of what happens to be in the stomach at the time or of mucus and, if the symptom is prolonged, of green, bilious matter. The primary vomiting is followed by abatement and exacerbation.

The symptoms which are associated with these or succeed upon them vary with the nature of the cause and extent of the diseases. In fulminating cases due to perforation of the bowel, as in typhoid and appendicitis, they are the events of *collapse*, extreme *weakness*, *cold*, *clammy skin*, *frequent*, *small*, and *feeble pulse*. The pulse exceeds 120 and often reaches 160 and even more. The breathing rate is 30 to 40. The temperature is either slightly raised, remains about normal, or may be subnormal. Rarely it is high, 104° and 105° F. (40° to 40.6° C.), though the skin may feel cool and clammy. The expression is characteristic—Hippocratic. The eyes are sunken, the cheeks and temples collapsed, and the nose pinched. The urine is scanty and contains indican.

If the patient survive, the *physical signs* of effusion make their appearance. There is dulness on percussion, first in the flanks, whence it ascends as the fluid increases. If sufficiently abundant the dulness becomes general and fluctuation may be recognized. Palpation and percussion both occasion pain. A change of position from the back to the side causes a change in the position of the fluid and corresponding changes in the physical signs. In extreme cases the diaphragm is raised, the apex of the heart dislocated, and the liver dulness may be obliterated in the mammillary line by combined effusion and extreme tympany. The same may happen to the splenic dulness. Both may be restored by turning the patient on his side. Such obliteration is, however, far more characteristic in what is known as pneumo-peritonitis, a form of peritonitis caused by perforation from an air-containing organ into the peritoneal cavity, and of intense severity, excited by the pathogenic bacteria thus admitted. Extreme pain, rapidly developing collapse, scarcely appreciable pulse, icy coldness of the skin, and extreme distention of the abdomen are the symptoms. The air, of course, occupies the highest part of the abdominal cavity, covering the liver and spleen, causing the obliteration referred to. The distinctive point in the diagnosis between pneumo-peritonitis and the extreme degrees of the ordinary form is the fact that in the former hepatic dulness is absent even in the mid-axillary line when the patient is on his left side, whereas in the latter hepatic dul-

ness may be elicited when the patient is in this position, though it may not be if he is on his back.

Throughout all, the intellect is clear, and while there is often a total want of realization of the inevitable and usually dreaded end, it is as often thoroughly appreciated by the patient and viewed with a calmness which increases the awe which always attaches to the presence of the shadow of death. Rarely, in the course of his experience, is the physician called upon to witness a more painful scene. Toward the very end, however, a somnolence commonly supervenes which obscures the expiring moment, or a slight delirium, the visions of which may be interpreted by surrounding friends as the first glimpses into another world.

The course of such a case is steadily downward, reaching its end in from two to six days.

(*b*) *Of Acute Circumscribed Peritonitis.*—The symptoms include those of the general form in a very much milder degree. The pain is less severe and more circumscribed, the tenderness proportionate, while the definiteness of neither is very sharp. Vomiting may also usher in the attack and may be similarly modified. There may be also the signs of collapse and the patient is often very weak. There is, however, more decided and constant fever, though remittent, as in septic fever generally, and the cases run a longer course, ending not rarely in recovery, but more frequently in death from exhaustion.

As already mentioned, circumscribed abscesses are more frequently recognized by fluctuation and may even point toward the surface, while they are as liable to rupture into the general peritoneal cavity, producing there the symptoms and more usual fatal termination of general peritonitis. This mischievous termination, at the present day, is often prevented by the timely interference of the surgeon. As varieties of such abscess may be mentioned the perinephritic abscess, the pelvic abscess, the subdiaphragmatic abscess, arising from perforation of the stomach or colon or disease of the liver or spleen. The results of circumscribed peritonitis in children are sometimes seen in the shape of a painful, fluctuating tumor in the groin. Circumscribed peritonitis is also more or less associated with the symptoms of the disease which causes it.

Diagnosis.—That of general peritonitis is seldom difficult, especially in the fulminating variety. Some days may, however, elapse before the question is settled, for sometimes the symptoms are closely simulated by those of other conditions. Especially is this the case with the extreme tympany and tenderness which are sometimes associated with typhoid fever, especially where there is deep-seated ulceration. It not rarely happens that on these symptoms is based the diagnosis of a peritonitis, which is not found at necropsy. Entero-colitis may give rise to similar symptoms. On the other hand, it has happened that grave and fatal peritonitis has eluded detection, having been found for the first time at autopsy.

Hysterical peritonitis is a term applied to a condition met with in women when every symptom of acute peritonitis is simulated, even collapse itself. It is needless to say that patients do not die of this disease and that time settles the question ultimately, and where there is recurrence, as is often the case, a second attack is not likely to mislead.

Acute hemorrhagic peritonitis should be mentioned as a variety, the symptoms of which sometimes are the same as those of the ordinary form.

Circumscribed peritonitis is more frequently difficult of detection, and its diagnosis often requires a knowledge of the presence of the causative disease to suggest it. Fluctuation is only available in diagnosis where there is superficial abscess. The exploring needle may, however, be at times availed of.

Prognosis.—This in general peritonitis is almost invariably fatal, the mildest cases only offering the possibility of recovery. Modern surgery has occasionally saved life even in peritonitis which succeeds perforation in typhoid fever. The duration of most cases is from two to six days.

Localized peritonitis is a more promising malady. A few cases get well by spontaneous discharge of resulting abscesses, more with the assistance of the surgeon, and some neglected cases doubtless perish where timely aid from this source would have saved life.

Treatment.—The treatment of general peritonitis succeeding perforation consists for the most part in measures calculated to relieve the patient's sufferings while awaiting the end. If the opportune moment can be seized a laparotomy may be performed, for life has been saved even after perforation in typhoid fever, but no rule can be laid down which will aid in the selection of such moment. Local measures looking to cure, such as blisters and other counter-irritating agencies, are useless. To relieve pain the hot poultice or ice bag may be used in turn. Sometimes one gives more relief, sometimes another. After this opium may be administered to the minimum degree necessary to relieve pain. I see no advantage in the use of opium for any other purpose unless it be also to allay vomiting. It has no effect in limiting the spread of the inflammation. When doubt as to diagnosis exists, as to whether there is true peritonitis or painful distention of the bowel, turpentine may be administered with full doses of strychnine, say $\frac{1}{30}$ to $\frac{1}{20}$ grain (0.002 to 0.003 gm.), while turpentine may be applied locally. Turpentine enemata under these circumstances are of doubtful advantage, in fact, may do more harm than good and should be discouraged.

Special symptoms, such as nausea, faintness, and exhaustion, require the treatment usually appropriate to control them. For the first, ice by the mouth or locally, small doses of champagne, and counter-irritation are useful. For failing strength, stimulants, local heat, hypodermics of ether, digitalis, brandy, and strychnine are available, but I do not approve of the practice so often pursued by young hospital physicians of indiscriminately plying these measures when they must be unavailing.

The treatment of circumscribed peritonitis permits the use of local measures not admissible in the general form. Counter-irritation by blisters and especially by leeches is sometimes of signal service in relieving symptoms, and even effecting a cure if the primary causing disease is removed. The surgeon and the gynæcologist should be early called, as it is more frequently through their assistance that a cure is accomplished.

CHRONIC PERITONITIS.

Etiology.—By far the largest majority of cases of chronic peritonitis are tubercular in origin. Some cases are caused by cancer and other morbid growths in the abdomen, while there are also others of simpler origin. Thus originating, we have both a circumscribed and diffuse adhesive peritonitis.

Local Circumscribed or Chronic Adhesive Peritonitis occurs between adjacent organs, such as the spleen and diaphragm, liver and diaphragm, stomach and liver, and organs in similar relation, as the result of chronic disease in one or the other. Such adhesive connections are not always close, but sometimes consist of bands of considerable length, such as have already been referred to as occasional causes of obstruction.

Symptoms.—In fact, the symptoms of obstruction are often the first evidence of the existence of such adhesive bands. Other symptoms are a sense of *restriction in the motion* of organs involved, with pain when such motion occurs; also *constipation*, *colicky pains*, and pains resulting from traction exerted in peristalsis. Other vague symptoms occur which go to make the patient uncomfortable, but are not distinctive. Should a peritoneal friction, however, be felt, more conclusive evidence is thus furnished. Should suppuration attend chronic inflammation, more distinctive symptoms also arise. In addition to the pain and tenderness a *hectic fever* may be present, which may guide to a correct conclusion with or without the aid of the exploring needle or eventual rupture into one of the hollow abdominal organs.

Diffuse Chronic Peritonitis may succeed upon acute diffuse inflammation of mild degree, which is followed by an abatement in all the symptoms. It may happen in connection with chronic cardiac or hepatic disease where there has been long-continued venous stasis. Or it may succeed the punctures of numerous tappings and most rarely chronic intestinal disease.

Morbid Anatomy.—The peritoneum is thickened. The intestinal coils may be cemented to each other and neighboring organs. The liver and spleen are sometimes covered by thick, tough, gristly capsules. The omentum and mesentery may be thickened and shrunken. There may be thickened nodules, not tubercular. There is in these cases rarely any considerable effusion. A hemorrhagic form suggesting hemorrhagic pachymeningitis was described by Virchow, more commonly situated in the pelvis and characterized by bloody effusion.

Symptoms.—These exhibit for the most part a diminished degree of those characteristics of acute peritonitis, to which may be added *tumor-like swellings and thickenings* and swelling difficult to interpret. Other vague symptoms are engendered by them as the result of contraction and pressure, including *pain*, *œdema*, *albuminuria*, *irregularity of bowel action*, and sometimes *feverishness*. There is little that is characteristic unless it be the occasional presence of recognizable effusion. The very slow forms attended with large effusion are not separable from ascites, the result of hepatic disease, although there are differences in the effu-

sion. In peritonitis the effusion is more turbid, contains abundant albumin, and has a specific gravity rather higher than the fluid of an ascites, 1018 as compared with 1012.

A chronic peritonitis is described by Strümpell and others not unusual in children from two to ten years old. It is associated with decided ascites, debility, and other symptoms of ill health more or less marked, while recovery is the usual termination. Such a cause for the ascites should not be assigned without careful search for others, especially disease of the liver.

Treatment.—The treatment must be determined by circumstances. It is chiefly palliative unless operative interference promises more.

TUBERCULOSIS OF THE PERITONEUM.

SYNONYMS.—*Tubercular Peritonitis; Tabes mesenterica.*

Tuberculosis invades the peritoneum in two ways:—

(1) As a more or less diffuse deposit of miliary tubercles over the visceral and reflected layer, unattended by active inflammation.

(2) As a tubercular peritonitis where the tubercular deposit is associated with an inflammatory proliferation more or less abundant. In a simpler variety of the latter, the *diffuse adhesive*, the peritoneal cavity is obliterated, the coils of intestine being matted together and adherent to the abdominal walls. In a second variety, known as *proliferative* peritonitis, there is marked thickening of the peritoneal layer with less tendency to adhesion and obliteration of the cavity. The omentum is sometimes an inch in thickness and composed of tubercular tissue in various stages of degeneration. The mesentery is similarly infiltrated and shrunken, drawing the intestines together into a ball-like mass as large as a child's head. The coats of the bowel, especially the large gut, also show localized areas of similar morbid changes. Tubercular peritonitis is sometimes associated with cirrhosis of the liver, of which the capsule and that of the spleen may be infiltrated to enormous thickness. There is often in this form considerable effusion, which may be serous or purulent, at times bloody.

Symptoms.—The symptoms include those of chronic peritonitis, except that the abdomen is apt to be harder and more tender. Indeed, a *stiff and rigid abdomen* is quite characteristic of tubercular peritonitis. Later, however, is added, particularly in the upper part of the abdomen, the tympany so characteristic of peritonitis.

In connection with this must be taken the history of the patient, his appearance, the condition of the lungs and the presence of tuberculosis there and elsewhere, particularly in the pleura and bowel, whence extension to the peritoneum is easy by the lymphatic vessels. Four-fifths of all cases of tubercular peritonitis are said to succeed primary tuberculosis of the lungs. In children tubercular peritonitis is frequent as a part of a generally miliary tuberculosis. By primary tuberculosis of the peritoneum is meant simply a tuberculosis in which no primary focus has been found elsewhere.

TABES MESENTERICA, or *abdominal scrofula*, is a tubercular infiltration

of the mesenteric and post-peritoneal glands, most frequently met in children but also in adults. In point of fact, it is often a tuberculosis of all the abdominal organs, including the bowel, peritoneum, liver, and abdominal lymphatics, secondary to tuberculosis of the intestine, but may be also primary without tubercular disease in the intestine or elsewhere. The symptoms are mainly those of the peritonitis and associated enteritis. Thus there is slight fever, abdominal tenderness, effusion, tympany, and diarrhœa, with thin and offensive stools. The children are anæmic, puny, and wasted, while the abdomen is prominent. Sometimes nodules are recognizable by palpation through the abdominal walls. Sometimes large tumors may thus be felt in adults in whom no tubercular disease is discoverable elsewhere.

Diagnosis.—To the symptoms above described may be added, if needed for the purposes of diagnosis, the information to be derived from a test injection of Koch's tuberculin and an examination for tubercle bacillus of the fluid obtained by tapping. The rise of temperature succeeding the injection is almost infallible evidence, due antiseptic precautions being taken, of the presence of tuberculosis.

Treatment.—The treatment for tubercular peritonitis is the general treatment for tuberculosis with such operative interference as may be deemed appropriate after a careful study of each case. The results of operation thus far have been quite sufficiently satisfactory to justify its repetition in suitable cases.

CANCER OF THE PERITONEUM.

Primary cancer of the peritoneum is an event of extreme rarity. Its occurrence as a true epithelial cancer must, however, be admitted. Colloid cancer also occurs as a diffuse and extensive growth, relatively firm, and without fluctuation. More frequently peritoneal cancer is secondary to cancer of the stomach, bowel, pancreas, uterus, or other organ; most frequently, perhaps, as an extension by contiguity, though also by metastasis. It occurs in the shape of small or larger nodules scattered over the peritoneum. The former constitutes what is known as miliary cancerosis. The larger nodules are found in the omentum, in Douglas's cul de sac, around the navel and elsewhere, while the retro-peritoneal glands may be simultaneously involved.

Symptoms.—These are those of chronic peritonitis, including effusion, with the added cachexia, and a diagnosis must be based on these, the antecedent history, and possible presence of cancer elsewhere. The investigation must include the uterus and the rectum. The physical resemblance of the miliary form to tuberculosis is very marked, and in primary carcinoma the distinction is difficult. Palpation may recognize friction in both. In both, the effusion may be bloody, but is more apt to be so in cancer than in tuberculosis. The test injection of tuberculin may also be availed of. The cancerous patient is past middle life, the tubercular occurs in those younger, and especially in children.

The possible presence of echinococci in the peritoneum is to be remembered. The local symptoms may resemble those of cancer very

closely. The presence of hydatid tumors elsewhere, as in the liver, of course suggests the true nature of the simulating disease.

ASCITES.

Definition.—Any freely movable collection of fluid in the abdominal cavity sufficiently copious to be recognizable by the physical signs furnished.

Etiology.—Ascites is a symptom of any one of a number of diseases causing venous engorgement of the vessels draining the peritoneum, but a symptom of such importance as to demand separate consideration. Its causes are local and remote. The most frequent local cause is obstruction to the portal circulation, commonly by some disease of the liver, especially hepatic cirrhosis. Any growth or inflammatory new formation in the gastro-hepatic omentum or hepatic fissure exerting pressure on the portal vein may have the same effect. Abdominal tumors outside of the liver large enough to exert the requisite pressure may also produce ascites. Such are enlarged spleen, tumor of the ovary and even of the uterus. Chronic inflammation of the peritoneum, whether tubercular, cancerous, or simple, especially when the cancer and tuberculosis involve the omentum, is also a cause. More rarely, cirrhosis and emphysema of the lungs and chronic pleurisy cause it.

Remote causes include, first of all, valvular heart disease, the general obstruction due to which causes ascites as a part of a general anasarca, the peritoneal cavity being the last invaded. Rarely it is the only dropsical symptom of a heart disease, in which event there must be associated some intermediate obstructing state of the liver. Bright's disease is also a cause of abdominal dropsy, in which disease, too, the peritoneum is as a rule last invaded. More rarely it occurs as a consequence of intense cachectic states, such as the gravest forms of anæmia.

Symptoms.—Some 14 to 20 pints (seven to ten liters) are required before the physical signs to be described are developed The abdominal cavity thus occupied is more or less distended, pendent when the patient is upright and widened when the patient is on his back, the flanks dropping down and outward. The fluid also flows from one side to the other when the patient turns on his side. If the distention is excessive, "*silver lines*," such as extend across the abdomen in pregnancy, make their appearance, and the umbilicus is obliterated or protuberant. The superficial veins—branches of the epigastric—are dilated and distinctly visible from pressure by the fluid on the vena cava, by which the return of blood from the lower extremities is interfered with. Sometimes these superficial veins from below are seen to join those of the mammary from above. Such dilatation, however, is often contributed to by coincident portal obstruction. (See p. 353.) There may also be œdema of the lower extremities. There is no caput Medusæ about the navel unless the portal circulation is also obstructed.

As intimated in the definition, the physical examination affords the most reliable evidence. To palpation there is the succussion-wave, which is elicited by placing the palm of one hand on the side of the abdomen

and tapping with the fingers on the opposite side. A false succussion-wave is sometimes produced by this procedure in persons with fat, flabby belly walls, but error may be avoided by having an assistant place the edge of his hand vertically on the median line while the tapping is done, as in this way the false wave, which travels around through the abdominal wall, is obliterated. It is always difficult, and sometimes impossible, to palpate solid organs when the abdomen is distended with fluid. Such palpation is, however, facilitated by a modification of the ordinary method, viz., first applying lightly only the ends of the fingers, then suddenly depressing them and so displacing the fluid that the solid organ can be felt.

Percussion elicits absolute dulness over the fluid, while over the bowels, which are floated upward, a tympanitic note is produced, which changes with the position of the patient also. If there is considerable effusion and the patient lies on his back, there is a small oval area of tympany in the middle of the abdomen. If a small amount of fluid is present, the flanks only are filled when the patient lies on his back, and there is a large superficial area of tympany in front which will be substituted by dulness if the patient be placed in the knee-elbow position.

There are also two sites posteriorly where a tympanitic note may be produced by percussion, viz., upward from the neighborhood of the kidney on each side behind the midaxillary line, because in this situation lie the ascending and descending portions of the colon with the posterior aspect uncovered by peritoneum and therefore inaccessible to the fluid.

Differential Diagnosis.—The morbid condition which the physician is most frequently called upon to distinguish from ascites is probably the ovarian cyst. The ovarian cyst, especially when large, furnishes some points of resemblance. Yet there are striking differences. It begins in one side and rises up from the pelvis toward the centre of the abdomen, which soon becomes the most prominent portion, while the dropsical effusion spreads out into both flanks. The ovarian cyst distends one side more than the other at first, and continues to do this even when large and fully developed. It produces no obliteration or projection of the navel, as does abdominal dropsy. Palpation also recognizes fluctuation in the ovarian cyst, but it is usually less distinct and more circumscribed, while in ascites the wave passes all the way across the abdomen. To percussion, the latter condition affords a central tympany and dulness in the flanks, while in ovarian cyst the flanks are resonant because the bowels are pushed into them. This is at least true of one flank, even if the other is completely occupied by a large tumor. If there is tympany in the upper abdomen with an ovarian tumor, it is bounded below by a convex line, while in ascites its lower border is concave.

A change of position has less influence on the dulness in ovarian tumor than in ascites. Vaginal examination affords some information. In ascites the vaginal vault is obliterated, the uterus prolapsed but freely movable, while in ovarian tumor the vagina is less encroached upon, the uterus being sometimes drawn up and less movable.

The characters of the contained fluid are, as a rule, widely differ-

ent. That of a simple ascites is usually transparent, has a low specific gravity, usually below 1012, and contains a small quantity only of albumin and a few leucocytes. The ovarian fluid is usually dark and grumous in appearance, highly albuminous, with a specific gravity of 1020 or more, and reveals to microscopic examination numerous granular fatty cells (compound granule cells), cholesterin plates, and small, pale granular cells. These last are round or slightly oval, about the size of a white blood corpuscle, and are by some regarded as pathognomonic of ovarian cyst contents, and therefore called "ovarian cells." They are found in pleuritic fluids, pus, and even ascitic fluids, but they are much less numerous in these situations. The cell is probably a degenerated endothelial cell from the peritoneum. Their presence in large numbers is certainly a help to the identification of ovarian fluids.

In rare instances the fluid of ascites is milk-white. This occurs when from any cause there is leaking of chyle into the peritoneal cavity, *ascites chylosus.* In the effusion associated with morbid growths, such as cancer and tuberculosis, the fluid is also sometimes white in color from the presence of an unusual number of fattily degenerated cells from these sources or from the peritoneal endothelium.

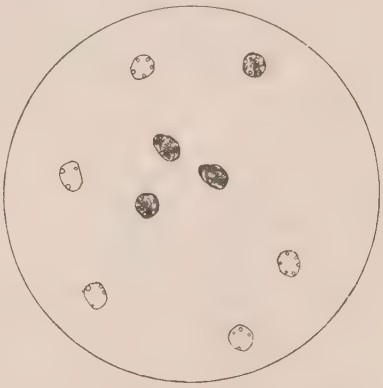


FIG. 22.—SO-CALLED "OVARIAN CELLS."

The over-distended bladder has been more than once punctured by mistake for ascitic fluid, but this accident can never occur if the patient is directed to empty his bladder or the catheter is used before tapping.

Hydronephrosis has been confounded with ascites, but this is less excusable than confounding of hydronephrosis and ovarian cyst. In advanced hydronephrosis the fluid may be almost identical with that of ascites, but its mode of development is from one side and exceedingly slow, while there are pain and tenderness in the region of the kidney. W. v. Leube also relates a case in which he mistook an enormously dilated stomach filled with fluid for ascites, and points out how easily the mistake could have been avoided by the previous use of a stomach tube.

A cyst of the omentum is a rare condition, but should be remembered as a possible one to be distinguished from ascites.

Chronic peritonitis is also attended by effusion, which is, however, more limited than in ascites, and the change in the area of dulness on change of position is less complete because of the peritoneal adhesions, which interfere with the ready movement of the fluid. In tubercular peritonitis, where there is less limitation by adhesions, there is also tenderness. The withdrawn fluid is more highly albuminous and of higher specific gravity than the ascitic fluid.

Treatment.—The treatment of ascites is that of the causative disease. Paracentesis is often necessary to relieve the discomforts of

the patient. The fluid may accumulate with rapidity and the tapping require to be repeated quite frequently, but it is not true, as commonly supposed by the laity, that a first tapping necessitates a second *per se*. When frequent tapping is necessary it is sometimes better to keep the orifice open and allow the fluid to drain away continuously, rigid antiseptic precautions being taken. Under these circumstances the patient sometimes improves rapidly, as he is relieved from the exhausting effect of the pressure and weight of the large mass of liquid and of the constant dread of repeated tappings.

SECTION III.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NOSE.

The first in the natural order of this system are the nasal passages. These are subject to but few medical diseases. They are explored from the front by nasal speculæ or dilators, illustrated in the text. The nares are investigated posteriorly by the rhinoscope, which is only another name for a very small laryngeal mirror. The mirror is, however, introduced

FIG. 23.—HARD-RUBBER NASAL SPECULUM.

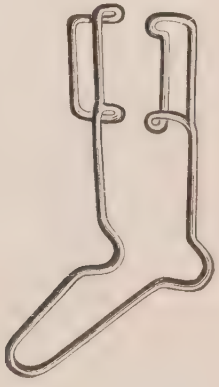


FIG. 24.—SPRING NASAL SPECULUM.

differently. The position of the patient in relation to the observer and source of illumination is much the same, but the head of the former is not raised and the tongue is best held down by the tongue depressor or the forefinger. The warmed mirror is introduced with the reflecting surface upward and is passed backward over the tongue behind the uvula until against the posterior wall of the pharynx. It is then directed upward and forward, and upon it will be found the nasal image and that of the vault of the pharynx. (See Fig. 25.)

RHINITIS.

SYNONYM.—*Coryza.*

Simple acute inflammation of the nasal passages, giving rise to the well-known uncomfortable full feeling which all have experienced under the name of "cold in the head," is a frequent event. There may be previous sneezing. The fulness is due to the swelling of the mucous membrane, the result of inflammation, and is sooner or later followed by a discharge which, at first watery, may or may not become mucopurulent. With it comes relief to the most uncomfortable symptom, the nasal obstruction, this being most serious in nursing children, in whom it

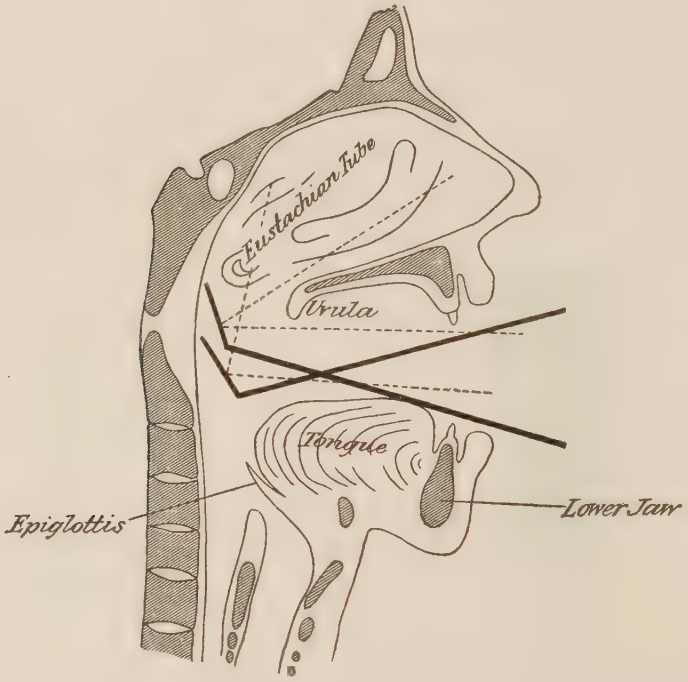


FIG. 25.—ILLUSTRATING TECHNIQUE OF RHINOSCOPIC EXAMINATION.—(*After Sahli.*)

renders sucking often very difficult. There may be slight feverishness, but the constitutional disturbance is always slight and the elevation of temperature is correspondingly trifling, rarely exceeding a degree.

Etiology.—Cold is the most frequent cause of simple acute rhinitis. The exudative forms, including simple fibrinous rhinitis and nasal diphtheria, are, of course, of an infective nature.

Treatment.—When this condition is associated with inflammation of the adjacent mucous membrane of the respiratory passages and of the throat, its treatment is that of the concurrent affection. For the ordinary cold in the head when the discomfort is sufficient to require treatment, I have had excellent results from the inhalation of a solution of

iodine in ether, one or two grains to the ounce. The discomfort is partially due to the dryness, and this is overcome by the application of any simple ointment, as cold cream or vaseline, applied by means of a brush or the end of the finger. Such applications to the adjacent parts are also useful where the discharge is irritating. In infants it is not uncommon to apply the grease to the exterior of the nose, and it is not impossible that some good effect may be thus produced. Dobell's solution may also be sprayed into the nose, and where dry discharges accumulate they should be washed out by gentle injections of tepid salt water. Dobell's solution is composed of sodium borate one drachm (3.8 gm.), glycerite of carbolic acid (U. S. P.) two drachms (7.6 gm.), sodium bicarbonate one drachm (3.8 gm.), and water one pint (0.5 liter).

CHRONIC NASAL CATARRH.

SYNONYM.—*Chronic Rhinitis.*

Chronic catarrh of the nasal passages may be the result of acute inflammation frequently recurring, but more commonly it arises from special causes. A very few of the cases of rhinitis which so commonly occur in the so-called scrofulous may be the result of tuberculosis of the mucous membrane of the nose, but tuberculosis of the nasal passages does occur, and is recognized as a possible cause of chronic nasal catarrh. A more frequent cause is syphilis. In consequence of the offensive odor frequently associated with one form of chronic nasal catarrh, the atrophic, it has been termed "ozœna."

Morbid Anatomy.—Two broad divisions of chronic nasal catarrh are made from the anatomical standpoint—the hypertrophic and the atrophic. In the *hypertrophic*, there is a thickening of the nasal mucous membrane, while in the latter, a thinning or atrophy is present. In the hypertrophic catarrh the mucous membrane is red, swollen, and spongy. The cavernous tissue over the turbinated bones shares in the process and from all sides the nasal cavities may be encroached upon. The protrusion becomes more prominent as the disease progresses, and to it is added a greater or less hypersecretion of mucus.

In the *atrophic* or fetid form, the nasal mucous membrane is thinned, the cavities are enlarged, and within them are found the thick, yellowish-green crusts which, in decomposing, give rise to the characteristic offensive odor of this form of rhinitis. The atrophic process involves all the tissues, from the epithelium down to and including the underlying bone. The accessory sinuses connected with the nose—the frontal, ethmoidal, and maxillary—may all become implicated in this disease, by extension from the nasal chambers, and become the seats of chronic purulent inflammation.

Symptoms.—The two principal forms of nasal catarrh have certain symptoms in common. In both there is more or less *marked obstruction to nasal respiration.* In the hypertrophic form, however, this is due to actual narrowing of the nasal chambers by the overgrowth of the contained structures, while in the atrophic form it is due to the choking of the passages by the large masses of inspissated mucus and muco-pus. There is generally some slight *impairment of the sense of smell* in the

hypertrophic form, while, in the atrophic, it is more often completely abolished. Both forms are usually accompanied by *disturbances of secretion* in the naso-pharynx, and these lead to those noisy efforts at clearing the throat termed "hawking." The "ozœna," or fetid odor, is symptomatic only of the atrophic variety. No odor is attached to simple hypertrophic catarrh.

Hypertrophic nasal catarrh is apparently much more common in the United States of America than in Europe,—indeed, the observations of the specialists go to show that almost every person is more or less the subject of these hypertrophic processes, of which, in many instances, he is quite ignorant until examination has shown their presence.

Treatment.—The proper local treatment of chronic nasal catarrh, which is by far the most important, demands such special measures as in the main can only be carried out by accomplished specialists. This treatment, therefore, so far as can be taken up in this book, can only be palliative, or curative only in the early stage of the disease. In all forms of chronic catarrh the most important measures to be employed by the physician, as distinguished from the specialist, are those which have for their purpose the attaining of the greatest amount of cleanliness of the affected regions. The simplest means for this purpose is sniffing from the palm of the hand simple salt solution of the strength of a teaspoonful of sodium chloride to a pint of water. This, however, accomplishes the purpose but feebly, and the same solution can be more effectually introduced by the irrigator or nasal douche. There is much difference of opinion among specialists as to the efficiency and safety of the douche. Doubtless harmful results have succeeded its careless use, among which are said to have been inflammation of the middle ear and meningitis. Carl Seiler claims, as did the late Dr. Elsberg, of New York, that these may be avoided by the use of proper precautions. According to Seiler, the best irrigator is a tin vessel holding a pint (500 c. c.) and provided with an opening in its bottom to which a rubber tube may be attached, furnished at the end by a nozzle made of glass, rubber, or wood, which fits into the nostril. The vessel is filled with fluid, warmed to a temperature slightly above blood-heat, and the douche should be so placed upon a table or mantel that it does not stand more than an inch above the eyebrows of the patient. If higher, too great pressure may result and the fluid be forced into the frontal sinuses, causing the frontal headache so often complained of, even when otherwise there are no harmful results, or into the Eustachian tube. The nozzle is introduced into one nostril and, the head being inclined forward, the water runs up in that side of the nose until it reaches the velum palati, when it passes around into the other side and through it, bathing the mucous membrane and washing out the mucus or loosening it so that it may be forced out by gentle blowing. It is important that the liquid used should be of the same temperature as the blood, and of the strength which is secured by the proportion mentioned. The plain salt solution may be substituted by alkaline solutions, such as solutions of sodium bicarbonate and borate, of the strength of one drachm (3.8 gm.) of either to the pint (500 c. c.), or a half drachm (1.9 gm.) of each combined. The douche sometimes fails of its purpose when the nasal passages are ob-

structed by deviation of the septum or bony hypertrophies. It should first be used, therefore, carefully under the direction of the physician, who will desist when he finds obstructions. It remains then to use the hand as directed or the nasal spray apparatus recommended by George M. Lefferts, who is one of those who strongly object to the nasal douche as inefficient and dangerous. The little instrument is figured in the text and is so constructed as to produce a coarse spray. He uses with it varying proportions of "listerine" * and water, say from one to four up to equal parts; also an alkaline solution composed of listerine one part, water four parts, and a half drachm (1.9 gm.) each of sodium bicarbonate and sodium borate. Where large quantities are required to wash out the nasal cavities the post-nasal syringe may be used instead of the nasal douche. Listerine is disinfectant and deodorizing, but salicylic acid and carbolic acid may be added to solutions for these purposes. A plug of borated or salicylated cotton may be used for the purpose.

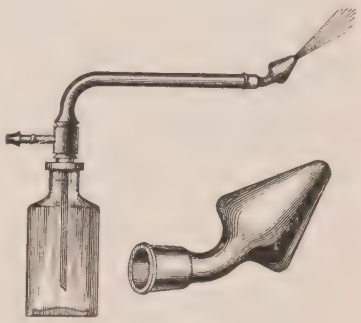


FIG. 26.—COARSE SPRAY APPARATUS.

General treatment, although not as important as the local, is still of great value, and the general health of the patient should be carefully looked after. In view of the fact that atrophic rhinitis is very apt to occur in scrofulous persons, cod-liver oil is a tonic always in order, and should be given for a long time, intermitting occasionally to avoid derangement of the stomach. It should be associated with iron and even with arsenic. Other tonics should be given as indicated and the best of food should be prescribed, including an abundance of meat, eggs, and cream. Wholesome ventilation should be secured for the indoor life, while as much time should be spent in the open air as possible. The air indoors is especially apt to be contaminated by the breathing of the patient with atrophic rhinitis, and on this account good ventilation is imperative. If syphilis is present it should receive appropriate treatment at once.

HAY FEVER.

SYNONYMS.—*Catarrhus æstivus; Hay Asthma; Autumnal Catarrh; Rose Cold; Pollen Catarrh.*

Definition.—A catarrhal affection of the upper air-passages, associated with asthmatic dyspnœa and occurring in the late summer or autumn and spring of the year.

Etiology.—In a large proportion of cases, hay fever has as its fundamental condition an anatomical change in the nasal passages such as hypertrophy of the mucous membrane, a polypoid growth, a deflection of the septum, or a lowered position of the inferior turbinated bones so

* For a formula for a solution similar to listerine, Sp. thymol comp., see p. 212.

that they rest upon the floor of the nose. These conditions are not always demonstrable, but they or some allied source of reflex irritation produce an irritability. This may be increased by a neurotic constitution, though the latter may not manifest itself until after the attacks have become habitual, so that at times at least it is more likely that the neurosis is a result rather than a cause of the disease. A third necessary ætiological factor is an irritant. This irritant, whatever it is, originates usually in the spring or the late summer. In the spring it has been regarded as coexistent with the fragrance of roses. Hence the term, "rose cold." In the autumn the pollen of flowering plants is commonly regarded as the exciting cause, and in certain instances this seems to have been conclusively demonstrated, as by Blakely in his own instance. Other substances are, however, capable of acting similarly, and it is not unlikely that they are numerous. Changes of temperature may excite attacks; also emotional causes, imagined odors, and the like. Heredity is an important factor in its causation, successive generations being attacked with astonishing regularity.

Localities variously favor it. Generally cities furnish more cases than the country, and low countries more than elevated ones, yet certain sea-side places are absolute cures for many cases. Such a place is Long Beach, New Jersey, where is located Beach Haven, a sea-side resort, 50 miles from Philadelphia, which has long been a resort for the victims of hay fever. The disease is more common in the United States than in Europe, and in the United States than elsewhere in America. It is more common in men than in women, there being three cases of the former to every two of the latter.

Historical.—Hay fever was first described by Bostock, an English physician, in 1819, the description being based upon his own experience. He ascribed it to heat. Elliotson, in 1839, appears to have been the first to suggest pollen as its exciting cause. Blakely's observations in his own case (1873) confirmed this view, which is now generally held. Phoebus's classic work was published in Germany in 1862. The first elaborate work by an American was that of Wyman, of Cambridge. George M. Beard, of New York, called the attention to the neurotic factor in 1876. In 1877 Elias Marsh, of New Jersey, read a paper in which he added further evidence to the pollen theory. Voltolini, of Breslau, was the first to point out an anatomical cause, a nasal polyp, the removal of which was followed by the cure of the case. Since then numerous observers have added evidence in this direction, including Hack, in Germany; Harrison Allen, Charles E. Sajous, William H. Daly, John O. Roe, and John N. Mackenzie in this country.

Morbid Anatomy.—There is no morbid anatomy other than that referred to in the remarks on the etiology of the disease.

Symptoms.—The onset of hay fever may be quite sudden, coming on with remarkable regularity often on the same day of the month each year. At other times it is more gradual in its onset. It frequently begins with *sneezing*, and, indeed, may consist entirely of inveterate sneezing. At other times there are *asthmatic* attacks of great severity, closely resembling those of bronchial asthma. Again, there may be *obstinate cough*, with or without expectoration. Or there may be an

alternation of the two symptoms, but generally there is more or less persistent *shortness of breath.* There is also often great *depression of spirits,* and victims have even been impelled to suicide. The eyes are suffused with redness and there may be conjunctivitis.

Diagnosis.—The diagnosis furnishes no difficulty. The season of the year, the periodical recurrence of the cough and asthma, combine to make the recognition easy.

Prognosis.—Patients seldom die of hay asthma, yet I have known cases which seemed to be almost dying when they reached the haven which afforded them relief.

Treatment.—The cure of an individual attack is seldom accomplished except by removal from the district in which the patient resides. The White Mountains and the Adirondack Mountains are favorite resorts in the eastern part of the United States, and Bethlehem, N. H., is the Mecca of American hay fever victims, though other places in the same neighborhood are equally exempt. The Catskills and Alleghanies are less celebrated. Certain sea-side resorts have also a deserved reputation. Beach Haven, N. J., has already been mentioned. Fire Island, on the Atlantic Coast outside of New York Bay ; the Isles of Shoals, Nantucket, and Mount Desert on the New England coast are others. Sometimes a sea voyage will abort a threatened attack.

A few cases have been totally cured by operations on the nasal cavities, such as correcting deviations, removal of hypertrophic process by the knife, or actual cautery.

Palliative treatment at best is uncertain and but partially successful, and, as is always the case with a malady so difficult to cure, the name of its remedies is legion. Of late, irrigation of the nasal passages by the nasal douche with simple salt solution or weak solutions of quinine, one grain (0.065 gm.) to the ounce (15.5 c. c.), have been used, but with varying results. Helmholtz was the first to suggest quinine solution and thought it efficient. A strong solution of cocaine, four to ten per cent., applied with a brush, affords temporary relief, but the effect soon wears away, and there is danger of forming the cocaine habit. Subnitrate of bismuth and boric acid, ½ drachm (1.95 gm.) to the ounce (15.5 gm.) of vaseline or simple ointment, will sometimes allay the itching. Boric acid, ten grains (0.65 gm.) to the ounce (30 c. c.) of water, may be used for the conjunctivitis.

It is usual to give quinine internally also, in doses of ten to 15 grains (0.65 to one gm.) a day. Iodide of potassium and belladonna, so efficient in bronchial asthma, are of little use in hay asthma, but I have known them to be of service. The iodide is better given in small doses, frequently repeated, as three grains (0.2 gm.) every two hours. Fowler's solution has considerable reputation. Morphine is undoubtedly a useful palliative, but its employment is to be deferred until other measures fail. From ⅛ to ½ grain (0.008 to 0.03 gm.) may be required, and the smaller doses should be tried first. Chloral is also of undoubted use as a palliative, and is much safer than morphine, with which it may be combined. It makes smaller doses of the anodyne more efficient, and may be given in combination with $\frac{1}{24}$ to $\frac{1}{12}$ grain (0.0027 to 0.0055 gm.) of morphine at short intervals.

DISEASES OF THE LARYNX.

Examination of the Larynx.—For the proper investigation of the morbid states of the larynx the laryngoscopic mirror has become almost as indispensable as the stethoscope in the study of diseases of the chest. Only under the most favorable circumstances, when direct sunlight is available, may it be used with natural light. In such event the head mirror intended to direct the light upon the throat and laryngoscope should be

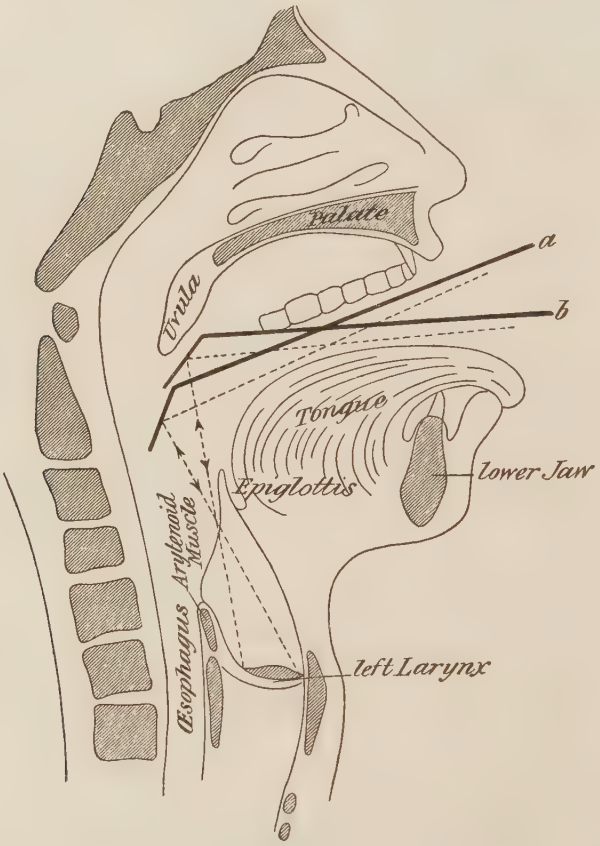


FIG. 27.—ILLUSTRATING TECHNIQUE OF LARYNGOSCOPIC EXAMINATION.—(*After Sahli.*)

plane. Artificial light is far more convenient, and for its management a concave mirror is required. The light may be directed upon the throat by means of a condensing lens, but the mirror is not only more convenient but also less costly. The patient should sit in front of the examiner, whose eyes should be on a level with the mouth of the former at a distance of one foot. The convenience of a head-rest and a stool which can be raised or lowered to suit the stature of the patient is at once apparent. The light should be placed on the right of the patient's head and a little behind it about the level of the ear. The head mirror is

then adjusted upon the middle of the forehead of the examiner, no attention being paid to the central perforation. The patient's head should be slightly raised and the light reflected into the open mouth, of which a general survey should be first made. The patient's tongue is held down by a tongue depressor or drawn out with the aid of a napkin, and may be held by the patient himself. The larynx is thus drawn up at the same time. The mirror, slightly warmed and tested upon the back of the hand, is then carried carefully over the tongue between it and the palate without touching either, or gagging will inevitably result, especially at the first examination. The uvula is gently pushed up by the mirror and the handle carried to one side and raised or lowered until the larynx comes into view. Practice is, of course, necessary to secure success. The patient is requested to say E in order that the epiglottis may be made to rise and the vocal cords approach each other.

As stated, much practice is required on the part of both patient and physician to enable the latter to avail himself of the mirror in the most satisfactory manner. Some persons can barely allow the mouth to be approached, while others are not at all sensitive. The mirror should be at once withdrawn on the occurrence of gagging, and gradually its presence will be sufficiently endured. Various devices have been suggested for the improvement of the illumination in the use of the laryngoscope, and they include the application of electricity, but for these the reader is referred to books especially devoted to the subject.

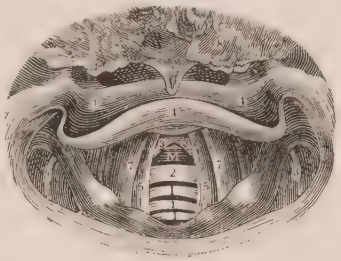


FIG. 28.—NATURAL SIZE OF IMAGE OF THE VOCAL APPARATUS.

1, 1, 1. Rings of the windpipe. 2. Cricoid cartilage. 3, 3, 3, 3. Thyroid cartilage. 4, 4, 4. Epiglottis. 5, 5. Vocal bands. 7, 7. Ventricular bands. 8, 8. Back part of the tongue. M. Crico-thyroid membrane.

The best shape for the laryngeal mirror is on the whole circular, but special conditions may demand the oval form. It must be remembered that the image on the mirror is inverted (Fig. 28) and shows the image at rest and during phonation.

ACUTE CATARRHAL LARYNGITIS.

Etiology.—The most common cause of catarrhal laryngitis is cold, but predisposition plays a most important part. Such predisposition may be the result of previous attacks of laryngitis or it may be brought about by constant use of the organ in speaking and singing; whence it is common with persons thus engaged. In these occupations the larynx is hyperæmic from over-use, and this hyperæmia is ever ready to be fanned into active inflammation. Exposure to cold is constantly at hand to furnish the exciting cause. Laryngitis is also brought about by the inhalation of irritating vapors or gases, while intemperate smoking and the use of strong alcoholic drinks are also causes of the hyperæmia

so readily converted into an inflammation. Catarrhal laryngitis is frequently associated with catarrh of the adjacent parts, as of the nose and pharynx, trachea and bronchi.

Morbid Anatomy.—It is characteristic of the mucous membrane of the larynx and, indeed, of the trachea and larger bronchi below it, that it loses, postmortem, the anatomical characters of the inflammatory process as they appear during life. It is only by the image in the laryngeal mirror, therefore, that we can obtain an idea of these appearances as they present themselves during active inflammation. The picture thus obtained by the laryngeal mirror is one of intense redness with swelling. These changes involve the true and false vocal cords and the trachea below as well as the epiglottis above. The latter appears in strong contrast with the yellowish-pink of health. Even greater is the contrast between the appearance of the vocal cords with the pearly white of health. If secretion has set in, streaks of mucus may be seen in places. Excessive swelling of these parts is known as œdema of the glottis, but it is not frequent in acute laryngitis and will be described separately.

Symptoms.—The most constant symptoms of acute laryngitis are *hoarseness* and *cough*, which vary with the degree of the swelling and hyperæmia, and which also give rise to a sense of something present in the larynx and a constant desire to clear the throat. To these there is sometimes added *pain in deglutition ;* with higher degree of inflammation, a feeling of *constriction* or *oppression.* The cough is more or less *husky* and often stridulous. It is further characterized by its *dryness* and more or less *pain.* Both these characters disappear with the establishment of secretion. There is generally a slight febrile movement, seldom very high. All of these symptoms are aggravated as the disease becomes more severe, culminating in the intense distress and impending suffocation accompanying œdema of the glottis.

Treatment.—The patient should be kept in a uniformly warm, moist air, while special inhalations of such air are extremely useful both in giving comfort to the patient and in abating the inflammation. They require no complicated apparatus. A piece of rubber tubing may be attached to the spout of a tea-pot or kettle, or the steam may be collected by an ordinary funnel and carried thence to the mouth. For obvious reasons care should be taken that the funnel be not allowed to become too hot. Special appliances in the shape of a steam atomizer, more costly and scarcely more useful, can be used instead of the simple measures. The irritative cough may require to be relieved by anodynes, which may be small doses of opium or some one of its preparations or derivatives. Expectorants are of doubtful value and certainly are not nearly so useful as the simple measures which have been mentioned.

Spasmodic or Catarrhal or False Croup.

What is known as spasmodic croup in children of from one to five years is acute catarrhal laryngitis, to which is added a spasm of the glottis producing the hard, stridulous breathing with croupy cough characteristic of this affection, which, once heard, is never forgotten. To the croupy cough is added extreme restlessness and an anxious expression.

The attacks generally come on suddenly at night, the child waking from a sound sleep, although warning is often given by some disturbance of respiration while the child still sleeps. The next day the child may appear almost or quite well; yet there may occur another attack the following night and even the third, while in very severe cases the recurrences continue for a week.

Prognosis.—The prognosis in all forms of acute laryngitis is generally favorable, and it is very rare to hear of a child dying of spasmodic croup. Carelessness may, however, prolong an attack.

Treatment of Spasmodic Croup.—The favorite measure to break the paroxysm of croup in children is an emetic. The simplest of emetics is ipecacuanha, which may be given in the shape of the wine or syrup in the dose of ½ drachm to a drachm (two to four c. c.) every few minutes until the effect is produced. The mineral emetics are more prompt but more depressing in their action. An excellent remedy for the purpose is powdered alum with molasses or honey, which may be given in teaspoonful doses repeated every ten or fifteen minutes until vomiting is produced, but it is not often necessary to give a second dose. While waiting for the action of the emetic the little patient may be put into a hot bath, temperature 98° to 112° F. (36.7° to 44.4° C.), and some mustard may be added. The temperature is kept up by the addition of hot water as required. The majority of attacks of spasmodic croup may be broken up in this way without further treatment. Between the paroxysms the child should be kept upon small doses of syrup or wine of ipecac, say five to ten minims (0.33 to 0.66 c. c.), until nausea is produced, or small doses of powder of ipecac conveniently in the shape of triturates containing $\frac{1}{20}$ grain (0.003 gm.) for an infant a year old. An opiate is particularly useful at bed-time, and by means of it a child may often be tided through a night without an attack.

Just as early as possible in the treatment an aperient should be given, than which none is better than castor oil, but calomel is also an admirable remedy for children given in doses of from one to three grains (.06 to .19 gm.). When there is fever, aconite and sweet spirit of nitre in appropriate doses should be given. Special pains should be taken to maintain a uniform temperature and avoid draughts, especially when the child is perspiring freely, and it is on this account that bed is the safest place.

Counter-irritation by weak mustard plasters is an adjunct to treatment which should never be omitted, while gentle permanent irritation is very useful. It may be secured by any of the rubber spread plasters now sold, known as porous plasters or capcine plasters. In severe cases ice to the exterior of the throat or cloths wrung out of ice water should be used, especially where there is much fever.

Parents are naturally anxious to secure some treatment by which the recurrence of attacks is prevented. Apart from the gradually increasing immunity which comes with added years, little can be done in this direction, certainly no medicine can accomplish anything. It is possible, however, to do something by care and judicious out-door life, by which is secured a "hardening" or protection against the more usual causes of laryngitis. As an instance of neglect of ordinary care may be mentioned the practice so common, especially in the poorer classes,

of allowing children with their heads uncovered at an open doorway or an open window in the cooler seasons of the year. Such exposure is very apt to be followed by an attack of croup the same night. Children are also often too warmly clad while in the house, so that their bodies are constantly moist with perspiration. In this condition a current of air, even when not very cold, will often excite spasmodic laryngitis. Children who are housed are much more susceptible to croup than those who spend a portion of each day in the open air.

SIMPLE CHRONIC CATARRHAL LARYNGITIS.

Etiology.—The causes of chronic catarrhal laryngitis are chiefly those which have been already mentioned as producing the predisposition to acute laryngitis, that is, the constant use of the voice in speaking and singing, excessive smoking, and the use of strong alcoholic drinks. Laryngitis occasioned by the latter cause is often accompanied by chronic granular pharyngitis. So, too, frequently recurring attacks of acute catarrhal laryngitis independent of predisposing cause, and the long-continued inhalation of slightly irritating substances, are to be included among the causes of chronic inflammation.

Morbid Anatomy.—The morbid anatomy of simple chronic catarrhal laryngitis is commonly not widely different from that of the acute form. There is the same redness and swelling, but the former is less vivid. The chronic hoarseness which is so constantly associated with it is due to a permanent thickening of the parts concerned in the production of the voice. Ulceration in simple chronic catarrhal laryngitis is not common, although there may be superficial erosions. The glands are often distended and, if the inflammation is long kept up, a hyperplasia of the squamous epithelium may result in a moderate villous outgrowth on the cords. Nodular swellings on the vocal cords are also a recognized but rare condition, known as *chorditis tuberosa* or *pachydermia laryngis*.

Symptoms.—The most prominent symptom of chronic laryngitis is *hoarseness*, which is found in every degree between a simple roughness of the voice and almost entire loss of it. There is also more or less *pain* and *discomfort*, but this is not ordinarily a conspicuous symptom except when an attempt is made to use the voice. There is more or less disposition to *cough* with a view of getting rid of some foreign substance which seems to be in the larynx. The cough also varies in degree. It may be a mere hack or it may be scraping or ringing. It is also variously effectual in bringing up for the most part a scanty secretion of mucus and muco-pus.

Prognosis.—The prognosis of chronic catarrhal laryngitis is not encouraging for total recovery, largely, perhaps, because it is so difficult to induce the patient to comply with the conditions essential to his cure. Could his entire coöperation be secured, sometimes withheld through no fault of his own, it is not unlikely that better results would follow treatment.

Treatment.—The treatment of chronic catarrhal laryngitis requires first the removal of its causes, whatever they may be. The public speaker

cannot expect to be cured of his malady while he continues the use of his voice, nor can the singer, or he who works among irritating vapors, nor the bon vivant who will not give up his alcohol. Next to the removal of the cause comes the use of local measures, for internal medication with a view to local effect is not promising. Of course, the patient's general condition must be looked after and his strength maintained, but local treatment is mainly to be relied upon. Applications to the larynx may be made in four different ways :—

(1) By inhalation.
(2) By lozenge or troche.
(3) By insufflation.
(4) Direct application.

The general practitioner must in the main confine himself to the first three, and much may be accomplished by them, particularly by the judicious use of the atomizer. Cases are rare in which the direct application of medicated substances to the larynx is necessary, and requiring as they do the simultaneous skilful use of the laryngeal mirror, they are for the most part relegated to the specialist.

Inhalations are further divided into three :—

(*a*) Nebulæ, or atomized fluids.
(*b*) Steam, or vapor.
(*c*) Volatile substances.

(1) A great variety of apparatus is employed for spraying medicated solutions into the larynx, and undoubtedly the most efficient is the compressed-air machine with spraying tubes, but the cheaper forms of atomizers are also useful. Many excellent machines are to be had, but rubber tubes are to be preferred where chemically active solutions are to be used. A double hand-ball secures a more continuous stream of spray than a single one. The spray should be thrown into the wide-open mouth, and advantage taken of inspiration to draw it into the larynx, but a very little practice will teach the patient how to accomplish this. Astringents are the favorite medicaments, of which alum and tannin are the most usual,—of alum a two to 2½ per cent. solution, 15 grains (one gm.) to the ounce (30 c. c.) of water, of tannin one to two per cent. solution, five to ten grains (0.32 to 0.65 gm.) to the ounce (30 c. c.). Other substances are sulphate of zinc, ½ drachm (two gm.) to the ounce (30 c. c.), chlorate of potash, 15 grains (one gm.) to the ounce (30 c. c.), sulphate of iron and ammonia, ½ to one drachm (two to four gm.) to the ounce (30 c. c.). Before any of these solutions are used, however, the larynx should be cleansed from mucus by an alkaline spray, say Dobell's solution, which may be modified by substituting for the carbolic acid and a corresponding portion of water two ounces (60 c. c.) of "Listerine."

Steam inhalation may consist of hot steam alone or the vapor may be charged with a volatile substance. The efficiency of the former in acute laryngitis has already been referred to, and it is in acute disease that it is chiefly used. Benzoin, benzoic acid, and, for chronic conditions, cubebs and benzoate of ammonia are among the substances added. A teaspoonful of the compound tincture of benzoin may be added to a pint of water at 140° F. (60° C.) and placed in any one of the numerous in-

halers, of which, however, none is better than an ordinary tea-pot. Volatile substances like nitrite are commonly inhaled for spasmodic bronchial troubles, to the treatment of which further allusion will be made.

(2) The *lozenge* or troche is a favorite medium for medicating the larynx and is often useful. Few have failed to realize the effect of a stimulating lozenge in clearing the throat, or the soothing effect of one of the anodyne or demulcent kind. An infinite variety is made and the properties sought are either stimulating, astringent, or anodyne, or a combination of two or more of these. Among the first is the lozenge of benzoic acid, the strength of a ½ grain (0.03 gm.) to each lozenge, the cubeb lozenge contains from one to two grains (0.065 to 0.13 gm.) each, the ammonium chloride lozenge contains three to five grains (0.20 to 0.32 gm.), the potassium chlorate lozenge contains five grains (0.32 gm.). The astringent lozenge is made up of tannic acid one grain (0.065 gm.), catechu two grains (0.13 gm.), kino two grains (0.13 gm.), gallic acid two grains (0.13 gm.). Sedative lozenges contain opium in very small quantities, say $\frac{1}{20}$ to $\frac{1}{10}$ grain (0.003 to 0.006 gm.) in each, or of morphia $\frac{1}{30}$ to $\frac{1}{15}$ grain (0.002 to 0.004 gm.), lactucarium extract one grain (0.065 gm.), althæa one grain (0.065 gm.). The selection of a suitable adjuvant in the preparation of lozenges is best left to the apothecary. Three to five lozenges per day are allowed to dissolve in the mouth.

(3) The treatment of the larynx by *insufflation* is sometimes very useful. The difficulty in the preparation of the powder is such that it may be irritating from a failure to secure sufficient subdivision of its constituents. The powder is applied by means of an instrument known as the insufflator. Starch is the basis of most of this class of remedies. Among the astringent powders are tannic acid and powdered starch, each two drachms (8 gm.); alum and powdered starch, of each two drachms (8 gm.). Sedative powders contain of acetate of morphine two, five, eight, and ten grains (0.13, 0.32, 0.52, 0.65 gm.) to ½ ounce (15 gm.) of iodoform. Pure iodoform is also used; equal parts of iodoform and subnitrate of bismuth; subnitrate of bismuth and powdered starch, of the former three drachms (12 gm.) and the latter ½ ounce (15 gm.); borate of sodium three drachms (12 gm.); powdered chalk ½ ounce (15 gm.).

(4) *The direct application* is usually made by means of a brush, sponge, or cotton wad. The favorite is the solution of nitrate of silver of the strength of from ten to 60 grains (0.65 to one gm.) to the ounce (30 c. c.), the weaker solutions being first used, as they are often quite effectual. The application should only be made after considerable experience and always with the aid of a laryngeal mirror, which, indeed, should be used wherever possible in making applications of any kind. Local treatment with nitrate of silver should be used every three or four days, the larynx being previously cleansed with a weak alkaline spray.

The selection of these various forms of medication in chronic laryngitis must be based on the requirements of the case, but the order in which they generally prove most useful seems to be about as follows, subject, however, to frequent variation :—

(1) Inhalation of medicated spray.

(2) Inhalation of stimulating vapor, especially where there is much secretion.

(3) Topical applications by the brush.

(4) Insufflation of powders. Topical applications with the brush are likely to be made much earlier by the specialist than by the general practitioner.

TUBERCULAR LARYNGITIS.

The occurrence of primary tubercular laryngitis, long denied, has come to be generally conceded as possible though rare. Indeed, with the accepted view of the etiology of tubercular phthisis, tubercular laryngitis of the primary kind ought to be of frequent occurrence. For if the tubercle bacillus reaches the respiratory passages from without, the first point of attack would naturally be the larynx. The fact that such is not the case can only be explained on the ground that the bacillus fails to find in the mucous membrane of the larynx conditions as favorable for its growth and multiplication as it finds in the deeper portions of the lung. Since tuberculosis of the larynx is commonly secondary to the same affection of the lungs, the bacillus must invade the larynx in the expectoration, inoculation being favored by the greater or less friction between the vocal cords.

Morbid Anatomy.—To the essential morbid anatomy of tubercular laryngitis is always added that of simple catarrhal laryngitis. The latter has been described. The first stage of miliary tuberculosis without ulceration is sometimes recognized by the laryngoscope as pearly granulations in the mucous membrane, more frequently as a less distinctive, close, small-celled infiltrate. The tubercular ulcer is more easily discovered, yet it possesses no one anatomical character by which it can be infallibly recognized. Nor are all the ulcers in the larynx associated with tuberculosis of the lungs necessarily tubercular. The larynx is more vulnerable to the ordinary causes of simple laryngitis under these circumstances, while the constant coughing and gagging in consumption may of themselves cause laryngitis. The true tubercular ulcer results from the caseation and disintegration of the miliary tubercle. The ulcer thus produced by the fusion of adjacent miliary tubercles is at one stage more or less characteristic by its racemose or sinuous edge, resembling in this respect the conglomerate tubercular ulcer elsewhere. Its favorite seat is over the posterior surface of the upper portion of the epiglottis, the aryteno-epiglottic folds, the vocal cords, and the ventricular bands.

Symptoms.—The early symptoms of tubercular laryngitis differ in no way from those of simple catarrhal laryngitis, and it is the intractability of the disease which often gives the first intimation of its tubercular nature. The stage of simple *hoarseness* with which it is always ushered in varies also in duration, but sooner or later it is succeeded by the *aphonia* and the *painful whispering voice*, which are so characteristic of ulceration of the vocal cords or the other parts intimately concerned in the production of the voice. Sooner or later, too, *painful deglutition* sets in as a result of the extension of the ulcerative process to the more exposed portions of the larynx. Inanition and emaciation characteristic

of the latter stages of the disease now rapidly increase, and death is often a welcome relief to the sufferer.

Diagnosis.—Just suspicion attaches to an obstinate laryngitis associated with acknowledged tuberculosis of the lung. As has been intimated, the distinctive features of the ulceration are scarcely sufficiently well marked to enable us to recognize the tubercular ulcer by the laryngoscope, and to distinguish it either from the ulceration of syphilis or that of certain stages of malignant disease. To distinguish it from the former, in the absence of the more infallible signs referred to, the history of the case may help to a conclusion, and, in case of further doubt, the therapeutic test by iodides and mercurials may be used. Syphilitic laryngitis, even when it is ulcerative, quickly yields to these remedies, as a rule, while the tubercular condition is quite unaffected by them. With the healing of the former comes also the tendency to contraction so characteristic of all cicatrization, and especially of that of syphilitic ulcers. It is also to be remembered that syphilitic ulceration and tubercular ulceration are sometimes associated. The involvement of the tongue in the infiltrating and ulcerating process is more characteristic of tuberculosis.

Prognosis.—The prognosis of tubercular laryngitis is bad at best. It is true that of late years the reported cures of laryngeal tuberculosis have become much more numerous, but these still bear a very small proportion to the cases that progress from bad to worse in spite of the most skilled treatment. It is to be expected that primary tubercular laryngitis is much more easily curable than the form secondary to consumption of the lungs.

Treatment.—All measures which have been mentioned as useful in treatment of chronic catarrhal laryngitis are also more or less so in tubercular disease, with, however, less complete and less permanent results. Marvelous effects have been reported as following the use of lactic acid, while iodoform and even alkaline inhalations are also said to have healed tubercular ulcers. The first requires the skill of the specialist for its application. The treatment must be, of course, associated with that of general tuberculosis of the lung. The painful deglutition, which is at once so characteristic and so distressing, has been relieved by the use of cocaine applied directly to the larynx by the brush or by the spraying apparatus. The former is to be preferred, and for the purpose ten and 20 grain to the ounce (0.65 and 1.3 gm. to 30 c.c.) solutions of cocaine are used. They should be used immediately before the taking of food, as deglutition is rendered painless for the time being by their successful application. Solutions of morphine may be sprayed for the same purpose, or the morphine either pure or mixed with powder or starch may be insufflated upon the painful larynx. Where the pain is persistent, and frequent applications are necessary, I have found none more satisfactory than the officinal solution of morphine sprayed into the larynx.

SYPHILITIC LARYNGITIS.

Symptoms.—It is not necessary to dwell on the etiology of syphilitic laryngitis, as there is but one cause, the virus of syphilis. Like tubercular laryngitis, the morbid anatomy of the syphilitic form is associated with that of simple catarrhal laryngitis of the chronic kind. Excessive mucus and muco-purulent secretions cover the surface of the epiglottis and the vocal cords, while the ulcer of syphilitic laryngitis is usually more distinctive in its characters than is that of tubercular laryngitis. The milder forms of syphilitic laryngitis are not accompanied by ulceration and are in no way peculiar, from the anatomical standpoint. The most distinctive anatomical manifestation of syphilis in the larynx is the mucous patch like that on mucous membranes elsewhere. It is found on the epiglottis, in the laryngeal wall, and on the epiglottidean folds, rarely on the vocal cords. They are rapidly replaced by ulceration. The breaking down of the syphilitic gumma gives rise to another form of syphilitic ulcer often of greater depth. The syphilitic ulcer may come to a standstill at any stage and cicatrization take place with a result which has been referred to. In addition, necrosis of the laryngeal cartilages is not infrequent, portions of these being at times expectorated. Among the results of this cicatrization are stenoses resulting sometimes in complete obstruction, necessitating even tracheotomy for their relief.

Diagnosis.—The diagnosis of syphilitic laryngitis is justified in the absence of tuberculosis elsewhere, especially when the history of primary syphilis is present. The phenomena of syphilitic laryngitis may be either secondary or tertiary, and may occur at any time in the course of disease subsequent to the second or third month following infection.

Prognosis.—The prognosis of this form of laryngitis is rather more favorable than that of tubercular disease, especially if the diagnosis be made early. The effect of contraction after healing is, however, often serious in producing stenosis, or, at least, a permanent impairment of the voice.

Treatment.—The treatment of syphilitic laryngitis is the treatment for the general affection plus the topical treatment. The latter includes the use of measures to free the larynx of mucus and muco-pus, these being followed by applications of strong solutions of nitrate of silver to the ulcers, or even the use of the solid stick. An insufflation of iodoform in combination with bismuth and a little morphine is an excellent addition to the treatment.

ŒDEMA OF THE GLOTTIS.

By œdema of the glottis is meant œdema of those parts which immediately surround that opening.

Symptoms.—Its consideration has been deferred to this place because it so commonly results from the other conditions which have just been treated or accompanies them. Thus, it may occur in connection, though rarely, with acute laryngitis, and occasionally with tubercular and syphilitic laryngitis. It not infrequently, also, is a complication of general

diseases attended with dropsy, especially Bright's disease, more rarely typhoid fever, small-pox, and even diseases of the heart. In any of the latter conditions it may come on quite suddenly. The more precise situation is the sub-mucous tissue of the ary-epiglottic folds or of the ventricular bands. The œdema may also involve the epiglottis. It occurs most frequently in middle life, but it also happens in the young.

Additional symptoms of this condition are a feeling of *intense oppression* or *suffocation*. The breathing is stridulous and the efforts of the patient to obtain air may bring into play all the extraordinary muscles of respiration, the whole expression being in extreme cases one of great anguish.

Treatment.—For the slight degrees of œdema of the glottis the prompt action of a blister to the larynx is often sufficient to relieve the symptoms. Another remedy of some value in the milder cases is a direct spray, frequently repeated, of a 20-grain (1.3 gm.) solution of alum. In the treatment of the severer cases cold plays an important rôle. Ice should be constantly kept in the mouth, as well as applied externally by means of ice-bags. If obtainable, the Leiter coil may be used. When the danger is imminent, and time is too limited to wait for the tardy action of blisters, a half dozen or more leeches may be applied over the region of the larynx. These failing to afford relief, scarification of the œdematous tissues is to be promptly performed, and, as "dernier ressorts," either intubation or tracheotomy.

The hypodermic administration of pilocarpine has been remarkably successful in some cases, and particularly when the symptoms are of a sthenic nature this should never be omitted.

PARALYSES OF THE LARYNGEAL MUSCLES.

To clearly understand these it is necessary to remember—

(1) That the epiglottis is depressed by the thyro- and the aryteno-epiglottidean muscles.

(2) That the opening of the glottis is accomplished by the posterior crico-arytenoid muscles alone, while its closure is effected by the lateral crico-arytenoids, the inter-arytenoid, and the thyro-arytenoids. These latter, acting in conjunction with the arytenoid, become tensors of the vocal cords, producing the different degrees of tension necessary to delicate modulations of the voice in speaking or singing, while, at the same time, narrowing the opening of the glottis. The action of the crico-thyroids is not only to assist in closing the glottis, but to lengthen and increase the tension of the cords and so to raise the pitch of the voice.

The right and left recurrent laryngeal nerves supply all the intrinsic muscles of the larynx on their respective sides, except the crico-thyroid, which derives its motor innervation from the superior laryngeal nerve. The thyro-epiglottidean and the aryteno-epiglottidean, the depressors of the epiglottis, are probably supplied by the same nerve, as is also the arytenoid.* The last three muscles probably also receive fibres from the inferior or recurrent laryngeal as well.

* This view is based on clinical experience rather than anatomical demonstration.

This is the generally accepted view of the motor nerve supply to the larynx, although it has been shown to be subject to occasional variations. It is to be remembered that the motor fibres of these nerves are originally derived from the spinal accessory nerve by branches to the vagus before the latter leaves the cranial cavity. Consequently, motor paralysis may be due to

(1) Degenerative changes in the spinal accessory nuclei in the floor of the 4th ventricle; or

(2) Pressure on or destruction of the spinal accessory fibres before they join the vagus; or

(3) Degeneration, injury, or pressure suffered by the vagus trunk, or its superior and inferior or recurrent branches; or

(4) The paralysis may be myopathic.

As effects of one or more of these causes, we may find the following conditions:—

(1) Paralysis of the thyro- and aryteno-epiglottidean muscles, resulting in a rigidly upright position of the epiglottis.

(2) Paralysis of the crico-thyroid, enfeebling the voice and lessening

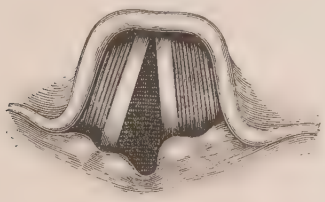


FIG. 29.—CADAVERIC POSITION OF THE LEFT VOCAL CORD, MIDWAY BETWEEN ADDUCTION AND ABDUCTION, IN COMPLETE PARALYSIS OF THE LEFT RECURRENT LARYNGEAL NERVE.—(*After v. Ziemssen.*)

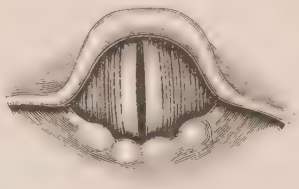


FIG. 30.—COMPLETE BOTH-SIDED ABDUCTOR PARALYSIS OF THE POSTERIOR CRICO-ARYTENOID MUSCLES (THE OPENERS OF THE LARYNX) AT THE MOMENT OF INSPIRATION. —(*After v. Ziemssen.*)

the ability to produce the higher tones. Examination by the laryngoscope discloses imperfect approximation of the cords, a lack of visible vibration in them, and, at times, if the paralysis be unilateral, a higher position of the unaffected cord.

(3) Laryngoplegia or total paralysis of the vocal cord. This is usually the result of pressure upon the recurrent nerve trunk. If the paralysis is unilateral the symptoms are not at all striking. Respiration is not affected; the voice, perhaps, is slightly modified and easily fatigued, but it is far from being wholly suppressed. Inspection by the mirror shows the affected cord in what is called the "cadaveric" position, one that is midway between abduction and adduction. On phonation the sound cord is seen to pass beyond the median line and approximate itself more or less closely to the other. The corresponding arytenoid cartilage is also drawn in front of that of the affected side, and the glottis is thus given an oblique position. (See Fig. 29.)

In bilateral recurrent paralysis both cords are in the cadaveric position, and remain so both during respiration and upon attempted phonation.

(4) Paralysis of the abductors of the glottis, the posterior crico-arytenoid muscles. Unilateral abductor paralysis due to pressure upon the recurrent nerve is not uncommon. The affected cord remains in the middle line in consequence of the unopposed action of the adductor muscle. Phonation is not interfered with, and dyspnœa occurs only in case of severe exertion, so that this affection is, doubtless, often overlooked.

Bilateral abductor paralysis is rare and is generally dependent upon

FIG. 31.—PARALYSIS OF THE INTERNAL THYRO-ARYTENOID MUSCLES (TENSORS OF THE VOCAL CORDS) IN ACUTE LARYNGITIS.

Position of the vocal cords during phonation.

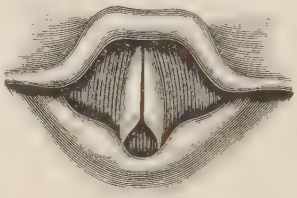


FIG. 32.—PARALYSIS OF THE TRANSVERSE AND OBLIQUE INTER-ARYTENOID MUSCLES (THE RESPIRATORY CLOSERS OF THE GLOTTIS).

Position in laryngitis of the vocal cords during phonation, the respiratory glottis remaining open. (*After v. Ziemssen.*)

central degenerative change. The cords are found in the middle line with but a very narrow chink between them and respiration is consequently noisy and much embarrassed. Slight exertion or emotional disturbance may provoke alarming dyspnœa and, at times, prompt tracheotomy becomes necessary. (See Fig. 30.)

(5) Paralysis of the thyro-arytenoid muscles, the chief tensors of the vocal cords. This is usually bilateral and is quite common. It may be caused by voice strain, by exposure to cold, or it may accompany

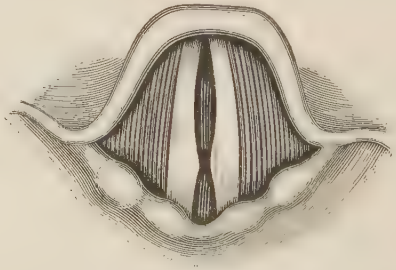


FIG. 33.—BILATERAL PARALYSIS OF THE THYRO-ARYTENOIDS COMBINED WITH PARESIS OF THE ARYTENOID.

catarrhal laryngitis. Approximation of the cords is incomplete, an elliptical space remaining between them which leads to hoarseness and feebleness of the voice. The laryngoscopic appearance is seen in Figure 31.

(6) Paralysis of the inter-arytenoid muscles. This may occur alone, the usual causes being cold, catarrhal laryngitis, or hysteria. A triangular gap is found between the vocal processes in attempted phonation and the voice is very feeble or wholly extinct. (See Fig. 32.)

(7) Combined paralysis of the inter-arytenoid and thyro-arytenoid muscles. In this condition the mirror discovers a narrow, hour-glass opening between the cords caused by the suspended activity of these muscles, while the lateral crico-arytenoids bring the tips of the vocal processes together. (Fig. 33.)

Etiology.—The causes producing paralysis of these muscles are largely pressure by morbid growths within and without the larynx, among the latter of which aneurism of the arch of the aorta and mediastinal tumors are conspicuous. Laryngitis and hysteria are also frequent causes.

Treatment.—The first effort of treatment in all these conditions should be directed to the removal of their causes. Unless this be possible the prognosis is necessarily unfavorable. Catarrhal processes must be cured. The asthenia following diphtheria is to be overcome by general tonics and nerve invigorants. Electricity is frequently of great value, the galvanic or faradic currents being selected as each case may require. Hysterical paralyses are best treated by electricity, chiefly for the moral effect. Brilliant results often succeed this treatment, but relapses often occur.

DISEASES OF THE TRACHEA AND BRONCHIAL TUBES.

ACUTE BRONCHITIS.

SYNONYMS.—*Acute Bronchial Catarrh; Acute Tracheo-bronchitis.*

Definition.—An acute inflammation of the tracheal and bronchial mucous membrane. It is essentially a symmetrical disease, the bronchial tree in both lungs being more or less uniformly invaded.

Etiology.—The most frequent cause of acute bronchitis is the action of cold in chilling the body. It often succeeds an ordinary coryza or cold in the head or a laryngitis, the inflammation extending from these upper air-passages downward. It is naturally more prevalent in the winter than summer. It is usually a symptom of influenza, whether epidemic or sporadic. Invariably, too, it accompanies measles, of which it is the most annoying symptom. More rarely it is caused by irritating fumes. A microbic origin of acute bronchitis is possible in certain cases, but such origin cannot supplant the more usual causes described.

Morbid Anatomy.—The mucous membrane of the trachea and large bronchi is congested and more or less covered with a tough mucus, rich in cells, the hyperæmia being especially marked about the glands whence comes the secretion. Decided cellular infiltration of the mucosa does not occur in ordinary cases, because of the almost tendinous basement-membrane which intervenes between the blood-vessels and the mucosa.

Symptoms.—*Cough* is the most constant and conspicuous symptom. At the beginning it is hard and dry, without expectoration, sometimes painful. As the disease advances it gradually becomes looser. In the milder degrees there is no shortness of breath, but in the severe there is varying degree of *dyspnœa* with a sense of *oppression* or *constriction in the front of the chest*, caused by stenosis of the bronchial lumina, due to the swelling of the mucous membrane and the presence of secretion. *Fever* in slight degree is commonly present, but the temperature rarely exceeds 101° F. (38.3° C.). If it does there is reason to suspect a more deep-seated involvement of the smallest or capillary tubules, whence the name capillary bronchitis, which will again be referred to in broncho-pneumonia. This extension is particularly apt to take place in children and old persons, in whom the physician should always be on the lookout for it. With the access of fever the pulse is correspondingly accelerated. Rarely a chill may usher in the disease.

The scanty sputum of acute bronchitis is at first glairy or mucoid, and later muco-purulent. With the appearance of the latter the cellular element, composed of pus cells and desquamated epithelium, becomes more abundant. With the abatement of the disease the pus cells become again less numerous and finally disappear.

Very often, without treatment, in the course of two or three days the symptoms subside. The cough becomes loose, expectoration is easy, fever and other unpleasant symptoms disappear, and in a week the patient is well. Suitable treatment may hasten such an issue. In other

instances, especially in persons who are weak and run down, no such speedy termination takes place, but even in many of these after a long interval the patient recovers. More rarely, particularly in the very young and old, the inflammation travels down into the smallest tubes, producing the capillary bronchitis alluded to. In other instances still, especially after several attacks, and in the old particularly, chronic bronchitis may supervene with the symptoms and signs which will be described under it.

Physical Signs.—There may be absolutely no physical signs,—inspection, palpation, percussion, and auscultation being alike negative. In other cases inspection may recognize increased frequency of breathing, and possibly increased rate of the cardiac apex beat if there be fever. Palpation may appreciate a rhonchal fremitus if there be sufficient narrowing of the breathing tubes. It may be found anywhere or on either side, and is usually transient. Percussion continues invariably clear so long as the bronchitis is uncomplicated. Auscultation furnishes the most distinctive and constant physical sign, the presence of dry râles, the sonorous and sibilant, which may invade either or both lungs, and may also be transient, coming and going. To these may be added harshness of breathing sounds. When resolution sets in, bubbling râles may substitute the sonorous and sibilant, in consequence of the presence of liquid secretion. For physical signs of capillary bronchitis, see Broncho-pneumonia.

Diagnosis.—This is generally easy. The presence of the dry râles and a clear percussion note belong to no other condition than acute bronchitis and bronchial asthma, but to the latter are added the signs of spasmodic contraction of the bronchi, notably the panting breathing. The same clearness of percussion note continues with the appearance of moist râles, unless there be the complication of capillary bronchitis or pneumonia.

Prognosis.—The worst that can happen to a case of acute bronchitis is to become chronic.

Treatment.—The best treatment for a case of ordinary acute bronchitis is "the bed." Twenty-four to forty-eight hours in a warm bed will go farther to cure such a case promptly than all the cough medicines ever prescribed. Such a course is not, however, always possible, and the physician is often expected to cure acute bronchitis while the patient is on his feet and even attending to business. The patient should, however, be put to bed if possible. Next to rest in bed is counter-irritation. Turpentine and mustard are the best agents. A turpentine stupe or weak mustard-plaster applied to the front of the chest will aid greatly in allaying cough and relieving the sense of oppression.

Cough medicines are, of course, expected, and are useful. In the ordinary simple bronchitis, especially where there is a little fever, there are few remedies more efficient than the simple solution of citrate of potash of the U. S. P. in doses of ½ ounce (15 gm.) every two hours. It may be desirable to add a few drops of wine of ipecac or wine of antimony to each dose to increase the relaxing effect, while if the fever is decided, one minim (0.055 c. c.) of tincture of aconite will aid in breaking it. A

diaphoretic effect is further encouraged by adding 30 minims (two c. c.) of the spirit of nitric ether. By such measures the cough is usually loose in twenty-four hours, the dry râles are substituted by moist ones, and convalescence progresses. If there is decided oppression it may be relieved by inhaling the steam from a hot saturated solution of chloride of ammonium, or the compound tincture of benzoin floated on hot water, while in children an emetic dose of ipecac may produce the desired relaxation.

The cough may, however, be so constant as to harass the patient and keep him awake in spite of the measures suggested. In this event an opiate is necessary, and a small quantity of morphine, say $\frac{1}{16}$ to $\frac{1}{12}$ grain (0.004 to 0.0055 gm.) for an adult may be added to the combination above recommended. It is perhaps on the whole better to administer the opium separately, and, of all the preparations, Dover's powder is probably the best. Indeed, Dover's powder alone is one of the best medicines in acute cough in doses of 2½ grains (0.16 gm.) every two hours, preferably in a pill or capsule ; or if it be at night and a prompt effect be desired, five grains (0.32 gm.) or even ten grains (0.065 gm.) in one dose will often act like a charm. Codeine is a good preparation of opium and has the advantage of disturbing the system less than some others. It may be given in doses of ¼ to ½ grain (0.016 to 0.032 gm.) as often.

Should convalescence be slow and expectoration be prolonged, the ammonium chloride in five- to ten-grain (0.32 to 0.65 gm.) doses with syrup or tincture of squills may be substituted for the sedative mixture, and quinine and restorative measures added to the treatment. If the cough is paroxysmal the preparations of belladonna may be given, and are often efficient in controlling the paroxysms where opium is contra-indicated or deemed unnecessary. So, too, where secretion is copious and cannot be expectorated, belladonna tends to diminish it, and may be given with expectation of relief. Copious secretion in children is somewhat gotten rid of by an emetic. To this end alum and honey may be given or syrup of ipecacuanha in teaspoonful doses. All such measures are, however, depressing and may be succeeded by recurrence of secretion, and should be used only when necessary.

CHRONIC BRONCHITIS.

Synonym.—*Chronic Bronchial Catarrh.*

Definition.—A chronic inflammation of the mucous lining of the large and medium-sized bronchial tubes, commonly symmetrical.

Etiology.—Uncomplicated and primary chronic bronchitis usually develops gradually, representing the accumulating remnants of frequently recurring "colds," each of which leaves something behind it until the chronic condition is established. A bronchitis that is associated with or consequent upon another disease may continue and become chronic after the disease has disappeared. This may happen with measles or influenza, or even, rarely, pneumonia.

Chronic bronchitis constantly attends other affections as a consequence. The most common of these causes is tubercular consumption,

but it is also the result of diseases which favor congestion of the air tubes by reason of the obstruction to the circulation which they cause, such as cardiac valvular disease. Especially is this true of mitral disease and Bright's disease.

Morbid Anatomy.—The bronchial mucous membrane is bathed with a dirty grey secretion derived from the mucous glands, which are sometimes hypertrophied. The darker color is due to inhaled blacks, exfoliated degenerate cells, and sometimes to decomposed blood. On scraping this mucus away there may be little or no change of appearance, at others there may be a decided hyperæmia. In places the mucous membrane may be thickened by cellular infiltration, at others it may be thinned, producing sometimes a lattice-like appearance because of the prominence of the bands of elastic tissue which resist the atrophic process. In old cases there is often dilatation, which may be saccular, fusiform, or cylindrical, and sometimes cavernous dilatations are present, usually about the centre of the lung. It is in the latter more particularly that the mucous membrane is found thinned and the mucous glands atrophied, at others ulcerated. In other old cases there is ulceration and necrosis of the cartilaginous rings.

Symptoms and Course.—The chief symptom of chronic bronchitis is *cough*, which is troublesome in various degrees, and is apt to be worse at night or in the morning. Frequently it is paroxysmal, the spells terminating in free expectoration of the secretion which has excited the coughing. Chronic bronchitis is commonly attended with *free expectoration* either in the manner just described or more uniformly distributed through the day.

The expectorated matter is usually muco-purulent or purulent, the color deepening to yellow as the proportion of pus corpuscles increases, and becoming darker in hue with the admixture of dead epithelium and decomposed blood. The quantity is sometimes very large, amounting to ½ liter (a pint) or more in the twenty-four hours. As the quantity becomes very large, however, the consistence diminishes, and it may be thin and watery. To such copious expectoration the name of *bronchorrhœa* is applied. More commonly it is purulent, containing greenish-yellow masses which are coughed up easily. The bronchi are usually more or less dilated in these cases. The more copious secretion of bronchorrhœa or bronchial blennorrhœa usually separates, on standing, into two portions, a superficial sero-mucous portion which may be frothy, and a lower thick portion made up more largely of pus cells. In addition to such pus cells, the microscope discovers squamous epithelium from the mouth, columnar cells from the deeper air-passages, bacteria, and sometimes a few blood corpuscles.

Respiration is accelerated in various degrees, but except in the rare forms to be alluded to and on exertion dyspnœa is never so marked as even in mild cases of tubercular consumption. The absence of fever is characteristic as contrasted with tubercular consumption, which chronic bronchitis so often resembles in other respects. Sometimes there is slight elevation of temperature, rarely exceeding 100° F. (37.8° C.).

The appetite commonly remains quite good and the patient maintains his weight for a long time. After a while, however, the appetite

and digestion are apt to fail, especially if there is much expectoration, and the patient loses weight. Some subjects of chronic bronchial catarrh remain quite corpulent and well nourished throughout a long illness, and outside the cough the amount of disturbance is often remarkably slight. There is no pain except sometimes about the attachment of the diaphragm in the lower thorax in consequence of the harassing cough.

After chronic bronchitis has existed for a long time in the old, especially when secretion continues copious while expectoration becomes difficult, there sometimes supervenes a condition of *low fever*, probably septic from absorption of putrid matters, and unless expectoration can be reëstablished the patient sinks and the fatal end is not very remote.

A variety of chronic bronchitis is the *asthma humidum* of the older authors, called by Laennec *catarrhe piluiteux*. It is characterized by still more copious serous expectoration, amounting sometimes to two liters (quarts) in the twenty-four hours of thin, frothy sputum, and by severe paroxysms of coughing.

Rarely the cough is "dry," without expectoration except small, tough, tenacious masses of mucoid matter. These are brought up after paroxysms of coughing, often of great severity. This dry variety —the *catarrhe sec* of Laennec—is commonly associated with emphysema and is a very troublesome form.

Still another variety of chronic bronchitis is well-called *putrid* or *fetid* bronchitis, in which the secretions decompose in the air-passages and acquire a sweetish, sickening, and disgusting odor, which may pervade the entire apartment and make the patient a nuisance to himself and others. The decomposition is due to the bacteria of decomposition, the action of which is doubtless favored by retention of secretion in dilated bronchi and phthisical cavities and in a decided majority of cases it succeeds an ordinary chronic bronchitis. It also sometimes follows an empyema which perforates into the lung. At times it is said to be primary. The expectoration is copious and correspondingly thin. It also separates into layers. Sometimes three may be differentiated, one of frothy, muco-purulent matter in which occur separate masses, a middle layer of dirty green muco-serous fluid, and an inferior of thicker, greasy, purulent matter. In the latter the naked eye often recognizes dirty grey masses about as large as a pea, known as Dittrich's plugs, which on microscopic examination are found to contain, besides pus, bacteria and detritus of uncertain origin, together with delicate acicular fat-crystals. Among other fungi are found also leptothria filaments which must not be mistaken for elastic tissue.

The chief additional symptoms are *fever*, it may be septic, with increase of cough and pain in the side. There is also sometimes a *chill*. These symptoms may again abate and those of the more usual form of chronic bronchitis prevail, subject to exacerbation and improvement. The effect of the fetid form, as might be expected, is more severe on the constitution, and there is loss of appetite, indigestion, and failing health. The fingers may be clubbed, as in phthisis. Secondary purulent meningitis and abscess have appeared from the transfer of pus germs. The physical signs do not differ from those of chronic bronchitis and bronchiectasis, to be described.

Physical Signs.—Physical signs of a decided character more constantly attend chronic bronchitis than acute. They present, however, no unchanging picture. To inspection there may be nothing apparent, or the frequently associated complication of emphysema of the lungs may be the cause of a diminished excursion of respiratory motion, and the roundness or barrel-shape of the chest characteristic of that disease may be seen. Such emphysema may give diminution of the normal tactile fremitus and to percussion a hyper-resonance. In the vicinity of a superficial dilated bronchus filled with secretion there may be impairment of resonance. The resonance is, however, restored after copious expectoration, or the percussion signs of a cavity may be substituted, though in the middle or lower part of a lung instead of the apex, as in consumption. Vesiculo-tympanitic or even tympanitic resonance may be present from relaxation of lung tissue, especially in the lower posterior part of the lungs.

Auscultation may also be negative, but much more frequently recognizes an alternation or combination of harsh and feeble breathing, sonorous and sibilant râles, with moist râles of all sizes, variously modified by different distances from the ear and varying consistence of the secretion. The moist râle is the most characteristic sign of chronic bronchitis.

Diagnosis.—This is not usually difficult, for while the symptoms, including coarser appearances of the sputum, sometimes closely resemble those of tubercular consumption the physical signs do not, except when a dilated bronchus presents the same signs as a cavity. Such a dilatation is, however, found in the middle of the lung, and furnishes its signs in the neighborhood of the angle of the scapula rather than at the apex. The absence of fever and especially of tubercle bacilli from the sputum after careful examination is confirmatory evidence of the absence of tubercular consumption.

Prognosis.—This is unfavorable as to recovery, but favorable as to termination. The patient rarely dies of the direct effect of the disease, being generally carried off by some intercurrent affection, often croupous pneumonia. In the old, however, a condition of affairs described on p. 414 may intervene or a broncho-pneumonia may supervene and terminate fatally.

Treatment.—If it were possible to remove every person with simple chronic bronchitis, uncomplicated by heart or kidney disease, to a warm climate they would probably get well. Certainly this is true of the earlier stages. Much may, however, be done at home to prevent the exacerbations due to cold, each of which adds a little to the previous chronic condition, by care in avoiding exposure. This consists mainly in dressing warmly and remaining in-doors in bad weather. Heretical as it may seem, my experience teaches me that old persons can better bear a little "bad air" than "cold air," and it is wiser to submit to a little "closeness" than to encounter very cold air for the sake of "ventilation;" it is, of course, better to have both if possible. It is especially important that the old should be warmly clad with wool next the skin, and precautions against cold feet should be especially secured. When bronchitis complicates other diseases, as heart disease and kidney disease, the treatment of these is important.

In the way of medicine much can be done by the stimulating expectorants. The terebinthinates are the best, and of these one of the best is terebene. Five minims (0.3 c. c.) in a capsule every three hours is a proper dose. Terpene, another derivative of turpentine, may be given in doses of 1½ grains (0.1 gm.) in pill as often, or it may be given in mixture with enough alcohol to dissolve it. Turpentine itself is a good remedy in doses of from ten to 20 minims (0.6 to 1.2 c. c.). Creasote is an admirable remedy in chronic bronchitis. One grain or minim (0.06 c. c.) or two minims (0.12 c. c.) three times a day, increased gradually to five grains (0.3 c. c.) or even more three times a day, begins after a while to diminish the secretion and the cough. Sandalwood oil or balsam of Tolu or Peru may be substituted. The compound tincture of benzoin is another old but good remedy. Other stimulating expectorants, like the carbonate of ammonium or the aromatic spirit of ammonium, are often useful, but they lose their effect after a while. The carbonate of ammonium to be useful must be given often—five to ten grains (0.32 to 0.65 gm.) every two hours. The ammonium chloride is indicated where less of a stimulating effect is necessary, five to 15 grains (0.32 to one gm.) four times a day in combination with the syrup of squill in 15 minim (1 c. c.) doses, both in the compound licorice mixture. In some cases the iodide of potassium is very useful, especially where secretion is scanty. It should be kept up for some time.

Inhalations of medicated vapors are sometimes useful. The compound tincture of benzoin may be thus used, also turpentine. They may be placed on the surface of boiling hot water, the vapor from which will carry the medicated preparation with it. These vapors are more efficient than atomized fluids. Simple steam or a vapor from two per cent. solution of common salt or of sodium bicarbonate may be used. If there is fetor, carbolic acid may be used in the atomizer, a two per cent. solution, or thymol one part in 1000.

Alkalinity is an essential condition of easy secretion from the air-passages, so that both inhalations and internal remedies should fulfil this condition. Hence, simple liquor potassæ U. S. P. in 15- to 20-minim (1 c. c. to 1.25 c. c.) doses in milk is a good remedy. To this end the free use of alkaline mineral waters, as those of Vals, Vichy, Ems, is useful.

Digitalis and strychnine are excellent medicines, especially the latter. Both stimulate the cardiac action and aid in pumping the blood through the lungs with increased force, thus causing relief to the congested mucous surfaces. Strychnine may be given with advantage in ascending doses.

As to health resorts suitable for cases of chronic bronchitis, those with a dry climate, not too cold, should be selected for cases with copious secretion, such as Southern Georgia and the Carolinas or New Mexico in this country, or for stronger persons the cooler climate of Colorado. For cases of dry bronchitis the warmer moist climates of Florida are very suitable. In this, as in all other diseases, the factor of complete bodily and mental rest enters largely into the cure.

Chronic bronchial catarrhs always improve in summer, and it is generally sufficient if the patient be directed to leave the hot and noisome city and spend his summers either in the mountains or at the seaside, where the air is pure and bracing.

Of foreign resorts those of southern Europe, especially Italy, the western Riviera, San Remo, Mentone, and Cannes, are suitable, while still better, if they can be availed of, are Egypt, Algiers, and the island of Madeira.

BRONCHIECTASIS, OR BRONCHIAL DILATATION.

Historical.—Laennec (1819) was the first to describe anatomically bronchial dilatation, and his admirable account remains at the present day the standard description of the condition. It should be mentioned, however, that Laennec himself tells how his attention was first called to it by Cayol, at that time a student but afterward a professor of medicine, who was "astonished at finding a diseased state of the lung which up to that time had remained undescribed." This, according to Laennec, was in 1808. Laennec attributes the formation of bronchiectatic cavities to mechanical cause, viz., the pressure of bronchial secretion. Andral (1823) called into play disturbances of nutrition, and Reynaud (1835) the respiratory act.

Etiology.—The most common cause of bronchiectasis is chronic bronchitis, either simple or tubercular, the effect of the inflammation being to weaken the bronchial walls so that they yield to the inspiratory and expiratory strain to which they are subjected in the act of coughing. It is therefore often associated with emphysema. The same cause contributes to the bronchial dilatation following broncho-pneumonia, measles, and whooping-cough in children. Accumulated secretion is also a factor, as seen in the dilated bronchi which succeed obstruction of a bronchial tube by a foreign body or compression by aneurism or mediastinal tumor. The traction associated with fibroid induration is also a cause of bronchial dilatation. Hence we find it in association with interstitial pneumonia and sometimes in chronic pleurisy. Finally, bronchial dilatation is rarely a congenital defect, in which event it is also commonly unilateral and general, *bronchiectasis universalis* of Grawitz.

Morbid Anatomy.—Bronchial dilatation is cylindrical and sacculated. The terms explain themselves. Both forms may occur in the same lung.

In the *cylindrical* form, which is the more common, dilated tubes of nearly equal calibre may run through the substance of the lung, from the root to the pleural surface, producing an appearance not unlike the fingers of a glove. More frequently the smaller tubes only are affected, dilatation being recognized at autopsy by the inequality of lumen rather than by *ante-mortem* physical signs. It may, however, be suspected in any case of chronic bronchitis with copious expectoration.

The *saccular* bronchiectases are spherical or oval dilatations, into which the tube merges gradually or suddenly. They may attain a diameter of two to three inches (five to eight cm.), more or less. The lung tissue about a saccular dilatation is rarely normal. Commonly the dilatations, single or multiple, are surrounded by indurated and contracted lung tissue, the traction of which on the bronchial wall produces the dilatation. Adhesion of the lung to the costal pleura also contributes, and

large subpleural cysts are at times thus formed by the contracting tissue. The cavities thus produced are commonly at the base of the lung, while in chronic phthisis they are found at the apex. Cylindrical and saccular dilatation may also be associated under these circumstances. In universal bronchiectasis the entire bronchial tree is converted into a series of sacs communicating one with the other. Many cavities in pulmonary consumption are primarily bronchiectatic cavities.

In all forms there is decided change in the bronchial wall, which is atrophied. The atrophy not only attacks the mucous coat, but also the muscular, and sometimes the elastic tissue and cartilage, reducing the wall to a thin, smooth membrane, lined with pavement epithelium instead of the usual cylindrical form. At times overgrowth, involving particularly the connective tissue, takes place, forming lattice-like projections on the inner surface of the tube, already referred to in treating of chronic bronchitis. At other times ulcerative processes develop, perforating the bronchus and invading the lung parenchyma, converting the bronchiectasis into an ulcerating cavity.

Symptoms.—These, in addition to those of the disease with which the bronchiectasis is associated, are the peculiar *sputum* and *paroxysmal cough.* The sputum furnishes the most distinctive feature, from which alone the diagnosis can be sometimes made. It is muco-purulent, of a dirty yellowish-green color and unpleasant, stale, and sweetish odor, though not exactly fetid, as in fetid bronchitis. It is often raised in mouthfuls—another characteristic. Since, however, the dilatation may be caused by, and may itself become a cause of, fetid bronchitis, the expectoration may have a true fetor. It also separates into layers, usually three, of which the upper is frothy and thin, the middle mucoid, and the lowest made up of pus and epithelium in various stages of fatty degeneration, acicular fat crystals, and sometimes red blood discs and hæmatoidin crystals sufficient to color it. There is, however, usually no elastic tissue or tubercle bacilli, unless there is associated tuberculosis with ulceration of the bronchial walls.

The cough is paroxysmal, because it is not usually excited until the sac, which is often insensitive, becomes full enough to irritate the healthy mucous membrane, when cough is at once excited and continues until the cavity is empty. The paroxysms are usually in the morning, when they may be excited by a change in position. After their termination there is commonly a long period of rest until the sac, or a sac, is again filled. The more paroxysmal the cough and copious expectoration in chronic bronchitis, the more likely is there to be a dilated bronchus. Very characteristic is the absence of fever.

Physical Signs.—When distinctively present these are those of a cavity in the lung, readily recognizable when near enough to the surface. They include tympanitic percussion, bronchial and even amphoric breathing, bronchophony or pectoriloquy if the cavity is empty. If it contains liquid, gurgling may be heard and the percussion note is dull. To palpation there is usually increased vocal fremitus caused by surrounding consolidation. These signs vary according as the cavity is filled or emptied of secretion. A restricted breathing excursion may also be present uninfluenced by the state of the cavity, whether full or empty.

A bronchiectatic cavity is usually distinguished from a phthisical cavity by the absence of tubercle bacilli and elastic tissue from the sputum, the situation of the cavity in the centre instead of the apex of the lung, the history of its development, the absence of cachexia and fever. Hypertrophy of the right ventricle is more frequent in bronchiectasis, but may also be present in fibroid phthisis with or without bronchiectasis.

A circumscribed empyema which has ruptured into the lung is much more sudden in its development than bronchiectasis, while the history of a previous pleurisy is superadded. A coincident external perforation of an empyema would clear up all doubt. A true abscess of the lung which has found its way into a bronchus has also a different history of origin, succeeding, as it usually does, a pneumonia, a massive hemorrhage, or traumatic cause. The same is true of gangrene of the lung, which is, however, disclosed by the extreme fetor of the breath and expectoration.

Treatment.—This includes that of chronic and fetid bronchitis, to which may be added, under favorable circumstances, the injection of sacs and their drainage. It is to be remembered, however, that physical signs are sometimes misleading, and that what seems to be the clearest evidence as to the exact site of a sac is not always to be relied upon. I well remember a case of my own in which there seemed to be the most conclusive evidence of the presence of a dilated bronchus below the angle of the left scapula, evidence not satisfactory merely to myself, but also to my colleagues, William Pepper and J. William White. At our request Dr. White opened the thorax by exsecting parts of two ribs, when, to our astonishment, no cavity could be found by the cautious use of exploring needles. Prompt closure of the wound was followed by healing, and the patient lived eight months afterward.

BRONCHIAL ASTHMA.

Definition.—Bronchial or spasmodic asthma is a paroxysmal asthma or a panting for breath, which is the direct result of a spasmodic contraction of bronchial tubes.

Etiology.—There is some diversity of opinion as to the etiology of bronchial asthma. This much, however, is admitted, that in some way there is produced a narrowing of the smaller bronchi.

Various explanations of the narrowing are suggested. Some allege a simple swelling of the mucous membrane as a cause. Such swelling is variously spoken of as "fluctionary" (Traube), "vasomotor turgescence" (Weber), "diffuse hyperæmic swelling," or "exudative" inflammatory (Curschmann). On the whole, the older view of Trousseau, that it is due to a spasmodic contraction of the muscular coat, wears best, and has recently received the support of Biermer. Mention should be made of the theory of Wintrich and Bamberger that asthma consists in a tonic spasm of the diaphragm, a theory which Riegel has further developed by ascribing the spasm to a super-excitation of the phrenic nerve, resulting in a partially inhibited excursion of the diaphragm.

Accepting Trousseau's view of a primary spasmodic contraction of

bronchi, it becomes necessarily a reflex act, the causes of which are various. It implies first a hyper-excitability of the reflex centre. Hence bronchial asthma is not infrequent in neurotic persons, and has even been classed as a functional nervous disease with neuralgia and epilepsy, with which it is said to alternate at times. Such hyper-excitability is sometimes inherited, so that bronchial asthma often runs in families. Presupposing such excitability, numerous peripheral causes may supervene, the most frequent of which is bronchitis. It very often happens that an asthmatic subject has his attack of asthma brought on by "taking cold," the incident bronchitis being the initial event.

Comparatively modern studies have demonstrated the association of some affections of the throat and nasal passages with bronchial asthma, and that their removal has resulted in its cure. Among these have been enlarged tonsils, chronic catarrh, nasal polyps, and the like. Other causes in susceptible persons are impressions of certain odors, pleasant and unpleasant, notably that of flowers or plants in early summer, whence the term "rose" asthma and hay asthma, both of which are allied affections. A change of air, as from town to country or the reverse, or from mountain to lowland, acts similarly. Causes more remote than those of the nasal passages, such as gastric derangement, intestinal worms, uterine disease, may be admitted. Purely emotional causes, as fright and emotion, may also act. The frequency of bronchial asthma in children has been already mentioned. It is more common in the male sex.

Morbid Anatomy.—Whatever may be the morbid state of the tubular structure of the lung during an attack of asthma, there are no post-mortem appearances which are distinctive of it. In the first place, the chance is seldom offered at the opportune moment, and I know of no report of a necropsy made on a person dying in an attack of asthma. In the case of the asthmatic dying at other times there may be found the morbid states peculiar to chronic bronchitis and emphysema, but nothing more.

Symptoms.—The symptoms of an attack of spasmodic asthma are unmistakable. The typical asthmatic is apparently in good health between the attacks and often is so up to the time of the attack, which then comes on suddenly, often at night. At others there is a prodromal stage, a feeling of thoracic discomfort or "*tightness*" *in the chest*, or an anxious, nervous, restless feeling, the import of which is well understood by the victim.

The attack consists of a long-drawn-out inspiratory act in which it is evident the air cannot get into the lung fast enough to meet the demands of the *besoin de respirer*. The auxiliary muscles of respiration, the sterno-cleido-mastoid and the scaleni, do their best to enlarge the thorax, but that is not the difficulty. It is the contracted tubes which resist the entrance of the air. Even more marked are the effort and the duration of expiration. Hence the dyspnœa is spoken of as an expiratory dyspnœa. The abdominal muscles are the auxiliaries here and they contract strongly and assume a board-like hardness. The air is heard to whistle as it enters and passes out of the chest. The patient sits in an upright position, or leans slightly forward, and often astride

of a chair grasps the back with his hands, for it is by fixing the shoulders that he can bring the extraordinary muscles of respiration into play. His face is anxious, pale, or it may be cyanotic, and few more distressing pictures are seen. Notwithstanding his efforts, they fail of their purpose and comparatively little air enters the lungs. These efforts do not, however, usually include accelerated breathing, at least accelerated to any marked degree, while in a few instances the breathing rate is diminished. The temperature is normal or subnormal, the pulse is accelerated and small.

The attacks last for a variable period, rarely less than an hour, and unless broken up sometimes several hours. They may terminate as suddenly as they begin, sometimes with a spell of coughing. On the other hand, cough is not a marked symptom and in brief paroxysms may be altogether wanting. In the severe ones, however, it is present, accompanied by a tough and scanty expectoration, containing rounded masses of matter, either yellowish or greyish translucent—the "perles" of Laennec. On minute examination the latter are found to be of spiral structure, more or less distinct, and to contain numerous swollen and fattily degenerated pus cells and cells shed by the bronchial mucous membrane and alveoli. They have long been recognized, but were first studied by Ungar and Curschmann and are known as Curschmann's spirals. The yellow masses contain, in addition to the cells named, various numbers of acicular crystals, which were first found by Leyden in the sputum of asthmatic patients and therefore called Leyden's crystals. They are identical with the so-called Charcot's crystals, found in leukæmic spleen, bone marrow, and semen. A second form of Curschmann's spiral contains in the inside of a tightly wound spiral of mucin fibrils another bright, clear filament. The spirals are believed by Curschmann to be formed in the finer bronchioles and to be a product of bronchiolitis. Their spiral form is unexplained. The sputum also sometimes contains crystals of calcium oxalate and calcium phosphate.

In addition to the cases of typical asthma in patients perfectly comfortable between attacks and for which the above description is intended, patients with chronic bronchitis and emphysema are subject to attacks which may be called symptomatic asthma. The symptoms are, however, similar and need not be repeated. It is to be remembered, too, that emphysema is caused by asthma as well as that chronic bronchitis and emphysema may cause asthma.

Physical Signs.—These are also characteristic. Inspection notes the most labored effort in breathing, yet the chest moves but slightly. It is in a state of permanent inflation. The spaces above and below the clavicle and above the sternum, the intercostal spaces, and the pit of the stomach are drawn in for the same cause,—that is, the thoracic cavity not being filled from within, the external atmospheric pressure forces the yielding portions inward. Rhonchal fremitus is recognized by palpation, while vocal fremitus, obscured by the rhonchus, is further diminished by a frequently associated emphysema. Percussion is negative in uncomplicated asthma, but if asthma is associated with emphysema it may produce abnormal resonance. Auscultation recognizes the most striking and easiest recognized of the physical signs. All over the chest are

heard sonorous and sibilant râles, inspiratory and expiratory, the latter longer and more marked. In fact, for the most part, they do not require the ear to be placed close to the chest. They may be heard at a distance. The vesicular murmur, on the other hand, is inaudible. Later in the attack, as secretion increases, the râles become moist. It is to be remembered that chronic bronchitis, emphysema, and asthma may also complicate each other, and render correspondingly complex the physical signs.

Diagnosis.—This can usually be made at a glance. Spasm of the glottis and paralysis of the abductors of the glottis produce similar efforts at breathing, but the dyspnœa is inspiratory and unattended by the lung sounds characteristic of asthma, while the history will be found different. Hysterical dyspnœa furnishes no physical signs, while in cardiac asthma also the breathing sounds are normal, or there is crepitation. (See also Cardiac Asthma.)

Prognosis.—Bronchial asthma, though a distressing, is not a fatal disease. Very often the attacks grow more infrequent and milder as the patient grows older, and they may disappear altogether, while in some cases they increase in severity and frequency with age. In other cases a cure is effected by discovering and removing the cause.

Treatment.—The first object in the treatment of asthma is to relieve the paroxysm. This is best accomplished by a hypodermic injection of morphine, ¼ grain (0.0165 gm.), with $\frac{1}{150}$ grain (0.00044 gm.) of atropine, which may be repeated in an hour if ineffectual. If morphia is not at hand, nitrite of amyl may be inhaled from a handkerchief on which a few drops have been placed, or a pearl may be broken if one of these be at hand. In the absence of amyl nitrite, chloroform or ether may be similarly used. After the paroxysm is broken every effort should be made to discover a cause for the recurring attacks. The nose may be responsible and should be carefully examined for any one of the causes referred to. Possible peripheral irritation, whether by error of diet, gastric derangement, uterine or other distant reflex cause should be sought and corrected. These are not always easily found, but sometimes they are. Bronchitis, when present, also requires treatment by the usual remedies.

It is needless to say that where special external causes, such as odors or exhalations or undiscoverable peculiarities of location, are responsible, they should be eliminated. With all our efforts, however, the cause remains in perhaps a decided majority of cases undiscovered. But even under these circumstances we have in the iodide of potassium and belladonna two drugs which possess undoubted power to relieve bronchial asthma and even to avert attacks. A certain measure of relief is almost always secured by these drugs, and in many cases the effect is magical. From five to ten grains (0.32 to 0.65 gm.) of the iodide and three to seven minims (0.16 to 0.38 c. c.) of the tincture of belladonna should be given every three hours until relief is permanent. Lobelia, formerly much used instead of belladonna, has fallen into disuse, probably on account of its very disagreeable nauseous effect. The fumes of burning paper impregnated with nitrate of potash and stramonium are also useful adjuvants, and cigarettes and pastiles made out of such paper are con-

stantly employed for their effect. These substances form the bases of most of the advertised remedies for asthma.

The diet of asthmatics should be exceedingly simple, as indiscretions in it are often the exciting causes of attacks. No fixed rules for climatic treatment can be laid down, as conditions favorable to different cases are exceedingly capricious. On the whole, high, dry climates are most suitable for pure asthmatics, *i. e.*, those cases uncomplicated with emphysema; though moist, warm climates, such as those of Florida and Madeira and the Canary Islands, are also serviceable, especially when there are catarrhal symptoms; or Southern California, where the climate is also warm and equable, but drier. When there is emphysema high altitudes are not well borne. Cold and moist climates are harmful. Oxygen breathing is often helpful, as is also inhalation of compressed air in the pneumatic cabinet.

PLASTIC OR FIBRINOUS BRONCHITIS.

Definition.—This is a rare form of inflammation of a part of the bronchial tree, commonly chronic, but occasionally acute, in which a fibrinous mould of the bronchus and its branches is formed and expelled. It does not include those instances which occur in connection with croup or diphtheria as an extension downward, or in pneumonia by centripetal extension.

Etiology.—No definite cause for this bronchitis is known, though it is frequently associated with tuberculosis—in ten out of 21 cases studied by Model. It occurs at all ages, and though more common between ten and thirty, has occurred at seventy-two. It has happened in more than one member of a family. It is found more commonly in males and in the spring months. Other associations named are probably accidental, as with skin diseases. In the chronic form, which consists in recurring attacks extending over many years, the same part of the bronchial tree is apparently attacked each time.

Morbid Anatomy.—As primarily expectorated, the exudate is a round mass mixed with blood and mucus. This mass, sometimes considerable, may be unrolled, when it is found to be a true cast of dendritic shape and hollow interior of the trunk and branches. The latter may even terminate in bulbous ends corresponding to the infundibula. The mould is true fibrillated fibrin, in which are imbedded numerous leucocytes. Charcot's crystals and Curschmann's spirals have occasionally been found. It is whitish or yellowish-grey in color, and concentrically laminated. In the latter feature it differs from the branching clots which occasionally form in a bronchus and branches after hemorrhage into the lungs. These are solid and homogeneous. A fine example of one of these is in the pathological museum of the University of Pennsylvania. The true fibrinous casts are usually 1½ to two inches (3.75 to five cm.) long, but may be five or six inches (12.5 to 15 cm.). The tubes whence the casts come are not superficially changed, but on minute examination have been found bereft of epithelium. The submucous tissue may be swollen and infiltrated with serum.

Symptoms.—These are those of an ordinary bronchitis of severe form. There is aggravated *cough* and *dyspnœa*. Sometimes this is preceded by a stage in which there is for a variable time, sometimes prolonged, bronchial *catarrh* of ordinary severity. At times the attack is ushered in by *rigor*, and there is *high fever*, *pain* in the side, and *soreness*. There is slight expectoration until the cast is loosened and expelled. The cough preceding the expulsion does not usually last more than a few hours, though it does sometimes continue for days. With the expulsion of the cast comes prompt relief for the time being. It is sometimes followed by slight *hæmoptysis*, which may also rarely precede the expulsion. The expectoration of a single cast does not, however, terminate the attack. After twenty-four to forty-eight hours the cough and dyspnœa return, and another cast is expelled. This may be kept up for several days, after which the attacks cease to recur. Smaller pieces may be expelled. The attacks may occur but once in a lifetime, or they may be repeated at intervals for years.

Physical Signs.—These are usually those of bronchitis. There is no dulness on percussion unless it be from consolidation due to collapse of the lung. There may be, according to Walshe, circumscribed pneumonia with crepitant râle and rusty sputum. The effort at breathing is labored, and if there is obstruction of a large tubule there may be retraction of the lower ribs during inspiration. The cast then begins to be loosened and moist râles make their appearance.

Diagnosis.—The rarity of the disease is so great that in the absence of distinctive physical signs the true condition is rarely suspected. In recurring attacks the true nature of such a severe attack of bronchitis may be suspected.

Prognosis.—This is usually favorable, although the symptoms are often alarming. N. S. Davis has reported two fatal cases of the acute form.

Treatment.—The disease, as long as its true nature is undetermined, is treated as an ordinary bronchitis. If its true nature is suspected the vapor from alkaline solutions should be inhaled, or these should be sprayed into the larynx. Lime water is one of those commonly employed. Alkaline solutions may be of the strength of 30 grains (1.95 gm.) of sodium bicarbonate to the fluid ounce (30 c. c.) of water. Jaborandi or its active principle, pilocarpin, may be tried. Emetics should also be employed where the breathing is much embarrassed. They sometimes have the effect of discharging the cast. Iodide of potassium is recommended and certainly should be used where the attack is protracted.

DISEASES OF THE LUNGS.

EMPHYSEMA.

There are two applications of the term emphysema, and they have very different significations. In the *first* place, there is *interlobular* or *interstitial* emphysema, in which, in consequence of rupture of air vesicles deep in the lung structure, the air escapes into the interlobular tissue and may collect there like rows of beads outlining the lobules, while under the pleura larger vesicles may form. This form occurs after wounds of the lung and in severe and persistent whooping-cough and in cough of bronchial asthma, in both of which the expiratory strain is very great. It is termed also acute emphysema. It is not, however, demonstrable clinically, except in those cases where it takes place at the root of the lungs and the air travels along the trachea and enters the subcutaneous tissue of the neck and chest walls. It gives rise to a peculiar crepitation to the feel. A similar condition may be due to infiltration of the tissues with gas, arising from decomposition. It is found in the neighborhood of wounds which take on an unhealthy action and where decomposition leads to the generation of gas. This form of emphysema is, of course, more circumscribed than that due to a wound of the lung.

The *second* form, *vesicular emphysema*, is an over-distention then atrophy of air vesicles, either symmetrical, involving both lungs, or localized. *Localized* emphysema is also called compensatory or vicarious. It occurs where certain portions of a lung are adjacent to another which cannot from some cause expand fully in inspiration. Such are portions of the lung adjoining tubercular areas, or areas of collapsed lung, or parts whose expansion is prevented by pleuritic adhesions. It is particularly the anterior parts of the lung that are the seat of compensatory emphysema in the latter case. Where such complemental dilatation is impossible, as is often the case in extensive pleuritic adhesion, the chest wall must sink in to occupy the space. Perhaps all emphysema is more or less localized, but in general or symmetrical emphysema very much larger areas of both lungs are involved and the dilatation is in no sense compensatory. The distended air vesicles are useless, while many of them are also atrophied. The former is also called hypertrophic, but *pseudo-hypertrophic* would be a much more suitable term because there is no true hypertrophic enlargement.

A *third* form of emphysema of the lungs is known as *atrophic emphysema;* called also by Sir William Jenner *small-lunged emphysema.* In it the whole lung and thorax may be reduced in size and even the respiratory muscles may be atrophied. It is a disease of old persons and is to be regarded as an involution process. There is a true atrophy of air vesicles, and bullæ of various sizes are formed by the wasting of intermediate vesicles.

Vesicular Emphysema—Pseudo-hypertrophic Emphysema.

Etiology.—By far the larger number of cases of emphysema are the result of chronic bronchitis. This bronchitis may begin in childhood.

It may begin as whooping-cough, from which the child has not completely recovered, or succeeding which it has been subject to constantly recurring attacks of acute bronchitis. It is scarcely likely, if the lung preserved the proper integrity of its tissue, that even under the forced strain of coughing it would undergo the dilatation and destruction characteristic of emphysema. Sooner or later, with chronic bronchitis, there is a change in the integrity of the tissues of the air-vesicles, which makes them more yielding and more likely to give way under the strain. Blowing on wind-instruments and glass-blowing, as well as occupations requiring muscular strain and the lifting of heavy weights, are assigned as causes. Bronchial asthma is another cause. In all these cases both inspiration and expiration coöperate to produce the strain, but it is probable that expiration is the more potent factor. The severe cough of chronic bronchitis begins with a deep inspiration, which, while harmless to a healthy air vesicle, may over-distend a weak one. Then follows closure of the glottis and a forcible contraction of the muscles of expiration—abdominal muscles. The latter compress the lower part of the lung especially, and as the air cannot escape it is forced into the upper parts of the lung, over-distending the air vesicles there. Apart from the cough, in chronic bronchitis of the middle and small bronchi the swelling of the mucous membrane, and in bronchial asthma the contraction of the bronchial tubes, impedes the passage of air, and deep and strong inspirations are taken which tend also to over-distend the alveoli. In like manner the same narrowed tubes resist the exit of the air, and the ordinary elastic recoil of the lungs is not sufficient to empty them. Again, the expiratory muscles by being called into play compress the bronchioles more than the air vesicles, impede the exit of air from the latter, and thus over-strain them. So also under horn-blowing and muscular strain we have the effect of deep inspiration, and especially the increased pressure during expiration, with the glottis closed. We may admit also a valve-like effect of certain plugs of mucus, which permit the entrance of air during inspiration, but do not allow its exit. Finally, the vesicles become filled with air, which cannot get out. In the act of expiration the lung is compressed, and the air is forced back into all the little corners; it is forced in the direction of least resistance toward the air vesicles in the apices and edges of the lungs, where dilatation takes place. This is probably one way in which expiratory strain acts in producing dilatation. The valve-like action may also be in the opposite direction, permitting the air to get out of the vesicles, but preventing it from getting into them, and thus finally a portion of the lung becomes collapsed. The inspired air must go somewhere else and produces what may be called a collateral dilatation. Thus it is that the supplemental or vicarious emphysema, already referred to, may be produced.

The vesicles thus over-distended finally lose their elasticity, like an over-distended India-rubber air balloon, which, being distended over and over again, finally loses its power to recoil. Succeeding the over-dilatation comes atrophy of the vesicles, and with this the blood-vessels surrounding them are destroyed. Although under these circumstances the lung occupies more space, its blood-aërating power is diminished. The circulation is cut down to the larger trunks, and the blood takes a short

cut, as it were, from the pulmonary arteries to the pulmonary veins. The aëration of the blood is thus rendered difficult or impossible, accounting thus in part for the dyspnœa.

There is also reason to believe that heredity plays a decided rôle in the causation of emphysema, and that congenital defect often takes the place of acquired nutritive retrogression. This was first shown by the late James Jackson, of Boston, who found that in 18 out of 28 cases one or both parents were affected. Accordingly, too, emphysema is surprisingly common in children, and in adults may often be traced back to childhood.

Morbid Anatomy.—The emphysematous chest is often highly characteristic, in that the antero-posterior diameter is greatly increased, making the two diameters nearly or quite equal, producing the "barrel shape." On opening the thorax in an adult the cartilages are found calcified, and on raising the sternum the greater volume of the lungs at once shows itself. They are in a state of permanent inspiration, meeting by their edges in the mediastinal space and almost or entirely covering the pericardium. Nor do they collapse when removed from the chest.

The individual air vesicles are not only dilated, but large numbers of them are atrophied, producing bullæ of various sizes, from the walls of which extend inward edges which are the remnants of vesicles, so that the large vesicle has been aptly compared to a frog's lung with its semi-partitions. The pleura is pale and the lungs especially so, partly from atrophy of the pulmonary capillaries which goes with the destruction of the vesicles, but there seems also to be diminution in the natural pigment. They pit readily on pressure. The distention and destruction are not limited to the periphery of the lungs, but are also found in the centre and toward the root, where large bullæ two to three inches (five to eight cm.) in diameter may be found.

The bronchi exhibit the changes already described under chronic bronchitis and bronchiectasis. An important anatomical change not usually demonstrable before death because of the voluminous lungs is *hypertrophy of the right ventricle of the heart*, due to the extra effort required to drive the blood through the diminished vascular area in the lungs. In the later stages the hypertrophy has given way to dilatation and there may be relative insufficiency of the tricuspid valve with dilatation also of the right auricle. In a few cases there is hypertrophy of the whole heart. There is sometimes, also, atheroma of the pulmonary artery and of the other blood-vessels, or there may be associated pulmonary tuberculosis of the fibroid variety, as well as Bright's disease.

Symptoms.—The typical emphysemic subject may often be recognized by his peculiar round-shouldered stoop and barrel-shaped chest. Rarely this form of emphysema is an acute or comparatively rapid development, succeeding whooping cough; but the approach of the disease is mostly gradual, the first symptom to develop and remain constant being *shortness of breath*, which is partly due to the fact that the air in the vesicles does not undergo the usual interchange. In health the intercostal muscles, the diaphragm, and auxiliary muscles of respiration enlarge the thoracic box, and the lungs expand to fill it partly by their own resiliency, but chiefly to fill the vacuum, producing the act of inspi-

ration, while the air is expelled in expiration partly by the recoil of the elastic tissue and partly by the pressure of the contracting thorax. This natural resiliency is absent in a large degree, while the thoracic box also remains in a state of "rigid dilatation." The lung is always filled with air, but it is air charged with carbonic acid and does not change. As a consequence, the patient makes increased efforts to draw the air into the lungs, but as the air vesicles are already filled with air these efforts are ineffectual. The *dyspnœa*, which is but slight at first and is brought about only by exertion, soon becomes decided and constant. The *pulse rate* is also accelerated, but the temperature is usually normal. *Cyanosis* is a characteristic symptom in established cases, owing to the universal presence of unaërated blood.

Outside of these symptoms are mainly those of the associated bronchitis, viz., *cough*, *expectoration*, and sometimes *oppression*, while variations in these add to or abate his discomfort. With failure of the right heart come venous engorgement, dropsy, and effusions into the serous sacs. Tuberculosis of the fibroid type sometimes develops.

Physical Signs.—The physical signs are more or less distinctive. Inspection discovers a rounded chest, with increased circumference, and wide intercostal spaces in the hypochrondriac regions but narrow above. The epigastric angle is obtuse. The result is the well-known "barrel-shaped" chest. More rarely the emphysema may be so circumscribed as to produce local bulging, by preference in the upper lobe of the right and lower lobe of the left lung. Expansion of the chest wall is diminished, while the scaleni and sterno-cleido-mastoid muscles stand out distinctly. The chest does not expand, but is raised up by these muscles, which are hypertrophied; the apex beat is not visible, but may be felt displaced downward and to the right, and is often difficult to find, because covered up by the enlarged lung. The breathing is rapid. There may be retraction of the lower intercostal spaces and the upper abdomen instead of elevation during inspiration, because of failure of the diaphragm to descend. Vocal fremitus is diminished, while the natural resiliency of the chest walls is substituted by increased resistance.

Percussion recognizes resonance exaggerated in various degrees, sometimes amounting almost to tympany, the result of the over-distention of the air vesicles, whose elasticity is spent. Vocal resonance is decreased because of the diminished vibration in the air columns. Feeble crackling is said to be sometimes heard. Strümpell says the vesicular murmur is at times exaggerated and "shuffling," at others "rougher and more indefinite." Roughness and exaggeration seem impossible in true emphysematous areas. It may be in adjacent supplementally acting areas. If bronchitis is present, its sounds are associated and often obscure all else. The pulmonary second sound at the second left interspace is accentuated on account of the hypertrophy of the right ventricle, but the heart sounds are usually obscured by the extra covering of the lung. With dilatation of the right ventricle, which sooner or later succeeds, the increased accentuation disappears.

Interlobular emphysema, in which the connective tissue between the lobules is infiltrated with air as the result of rupture of air vesicles from

violent acts of coughing or of wounds of the lung, affords no physical signs, rarely any symptoms. The shape of the chest in such cases is not altered. Suddenness of onset is characteristic of this form of emphysema, and it is apt to be associated with a similar infiltration of the tissues of the neck, which gives rise to a very distinctive crepitation on palpation.

Diagnosis.—This is not usually difficult, at least in true symmetrical emphysema. In pneumothorax there is some simulation of the symptoms of emphysema. There is the same shortness of breath and there is some resemblance in the physical signs. There is bulging of the chest, which is, however, more marked on one side than on the other. On the other hand, it is rather rare to have a uniform, symmetrical emphysema, and we may have here also a greater prominence of one side than of the other. Thus the parts of the chest more likely to be affected with emphysema are the upper part of the right lung and the lower part of the left. Also in the matter of percussion, both emphysema and pneumothorax give hyper-resonance. Pneumothorax, however, gives more marked tympany than emphysema. It has always seemed to me that one gets an exaggerated idea of the clearness of resonance found in emphysema from reading the books. The hyper-resonance, although often marked, is not always so. The unyielding chest walls modify it. The hyper-resonance of pneumothorax is a real tympany, comparable to that obtained over the distended abdomen. In both the thickness of the chest walls exerts a modifying effect. Metallic tinkling is a distinctive sign of pneumothorax, caused by the dropping of fluid from the perforated lung into the air-resounding pleural sac. In the lower portion of the chest with pneumothorax there is always effusion, which gives dulness on percussion, and a line of separation between tympany and flatness is demonstrable. Pneumothorax is sudden in its occurrence, whereas emphysema develops gradually. It is, however, not impossible for the two affections to be combined.

There is still another condition with which emphysema may be confounded, though of rare occurrence; I refer to diaphragmatic hernia, in which tympanitic resonance is a striking symptom. I had under my observation for a long time a very singular case, in which there had been a sudden development of symptoms. The condition came on suddenly, while the patient, a man, was engaged in a scrimmage or wrestle. He was seized with a sudden sharp pain in his left side, and a day or two later began to have a peculiar puffing respiration. With this there was extraordinary clearness on percussion over the region of the left lung. He was examined repeatedly by myself and others, but no one thought of the true cause. None suspected diaphragmatic hernia. Acute emphysema was thought of. The autopsy showed that almost one-half of the abdominal viscera was in the pleural cavity, and that the lung was pushed into the upper portion of the chest, occupying a space about the size of the fist. This man presented the bulging, the shortness of breath, and the hyper-resonance peculiar to emphysema.

The skodaic hyper-resonance—resonance above a pleural effusion—also resembles that of emphysema, but attention to the history and other physical signs will discover the true cause of the resonance.

Prognosis.—This, except in cases of acute emphysema, which heals spontaneously, is unfavorable as to cure. The course is, however, always a chronic one and much may be done for the comfort of the patient. No classes of cases are so benefited by admission into hospital as members of the laboring class suffering with emphysema.

Treatment.—It is impossible, so far as we know, to restore destroyed lung texture. If a number of air vesicles have been converted into one sac or bladder-like cavity, there are no means by which these vesicles can be restored. At the same time, where the patient is young, there is some hope of cure if the structural loss is not too great. Effort must be directed mainly to averting those conditions which complicate and increase the emphysema. As I have said, chronic bronchitis is its most frequent cause, and, therefore, we must try to relieve this condition by every means in our power. As the general health is often impaired, it is as important that this should be reëstablished as that the bronchitis should be relieved. The blood is to be restored to a proper composition by tonic remedies, like cod-liver oil and iron, and the use of the very best food that the patient can procure. To the cod-liver oil and iron should be added strychnine in full doses, $\frac{1}{30}$ to $\frac{1}{24}$ grain (0.0022 to 0.0027 gm.), while arsenic is an admirable tonic either in the shape of Fowler's solution, five drops at a dose for an adult, or of arsenious acid, $\frac{1}{30}$ grain (0.0022 gm.).

While the bronchitis is treated by the usual remedies, it is of the utmost importance that the stomach should be kept in good condition, and that digestion should not be interfered with, while more than ordinary care is required in the selection of remedies for the bronchitis. A very useful measure in these cases is counter-irritation, which in no way interferes with digestion. This may be applied in various ways: blisters, iodine, and mustard may be used. A mustard-plaster can be so prepared that it may be worn continuously without discomfort—taking mustard and flour in the proportion of one to five, and using equal parts of the white of an egg and glycerine with which to mix instead of water.

Strychnine is an admirable remedy not only as a tonic, but it may also be regarded as an expectorant, and secretions in the lungs are often disposed of by its use. It has also the effect of improving the nutrition of the muscular tissue of the walls of the bronchi, as it has of improving the muscular tissue in general. Full doses should be given, not less than $\frac{1}{32}$ grain (0.002 gm.) three times a day, increased gradually to $\frac{1}{12}$ (0.0055 gm.). This is to be kept up for a long time.

Bronchial asthma is one of the most serious and frequent complications and often overshadows all else. There is no more efficient means of breaking up such an attack than the hypodermic injection of $\frac{1}{4}$ grain (0.0165 gm.) of morphine with $\frac{1}{120}$ grain (0.00055 gm.) of atropine. This will usually relieve the paroxysm almost immediately. If relief is not complete, the injection may be repeated and renewed every six hours. The various inhalations employed for asthma may be employed, such as the smoke of burning stramonium or tobacco, ether, chloroform, and amyl nitrite. Of course, in connection with the attacks of asthma, the other remedies of service in relaxing spasm, such as

belladonna and iodide of potassium, may be given. Tincture of belladonna in doses of five to seven minims (0.33 to 0.462 gm.) combined with ten grains (0.66 gm.) of iodide of potassium will break up so much of this condition as is due to spasmodic contraction of the tubules.

To relieve the constant dyspnœa, the treatment suggested some years ago by Waldenburg is one the usefulness of which is only limited by its relative difficulty in application and the costliness of the necessary apparatus. It consists in the inspiration of compressed air and the expiring into rarefied air. It is plain that if compressed air can be introduced into the vesicles the aëration of the blood will be more perfect, and that if the patient breathe into rarefied air the residual air which it is so difficult to get rid of will be more effectually sucked out. The compressed-air chamber has a similar purpose.

Expiration may also be aided by compression of the chest, intermittently applied so as to coincide with natural breathing. This must usually be practised by a nurse or attendant, but Strümpell describes in his text-book a simple contrivance devised by a patient of his own for self-treatment. It consists of two boards fastened behind and allowed to project forward on each side in front, so that the patient himself, taking hold of the projecting ends, can compress his own chest with each act of expiration.

CROUPOUS PNEUMONIA.

SYNONYMS.—*Pneumonitis; Lobar Pneumonia; Fibrinous Pneumonia; Genuine Pneumonia.*

Definition.—An acute infectious inflammatory disease of the lungs, characterized by high fever, and terminating by crisis in from five to nine days. A bacterium especially prone to occur in pairs or chains, known as the *diplococcus pneumoniæ*, is found in 75 per cent. of all cases of lobar pneumonia and is commonly regarded as its cause.

The term lobar pneumonia is used for this form because it generally involves at least a single lobe or the greater portion of one. The term pneumonia of the apex is used where one or more of the apices of the lung is involved. A rare form of pneumonia is double pneumonia in which both lungs are involved, though not necessarily the whole of each lung. A massive pneumonia is an inflammation not only of the air vesicles but of the bronchi of a lobe or even of the whole lung. A creeping or migratory pneumonia affects successively different lobes of the lung. Epidemic pneumonia involves large numbers or communities and seems to be contagious. The term *larval* pneumonia is applied to a form of the disease in which but a partial development of symptoms occurs, such as a moderate chill, slight fever, and imperfect local signs. It occurs more particularly in connection with epidemics or with pneumonias in crowded places, as ships, camps, and garrisons.

Historical.—Evidently what we now know as croupous pneumonia was known to the earliest medical writers, including Hippocrates (B. C. 460–357), who, with others, described it with considerable accuracy as *peri-pneumonia*, or *pleuritis*. Hippocrates said of it that it was a "disease quickly fatal, and characterized by sputa of various colors." Lesions

and symptoms corresponding to it were described by Thucydides in his description of the "Plague of Athens," B. C. 430. Sydenham (1670), Valsalva (1666–1723), Morgagni (1761), Boerhaave (1668–1738), all gave good decriptions, but failed to separate it from pleurisy. Laennec (1819) was the first to sharply separate the two diseases, and made the classification into the three well-known stages, which hold to-day as then,—congestion, hepatization, and resolution or suppuration. The nature of the exudate was first accurately described from the macroscopic stand-point by Rokitansky in 1841. Ziemssen (1857–1858) furnished valuable data on the geographical distribution of pneumonia. Grisolle (1864) especially valuable statistics relating to climate development and comparative frequency among different races.

The infectious nature of pneumonia was first advocated by Jürgensen in 1872. The presence of a special organism in the secretions of hepatized lung, in the fibrinous exudate into the alveoli, and in the sputum was demonstrated by Friedlander in 1883, and again by Fraenkel in 1886. The organism was called *pneumococcus* by Friedlander and *diplococcus* by Fraenkel. The two organisms are not identical. The *pneumococcus* is a short, oval bacillus, always enclosed in a capsule, which usually contains one coccus only, rarely two or more. It is non-motile and anaërobic, that is, grows without oxygen. When treated with the aqueous staining solutions (as carbol-fuchsin) the bacillus is stained, the capsule being only slightly colored. It cannot be stained by Gram's method. The cocci do not liquefy gelatine, and stick cultures develop into a nail-like growth with a thick head. The cocci flourish in agar and on the potato. They are found in 5½ per cent. of cases of pneumonia.*

The *diplococcus* of Fraenkel occurs in pairs, sometimes in rows or beads ; like the pneumococcus, it is encapsulated in the body, but not in cultures out of the body. It is also a bacillus pointed at one end, whence the term *bacillus lanceolatus*, "lancet-shaped." It differs from the pneumococcus in that when stained by the carbol fuchsin solution the coccus is intensely red, while the capsule assumes a light reddish tint. Further, it can also be stained by Gram's method, while the pneumococcus of Friedlander cannot. It thrives on agar, in bouillon, but not on gelatine. It is thought to be the same organism as that found by Pasteur and Sternberg as early as 1880 in the saliva, and occurs, according to Netter, in 20 per cent. of all persons. Fraenkel, Telamon, and especially Weichselbaum showed the relations of this organism to pneumonia. The latter found it in 92 per cent. of cases of croupous pneumonia. William H. Welch found it in every one of ten cases of croupous pneumonia studied at the Johns Hopkins Hospital at Baltimore. It has been found in the blood, in the spleen and kidney, in endocardial vegetations, and in the pus of cerebro-spinal meningitis, where there was no pneumonia, as well as in the saliva of healthy persons and in the dust on the floors of houses. Its route of entrance is probably the respiratory passages, since it has been found in the nose, larynx, and Eustachian tube, and is said to persist for months and even years in the saliva of healthy per-

* The opinion at present appears to be that the bacillus of Friedlander is a feeble pathogenic organism, a harmless saprophyte, as a rule, but able, at times, to produce inflammatory effects.

sons who have had pneumonia. On the other hand, it is a very perishable organism, maintaining its virulence for four or five days only out of the body.

That processes closely allied to pneumonia are induced by the introduction of the pneumococcus of Fraenkel into the lungs seems to have been conclusively shown. That it is not the only organism capable of producing pneumonia is, however, evident from the experiments of Fraenkel himself and Weichselbaum, as well as from those of Pansini and Neumann. It may be accompanied by pus organisms and others, which may be responsible for complications and modifications of the ordinary pneumonic process.

Thus *caused*, pneumonia may be regarded from two standpoints. First, it may be a general disease with a local expression in the lungs, analogous to the inflammation of Peyer's patches in typhoid fever; or it may be a local disease, which, like diphtheria, infects the general economy and produces the constitutional symptoms characteristic of it. As in the case of typhoid, there were facts which pointed to the infectious nature of pneumonia long before the discovery of any organism which could be regarded as its specific cause. The occurrence of pneumonia in epidemic form was recognized by Laennec and Grisolle, and since their day innumerable epidemics have been described: house epidemics, including those in which a number of individuals, from three to ten or more, have been attacked under the same roof, and general epidemics, invading institutions, ships, and garrisons, in which large numbers of persons are congregated. Out of a ship's crew of 815, 410 have been attacked in rapid succession, and out of 720 attacked, 298 fell victims.

While the state of knowledge at the present day seems to demand that we consider croupous pneumonia as an infectious disease due to the action of a specific organism, we cannot ignore the operation of causes, such as dampness and cold, which until recently have seemed sufficient to account for a large number of cases. Thus an overworked man is exposed to cold for a long time, and becomes thoroughly *chilled*. A few hours later he is seized with a rigor, and twenty-four hours afterward the physical signs of a pneumonia have developed. The lowered vitality consequent to the exposure in each case must be regarded as a predisposing cause, preparing the system for the operation of the ever-present organism as the exciting cause. The operation of cold is further seen in the influence of the seasons, pneumonia being much commoner in the winter months. Other predisposing causes are—a previous attack, fatigue of mind or body, and debilitating conditions of all kinds, such as previous or present illness, especially chronic disease like Bright's disease. A patient of my own had four attacks and succumbed to a fifth. Heredity is also said to be a factor, and injuries of the chest have long been regarded as predisposing causes.

Morbid Anatomy.—The lung in croupous pneumonia exhibits three distinct stages:—

(1) Congestion or engorgement.
(2) Red hepatization, and
(3) Grey hepatization.

Pneumonia seeks, by preference, the lower lobes of the lungs, and the right lung more than the left. Pneumonia of the apex does, however, occasionally occur, and more frequently in children than in adults.

The Stage of Congestion.—In this stage the lung is engorged with blood, yet permeable to air. The capillaries surrounding the air vesicles are turgid and intrude upon the lumina of the air vesicles. There is a small amount of transudate, in which may be found a few exfoliated alveolar cells and red blood discs. The part of the lung invaded is redder than normal and heavier, but not nearly so heavy as in the next stage. On section blood transudes from the cut vessels and bathes the surface.

The Stage of Red Hepatization.—In this the lung is dark red in color, hard, and very much heavier than in health, as much as three and four times that of the normal weight. A piece dropped in water rapidly falls to the bottom. The lung pits on pressure, and in consequence the marks of the ribs are often seen on it after removal. On section the aptness of the name, red hepatization, is at once apparent. The surface is darker in color than in the first stage, and it has the appearance of a section of liver. On passing the finger over it innumerable little hard spots like grains of sand are felt. These are air vesicles filled with the croupous exudate. Corresponding to this a granular appearance is recognized by the eye, the distended air vesicles appearing as glistening points. By scraping, little plugs of fibrin and cellular detritus mixed with serum can be removed. The lung, though thus hard, is nevertheless friable, and may be broken up by the fingers.

Histologically the air vesicles are found to contain a delicate reticulum, the meshes of which are filled with red blood discs and alveolar cells in different stages of degeneration, including numerous granular fatty cells or compound granular cells. The vesicular walls are found infiltrated with lymphoid cells, which extend even into the interlobular tissue beyond. Plugs of fibrin may sometimes be traced into the smaller bronchi from the air vesicles.

The diplococcus and pneumococcus may be demonstrated in cover-glass preparations made from the exudate. They may be associated with the streptococcus and staphylococcus.

The Stage of Grey Hepatization.—This is also well named, the cut lung exhibiting a greyish-white coloration. It is still dense and heavy, but much moister and softer, and even more friable. The granulations are, however, less distinct, and on microscopic examination the alveoli are found filled with white blood-corpuscles, while the red corpuscles and fibrin filaments have disappeared. Sometimes all three stages occur alongside of each other.

A stage beyond grey hepatization is sometimes spoken of as a stage of yellow hepatization. In this stage the lung has assumed a more yellow appearance, it is much softer, almost liquid in consistence, and more like pus. On minute examination the air vesicles are filled with pus cells, the points of greatest softness constituting small abscesses as large as a pin's head and larger. The stage of grey hepatization is the stage of beginning resolution, while that of yellow hepatization is probably one whence recovery is impossible, as it is only seen post mortem.

If recovery takes place the contents of the air vesicles liquefy, part being expectorated, but probably the most is absorbed.

The pleura adjacent to the inflamed lung is almost always inflamed, the most distinctive sign of this being a plastic deposit. There may also be thickening and some serous effusion.

After death from pneumonia, the heart is found in a typical pathological condition. The left cavities are generally found empty or nearly so, while the right are distended with firm coagula which often extend into the branches of the pulmonary artery. The spleen is often enlarged. The cells lining the renal tubes are often found in a state of cloudy swelling; rarely there is nephritis.

Symptoms.—Perhaps no other disease except malarial fever is so invariably ushered in by a *chill* as is croupous pneumonia, and often a chill of great severity. It may come on at night, waking the patient out of a deep sleep. It may or may not be preceded by a day or two of *prodromal discomfort*, with *headache*, which may be very severe. Almost immediately there succeeds a high fever in which the temperature rises rapidly to 103° to 105° F. (39.4° to 40.5° C.). A significant *flush in each cheek* is characteristic, occasionally more marked on the affected side. The pulse is full and strong, resisting pressure, 100 to 120. There is *thirst, scanty and high-colored urine.* Equally promptly ensues a *pain in the side*, which may be dull, but which is often also sharp and severe, caused in the latter instance by involvement of the pleura. The respirations rise rapidly in frequency, and there is *cough*, at first dry and hard. It is often restrained on account of the pain it occasions. Soon there is a small amount of *mucous expectoration* from the coincident bronchitis, but usually in twenty-four hours after the chill the sputum exhibits distinctive characteristics. It is tenacious, dark red in hue—"prune-juice" expectoration it is called, and is ejected from the mouth with difficulty. The amount of blood and the degree of coloration varies greatly. The respirations are exceedingly rapid, 50, 60, and even more in the minute. I have known them to be 82, and in a child they may reach 100. The appearance of a patient at this stage is a very striking one. The face is flushed, the eye is brilliant, the breath is rapid, the alæ nasi move with each breath, while a frequent short cough, held back until irresistible, increases at times the already anxious expression of the patient.

This state of affairs continues unchanged for from five to nine days, when, if recovery takes place, a sudden drop in the temperature occurs, accompanied often by free perspiration, while a state of comparative comfort succeeds to one of great distress, and it may be followed by a long and refreshing sleep. This is known as the *crisis.* It may be preceded by a fall of temperature a day or two earlier, which is again followed by a rise, whence such fall is called the pseudo-crisis. The accompanying temperature chart, from a case seen in consultation with Alfred Stengel, illustrates the actual crisis. The fall in crisis is sometimes as much as 7° F. (13.9° C.) in a single twenty-four hours, and the minimum is quite often slightly subnormal, whence it rises rapidly to the normal.

From this point onward convalescence is rapid, and in four to five days more the patient is seemingly well, the temperature and pulse rate

normal, the breathing natural. A muscular weakness and vulnerability, however, remain which demand care for a time longer.

Physical Signs.—The physical signs of a typical pneumonia are very distinctive.

The *first*, or *stage of congestion*, in which the air vesicles are still open, is of short duration, terminating within the first twenty-four hours, and may therefore be overlooked. *Inspection* shows the face flushed, increased frequency of respiration, with restricted movement upon the affected side and increased motion on the sound side. The patient lies by preference on the affected side because of the greater comfort it gives him. This posture not only diminishes the pain by hindering the

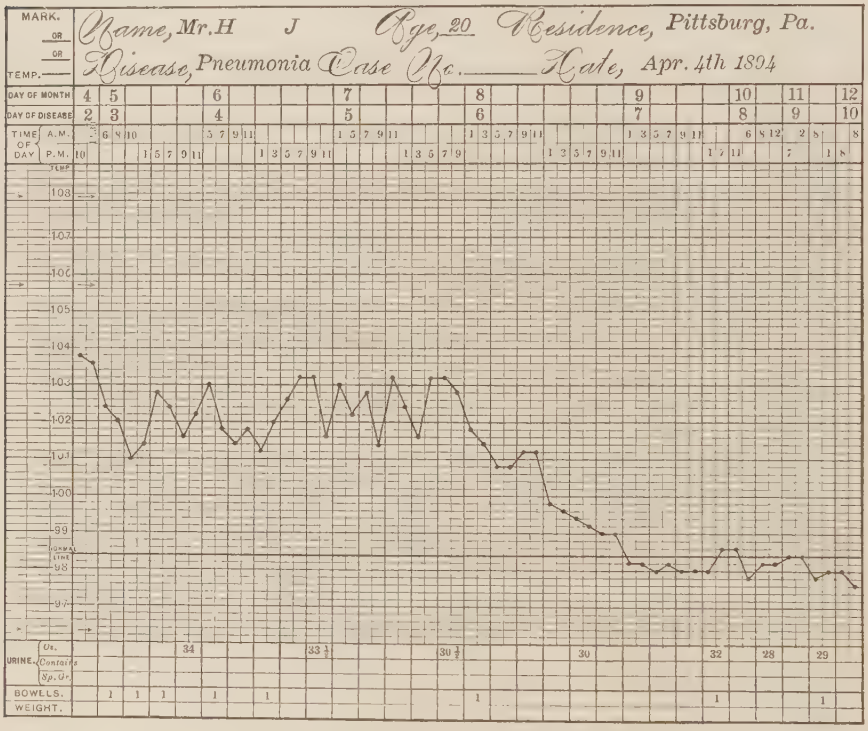


FIG. 34.—SHOWING CRISIS IN PNEUMONIA.

motion of the affected side, but also lessens the dyspnœa by permitting unrestrained expansion of the other side which is doing the work.

Palpation at first may even find vocal fremitus diminished on account of the relaxation of the air vesicles, but it becomes decidedly increased as the air vesicles fill up. The skin is hot and the pulse is frequent, full, and strong, as a rule. *Percussion* obtains but slight if any impairment of resonance. In fact, tympany, or the vesiculo-tympany of Flint—Skoda's resonance—is frequently present in this stage as the result of the relaxation of the partially filled air vesicles, giving resonance by immediate relaxation. In the latter part of the first stage there is, however, impairment of resonance.

Auscultation in the very earliest stage may find the vesicular murmur feeble, but very soon is heard the distinctive physical sign of pneumonia, the *crepitant râle* at the end of inspiration. If there be coincident pleurisy—pleuro-pneumonia—the closely simulating friction sound may be added. Under such circumstances it might be difficult to distinguish these two physical signs. Over the normal part of the lung there is exaggerated vesicular breathing.

But all these physical signs, even if carefully sought for, may be wanting if the pneumonia is central and deep-seated, as is not infrequently the case. They appear as the surface is approached, or they may not be recognized at all if the disease remains central.

The *second* stage, or *stage of red hepatization* or solidification, lasting four or five days, furnishes unmistakable signs. All the signs pneumonia reveals to *inspection* in the first stage are intensified in the second, and the breathing is markedly abdominal. To *palpation*, vocal fremitus is now intense, the skin is hot and dry, and the pulse continues frequent. Mensuration almost always and even inspection may recognize an enlargement of the involved side, the former to the extent of 0.5 to 2.5 cm.

Percussion gives absolute flatness over the solidified area, with high pitch and short duration, except in those very rare instances where the extreme consolidation throws the column of air in the trachea and bronchi into vibration, producing tympany. This explanation is perhaps the only one when it occurs in the upper lobe. In a lower lobe tympany may result in the same way, from the proximity of an air-distended stomach. Over the adjacent normal areas, also, resonance is exaggerated, not so much, perhaps, in consequence of supplemental function, as from relaxation of the adjacent air vesicles—Skoda's resonance by mediate relaxation. Even cracked-pot sound may be produced by percussion over the solidified lung as the result of the sudden expulsion of air from a large bronchus leading to the solidified area.

Auscultation discerns high-pitched bronchial breathing over the solidified lung. Indeed, these are the circumstances which give the typical bronchial or tubal breathing. The air vesicles are obliterated, and the resulting excellent conducting medium bring the tracheo-bronchial blowing to the ear. In very rare instances, where the larger bronchi are filled with exudate, there may be no bronchial breathing. The ausculted voice gives us typical bronchophony and occasionally even pectoriloquy, as well as whispering bronchophony and pectoriloquy. The heart sounds are also heard with great distinctness over the consolidated lung, owing to the improved conduction, while the sounds of a concurrent bronchitis are similarly intensified. A lingering crepitant râle may also be heard.

The *third* stage, or *stage of grey hepatization* or resolution, occupies six to ten days. It repeats largely, to inspection, palpation, and auscultation, the phenomena of the first. Resonance continues impaired for some time. The normal manner of breathing gradually returns, the temperature of the skin is notably less, the crepitant râle returns, technically known as the "crepitans redux," and is finally replaced by the normal vesicular breathing sound, by which time the dulness has disappeared.

Croupous pneumonia may rarely terminate in abscess or gangrene, when the signs of the second stage continue, the temperature does not fall, in a word, the crisis does not occur. The signs of a cavity, which might naturally be expected, are rarely present, and it is rather by the general symptoms, the failure to recover, the continued high temperature, the expectoration of pus, and, in the case of gangrene, the intensely disagreeable odor, that we are informed of the issue. These issues probably represent on a large scale what takes place in every instance in minute areas in the third stage of all pneumonias which terminate favorably. The occasional termination in tubercular phthisis exhibits a similar arrest of the resolving process in the second stage, and the phenomena of catarrhal or fibroid phthisis supervene.

The obscuring effect of a thickened pleura upon all of these signs is to be remembered, and too much stress cannot be laid upon the fact that we may have a central deep-seated pneumonia which may give no physical signs; also that in old persons the physical signs of a pneumonia are very apt to be delayed from one to three days.

Careful differential percussion and palpation may recognize a moderate enlargement of the *spleen*.

The *heart* should be carefully watched in pneumonia. The sounds, at first loud and clear, become less so as the disease progresses and the lungs become engorged. The pulmonic second sound is particularly sharp as long as the heart is strong, and its failure is an unfavorable sign, as it means that the right ventricle is failing in power and may be yielding to distention.

Special Symptoms.—The above is the course of a typical case of pneumonia, perhaps of three-fourths of all cases, and the symptoms mentioned suffice for a diagnosis. All of them are, however, subject to modifications.

Thus the chill may be absent or imperfectly developed, in which case all the symptoms arise more gradually. The temperature, especially in old persons and drunkards, may not be nearly as high, in children it may be higher. The same is true of the respirations, which may be increased to 100 to the minute in children. Pain is especially absent in old persons, cough and expectoration also, so that a careful physical examination of the lungs should be made in all ailments in the old and in drunkards also, as it not infrequently happens that pneumonia is overlooked in them. The pulse is often feeble and rapid instead of full and strong. Nay, more, even the physical signs may be absent in the old, and they are especially apt to be delayed in their development. It is unsafe to say of an old person at the first visit, after a negative physical examination, that he has not pneumonia, for the physical signs may not make their appearance until the second or third. It would seem, too, that central pneumonia is more common in the old than in the young, while even an afebrile pneumonia is a possibility in the old.

The expectoration varies a good deal when present, especially as to the quantity of blood. Sometimes it is bright red and quite liquid, almost like a hemorrhage. More frequently it is viscid and glutinous, simply stained with blood. The term "prune-juice" expectoration has long been associated with pneumonia, and sometimes when it is thin and

dark-hued the comparison is an apt one. Under the microscope the sputum is found to contain blood discs, leucocytes, alveolar epithelium in various stages of degeneration, including numerous compound granule-cells, also ciliated epithelium. Fibrinous bronchial coagula, sometimes large enough to be seen by the naked eye, are also met with, and, after suitable staining, diplococci. Should gangrene supervene the expectoration becomes very fetid.

The *urine* is especially characterized by a reduced amount of chlorides, which are often absent until the crisis is passed, when they reappear. It is supposed that during this period they are transferred to the exudate in the lungs. A trace of albumin is often present and it presents the other features of a febrile urine.

There is sometimes *marked jaundice.* It may even be the first symptom. It may be a catarrhal or hæmatogenous jaundice. The cases attended by it are rather more serious.

The *blood* exhibits usually a *leucocytosis*, the number of corpuscles being increased from 6000 per cubic millimeter to 19,000. The proportion of fibrin is also increased from four to ten parts in 1000. This increase of fibrin shows itself also on the microscopic slide in the shape of filaments of fibrin. According to Hayem the blood plaques are increased.

Herpes is very common on the lip, present, it is said, in from 12 to 40 per cent. of all cases. It may occur elsewhere, as on the nose and genitals.

In *typhoid cases* the tongue is coated, becomes dry and leathery. Constipation is usual, but occasionally there is diarrhœa, especially in epidemics. Except in typhoid cases *delirium* is not common, but may be very active in the young. In old persons it may be low and muttering. In drunkards, in whom the disease is common and very grave, especially after a debauch, the delirium may be taken for *mania a potu*, or the two may co-exist. Such a patient may rise from his bed and wander out into the city or to another hospital which he prefers, having just intelligence and strength enough to accomplish this purpose, and will die after its attainment.

Terminations.—When the pneumonia terminates favorably, promptly after the crisis is passed it is said to terminate by resolution, by which is meant that the inflammatory product liquefies, is absorbed or expectorated, and the lung resumes its natural state and normal physical features. The time at which these events are thoroughly established varies greatly, and if there happens to have been associated pleurisy with resulting thickened membrane, impairment of resonance may last a long while. This promptly favorable termination does not always take place. Resolution may be unduly delayed and yet ultimately take place. Such cases naturally occasion anxiety, for resolution may not take place at all, in which event one of four unfavorable terminations may occur. These are :—

(1) Abscess.
(2) Gangrene of the lung.
(3) Interstitial or fibroid pneumonia, and
(4) Tubercular phthisis.

(1) Abscess of the lung is a termination of pneumonia in about four per cent. of fatal cases. Flint, Sr., found it in four out of 133 cases recorded. When this occurs the interstitial tissue of the lungs becomes infiltrated with pus cells, small foci of leucocytes aggregate to form larger, until a large abscess results, which may occupy a whole lobe or even whole lung. In such cases the fever continues high, there is expectoration of pus containing elastic tissue of the lung, and the physical signs of a cavity may rarely be present. It is not impossible, however, for such a process to be arrested by a reactive inflammation, by which a tough protective layer of embryonic tissue is formed about the abscess.

(2) Gangrene of the lung occurs in about three per cent. of fatal cases. It is especially prone to occur where the pulmonary vessels become so engorged that the circulation is arrested, and where as a consequence the hemorrhagic element is conspicuous. Bronchiectatic cavities in an inflamed lobe which are swarming with putrefactive bacteria are an important predisposing cause. It is recognized by the horrid fetor, which pervades a whole ward, and which once met is never forgotten. The expectoration is thin and similarly fetid, and contains large quantities of elastic tissue from the lung. The lung is converted into a grey-green, fetid pulp, in which cavities with ragged walls arise from disintegration and expectoration of lung tissue. Gangrenous portions may be surrounded by a zone of true inflammation, contrasting by its red color with the grey of the gangrene. Such sloughs have been successfully excavated by surgical treatment.

(3) In fibroid induration or cirrhosis, which is occasionally met with, there is also invasion of interstitial lung tissue, but instead of being infiltrated by such an excess of leucocytes as to produce pus, only as many wander out as can undergo organization and conversion into permanent tissue. Sometimes this results from the lung failing to expand after resolution and absorption of the exudate, the walls of the unexpanded alveoli collapsing and uniting. In other cases there is partial absorption of the exudate, repeated extravasations take place into the alveolar septa, and organization takes place in both. The fibrinous plugs may also be transformed into connective tissue. Three successive stages may be present. In the first the cirrhotic patches are grey, greyish-red, or greyish-yellow, and a small amount of turbid exudate can be here and there squeezed out of them. In the second stage, where the formation of the fibrous tissue in the alveoli or their walls has set in, the lung is dense, firm, airless, and fleshy, whence the term *carnification*. In the third stage the fibroid transformation is complete ; the tissue is tough and slate-grey in color. Such induration is generally in bands and patches which merge gradually into the normal vesicular structure.

(4) Tubercular phthisis is another termination of pneumonia. It results from infection by implantation of the tubercle bacillus. Pneumonia of the apex terminates thus most frequently.

Complications.—The most frequent complication is *pleurisy*. It is probably always present to a certain extent, except in the central forms. It manifests itself in the first stage more by the characteristic severe cutting pain than by physical signs, as the friction sound characteristic of that stage is commonly obscured by the physical signs of the

pneumonia. Should the stage of effusion be reached, the physical signs of the pneumonia subside. A pneumonia on one side and a pleurisy on the other is a possibility. That very interesting pathological state known as pleurogenic pneumonia is sometimes seen in the human being as a form of tubercular pleurisy. In it the lung becomes partitioned off by an interstitial framework starting from the pleura. It has its typical anatomical product in the pleuro-pneumonia of cattle. The extension takes place chiefly by way of the lymphatics.

Endocarditis is a comparatively frequent complication. William Osler especially called attention to this fact in his Gulstonian lectures for 1885. He ascertained that of 209 cases of malignant endocarditis 54, or over 2½ per cent., occurred as complications of pneumonia. It is more prone to attack persons with old valvular disease, and to involve the left heart. There is good reason to believe that the specific lancet-shaped bacillus is responsible for this form of valvulitis, as a complication of pneumonia. The endocarditis constantly escapes detection, since physical signs are sometimes absent, at others deceptive, but it may be suspected—

(1) When the fever is protracted and irregular.

(2) When signs of a septic condition arise, such as irregular temperature with chills and sweats.

(3) When embolic pneumonia develops.

(4) When a loud, rough murmur, especially a diastolic aortic murmur, develops in the course of the disease.

Meningitis is another complication to which Osler has called especial attention, finding it in eight per cent. of fatal cases. It usually comes on at the height of the fever, and may be confounded with delirium. It is often associated with endocarditis and it may be accompanied by cerebral embolism, producing hemiplegia. *Neuritis* is a possible complication.

Diagnosis.—The diagnosis of a case of typical pneumonia is easy. The chill, the rapidly developed fever, and physical signs are, as a rule, easily recognized. It is to be remembered, however, that the physical signs may be delayed or not appear at all in the central varieties.

Pleurisy is the disease from which pneumonia has most frequently to be distinguished. The resemblance between the friction sound and the crepitant râle is often very close, while there is impaired resonance to percussion in both. Most valuable in diagnosis is vocal tactile fremitus, which is invariably increased in pneumonia and as invariably diminished in pleurisy of any variety. In the not very rare instances of pleurisy with effusion attended by bronchial breathing the same sign is pathognomonic, tactile fremitus being diminished, whereas it is increased in pneumonia. Commonly, too, in this stage of pleurisy, we have a change in the line of dulness as the patient changes position, though this is not invariable. The exploring needle, if needed, may also help settle that question.

Frequent examination of the lungs should be made in alcoholism, chronic valvular disease of the heart, in diabetes, and in Bright's disease, since all of these affections are prone to become complicated with insidious forms of pneumonia.

Typhoid fever and pneumonia are sometimes confounded. The former is apt to become associated with hypostatic congestion of the lungs and pneumonia with a typhoid state. The hypostasis, however, occurs late in typhoid fever, the dulness in pneumonia sets in early. A more excusable error is made in the case of *acute* tuberculo-pneumonic *phthisis*, which may begin with a chill, while the resemblance is otherwise very close, especially in physical signs. Microscopic examination of the sputum would recognize the bacilli of either disease. This should always be made where an apparent pneumonia is prolonged beyond two weeks without a crisis.

Prognosis.—Pneumonia is a treacherous and uncertain disease at any age. Young, robust men of twenty-five, taken mildly ill with every reasonable expectation of recovery, sometimes die suddenly and unexpectedly. On the other hand, while in the old and intemperate it is especially dangerous, old men and women over seventy often recover completely. The intemperate are less fortunate, yet even among them some surprising recoveries are met. It adds little to our power to cope with the disease to know that the mortality ranges from 20 to 40 per cent., or that about one in four or five die. Children recover often, even when desperately ill. The disease seems to be more fatal in cities than in the country, is certainly so during epidemics, whether house-epidemics or in ships or other crowded places.

The seriousness of an attack varies more or less with the extent of lung involved, pneumonia of a whole lung being more dangerous than that of a part, double pneumonia than that affecting one lung, while massive pneumonias are always fatal. Meningitis is invariably fatal, but its presence must not be inferred from every violent delirium. Endocarditis is almost as fatal. Death is usually by heart failure, the right ventricle becoming stretched by the accumulated blood, and the valves and columnæ carneæ embarrassed by fibrinous coagula, which may extend from auricle to ventricle and even into the branches of the pulmonary artery.

The conclusion is apparently unjustified by a study of statistics,* but it does seem to me that pneumonia is a more fatal disease now than when I began practice thirty years ago.

Treatment.—A fundamental principle which experience has established is that no single plan of treatment dare be recommended for pneumonia, but that each case is a law unto itself. This is more or less true of all diseases, but it is especially so of pneumonia. Undoubtedly cases occur which are best treated by general blood-letting, while many more do not require it, and a few may be harmed by it. Pneumonia may be a general disease and not a local one, and the lung involvement may be secondary, at the same time the patient often dies from the direct effect of such local involvement. It is the obstruction to the movement of the blood through the lungs which strains and wears out the right heart. The blood-letting lessens this congestion, if it is done early, and thus relieves the right heart. What are the indications for blood-

* For an excellent analytical examination of the statistics of pneumonia the reader is referred to a recent paper in the Medical News, July 27, 1889, by Drs. Townsend and Coolidge, Jr., based on a study of the cases treated in the Massachusetts General Hospital.

letting? They are found, as a rule, only in the first stage and first part of the second stage and include, first, great dyspnœa, second, full, bounding pulse, and, third, sharp, pleuritic pain. The relief to all of these symptoms is often magical. The amount of blood taken at such time should not be less than 20 ounces (600 c. c.), but not the quantity of blood so much as the relief to the symptoms should be the sign to stop the bleeding. The same results may be accomplished by wet cups, provided a sufficient amount of blood is taken, and cupping has the appearance of being less formidable, although it is actually more painful and disturbing to the patient. After the removal of the cups a poultice or warm cotton jacket is comforting. If doubt is entertained as to the propriety of either of these two methods of bleeding, the affected lung should be covered with dry cups and after the removal of these the hot-poultice or hot jacket applied. Even by this method the relief to the pain and dyspnœa is often very great, but it is more apt to be temporary. While there are cases in which the adynamia is so great as to make blood-letting in any form of doubtful propriety, there can be no possible objection to the dry cups. Bleeding, besides relieving the symptoms referred to, hastens the crisis and shortens the disease.

These measures may also relieve the cough, but usually something additional is required. Until expectoration sets in, opium is preëminently the remedy, and no preparation is so good as morphine in doses of from $\frac{1}{16}$ to $\frac{1}{12}$ grain (0.004 to 0.005 gm.) for adults every two hours in ½ ounce (15 c. c.) of the solution of citrate of potassium flavored with lemon or other syrup. Dover's powder in full doses sometimes acts nicely. It is best given in pill form. Expectorants are rarely needed at the outset, but ammonium chloride in doses of five to ten grains (0.32 to 0.65 gm.) in Brown mixture, also combined with morphine if necessary, will meet the indications. If a still more stimulating expectorant is required, the carbonate of ammonium may be used in doses of five to ten grains (0.32 to 0.65 gm.) frequently repeated. It is an important fact often overlooked in prescribing diffusible stimulants that to get a desired effect they should be frequently repeated, and it is better to give small doses often than large doses at longer intervals.

Pneumonia calls very soon, sometimes from the very outset, for alcoholic stimulants, which act not only on the heart, but also as antipyretics. Half an ounce or even an ounce (15 to 30 c. c.) hourly in cases of extreme adynamia may be necessary. The index of sufficiency or the reverse is the state of the pulse and heart. Whiskey or brandy, as selected, should be given in milk, which is the most suitable nourishment. Four to eight ounces (120 to 140 c. c.) every two hours containing the proper dose of stimulant may be given.

Strychnine is an invaluable heart tonic in pneumonia and may be given in doses of $\frac{1}{30}$ grain (0.002 gm.) or more every four to six hours, combined with eight to 20 grains (0.52 to 1.30 gm.) of quinine in the twenty-four hours, as may be required.

Digitalis is a remedy much used in pneumonia and it is a useful drug, but it is not always judiciously ordered. To whip up a flagging heart to increased effort, to drive blood through a lung almost as solid as a stone, is like whipping up a jaded horse to an effort beyond his strength,

and is about as ineffectual. On the other hand, such a stimulus may tide over an obstacle which is not insurmountable, but which might remain in the way unless removed. On the whole, I prefer to give digitalis in moderately full doses, five to ten minims (0.3 to 0.6 c. c.), as an adjuvant to alcohol rather than in very large doses. Occasionally in sudden adynamia very large doses, say a drachm (3.7 c. c.), hypodermically, may turn the tide toward recovery. Aromatic spirit of ammonia is an important adjuvant in straits like these, and may be substituted with advantage for the carbonate.

Inhalations of oxygen are of undoubted advantage in relieving the dyspnœa and thus comforting the patient. Whether it is curative I am unable to say.

High temperature may be reduced by sponging, though the temperature itself in pneumonia cannot be regarded as dangerous *per se.*

Should we ever blister in pneumonia? A blister in pneumonia sometimes does much good. It is especially useful in delayed resolution, late in the disease, where the crisis has been imperfect and convalescence does not set in. It may even take the place of a local or general bloodletting, especially when these have been deferred too long or are impossible from any cause. When a blister is applied let it be a good one. A large blister is no more painful than a small one, and neither is it as painful as is commonly supposed. In mild cases turpentine stupes may be sufficient to relieve pain.

I am well aware that pneumonia is regarded as a self-limited disease, reaching its crisis in from five to nine days, and that many think the only indication is to sustain the patient until this crisis is reached. In many cases this is true, but I believe that the fury of the disease may be diminished by treatment, and that a prompt bleeding at a suitable time will not only lessen the suffering and so spare the strength of the patient, but may also hasten the crisis. Another true stage at which a bloodletting is sometimes serviceable, is where the right heart from its ineffectual efforts to propel the blood through the lung becomes distended. Such a stage is indicated by intense cyanosis and gasping orthopnœa.

The use of veratrum viride is warmly recommended by some instead of bleeding in the earliest stages of the disease. It diminishes the force of the heart, furnishes a diverticulum for the excess of blood, and, as my colleague, Horatio C. Wood, says, "The patient is bled into his own circulation." I have never felt comfortable in relying upon it.

The treatment of pneumonia by *ice-cold applications* has lately been gaining favor. Its most ardent supporters in this country are Simon Baruch, of New York City, and Thomas J. Mays, of Philadelphia. My experience has not been large, but it has been such as to encourage me to continue it. I prefer the method recommended by Baruch, of enveloping the chest in a suitably fitted linen or muslin jacket (the ordinary cotton jacket answers well), wet in cold water; directing that the jacket be removed and substituted by the dry cotton jacket whenever the temperature falls to 100° F. (37.7° C.), and renewed if the temperature rises. In this way all danger is averted. Appended is the temperature chart of a case admitted to the University Hospital under my care, breathing at the rate of 58 a minute, and of whose recovery I had no expectation.

Dr. Mays prefers to surround the affected area with ice contained in bags which are wrapped in towels; but they are difficult to keep in place, especially when more than one bag is required, which is the case if more than a limited area is involved. He says, also, that if the temperature falls to the normal, or near it, with a tendency to remain there,

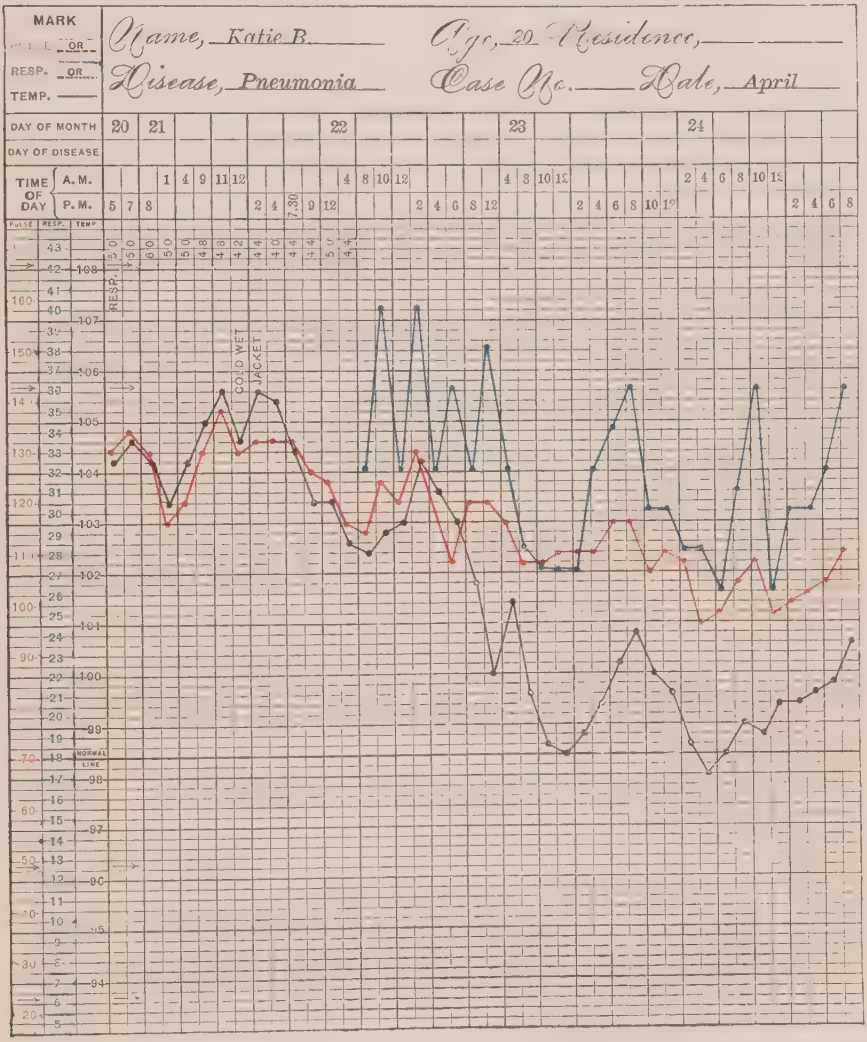


FIG. 35.—SHOWING DROP IN TEMPERATURE IN A CASE OF PNEUMONIA SUCCEEDING THE APPLICATION OF THE COLD WET JACKET.

The figures opposite RESP. in upper left indicate the breathing rate, which was too rapid on admission to be indicated in the usual way by the chart.

the ice is to be gradually removed. The fever is, of course, a mere index of the severity of the disease, and it is not for the direct effect on it that the ice is applied, but to arrest the process.

Serum Treatment.—Pneumonia was one of the first diseases whose treatment by serum engaged attention. The subject was studied by the brothers Klemperer, who utilized for prophylactic and curative purposes the antitoxine derived from the blood of immunized animals. Immunity is obtained by subcutaneous or intravenous injection of filtered bouillon cultures of the pneumococcus or of their glycerine extract. Such immunity is limited to six months, but the young born within this period are also immune, while the serum of the blood of such animals has the power to immunize other susceptible animals. Nay, more, these fluids when introduced into the blood of animals already infected were found capable of curing them. Thus, in such animals with a body temperature of from 104° to 106° F. (40° to 41° C.) the fever fell to normal in twenty-four hours after the injection of the serum. It is held by these experimenters that the pneumococcus produces a poisonous albumin or pneumotoxin, which when introduced into the circulation of an animal causes a rise of temperature and later an antipneumotoxin, which has the power of neutralizing the poisonous albumin formed by the bacteria. During the pneumonia the pneumotoxine produced by the bacteria in the lungs is constantly being absorbed into the circulation. In natural recovery this continues until enough antidotal substance is generated in the circulation to exert its effect when the crisis occurs. The bacteria are not destroyed nor is their power to produce poisonous products, but these latter are neutralized by the antitoxine, the presence of which has been demonstrated in the serum of the blood of pneumonia patients after the crisis. Klemperer first injected this serum into infected animals with the effect of curing them. Finally, he injected into persons ill with pneumonia the blood serum from others convalescent from pneumonia with a view to hastening the crisis. In six cases there was a decided reduction in temperature in from six to twelve hours after injection of from 65 to 95 minims (four to six c. c.) of the serum. The pulse and respirations also fell. The serum has no effect when injected into healthy individuals.

CHRONIC INTERSTITIAL PNEUMONIA.

Synonym.—*Cirrhosis of the Lung.*

Definition.—A chronic inflammatory disease consisting in a gradual invasion of a lung by fibroid tissue. According as it involves limited or more extensive areas it is local or diffuse.

Etiology.—There are few chronic affections of the lung which do not cause a certain amount of fibroid overgrowth. Especially is this true of tubercular consumption and broncho-pneumonia. A form of the latter is the so-called *pneumonicosis*, a fibroid induration succeeding a broncho-pneumonia due to the irritating effects of minute particles arising in the occupations of coal-mining, stone-cutting, steel-grinding, and iron-working in general. To the form associated with tuberculosis the term fibroid phthisis is applied, and it will receive separate consideration. The seat of a healed tuberculosis is also occupied by fibroid tissue, which may be regarded as an example of interstitial pneumonia. Less frequently it succeeds croupous pneumonia as fibroid induration, which has been con-

sidered on p. 440, and constitutes an important product in pleurogenic pneumonia, alluded to on p. 441. Even abscesses of the lung may excite it, while the various forms of morbid growths, as sarcoma, carcinoma, chondroma, and hydatid cysts, are causes of it and are surrounded by it. Especially does the fibroid change occur in a lung which has been long in a state of compression, as by a pleuritic effusion.

Morbid Anatomy.— *Pathological Histology.* — In broncho-pneumonia the fibrosis usually starts from the outer sheath of the bronchi, invading the alveolar walls and converting the entire lobule into greyish fibroid tissue, in which no lung structure is distinguishable. This form is frequently associated with dilated bronchi, of which it is probably the direct cause, its contraction drawing the walls apart. The line of demarcation between interstitial pneumonia on the one hand and tuberculosis on the other is often not very sharp.

In interstitial pneumonia after croupous pneumonia a gradual organization takes place of the fibrinous plugs in the air vesicles; the alveolar walls themselves become thickened by a new formation, at first cellular and subsequently fibrillated. Death usually occurs in these cases in one to three months after the onset of the disease. The whole of the part primarily invaded may become thus altered.

Macroscopic Morbid Anatomy.—The chest-walls of the side affected are often depressed, and on opening the thorax the lung, or as much of it as is involved, is found retracted; it may be drawn back into the spinal gutter. If on the left side the heart may be retracted with it. Commonly the two pleuræ are found united, but not always. On section the lung is hard and tough. It is grey, fibrous, and the alveolar structure has, to a varying extent, disappeared. The bronchi and the blood-vessels, however, remain, the former being often dilated to produce the so-called bronchiectatic cavity, of which there may be a number. In the phthisical variety there may also be a cavity at the apex, and this will be an aid to diagnosis. Otherwise a careful study is often necessary to distinguish the two varieties, unless the tubercle bacillus has been found.

The uninvolved lung is usually enlarged and emphysematous in proportion to the degree of contraction of the affected lung. The right ventricle, which has increased work imposed upon it in forcing the blood through the contracted lung, becomes hypertrophied and may become ultimately dilated. The pulmonary artery may be atheromatous.

Symptoms.—The principal symptom is *cough*, which starts with the condition causing the fibrosis and continues to the end. It varies greatly in its severity, being sometimes trifling, at others very troublesome. The *expectoration* is as variable as the cough, more copious as the cough is more troublesome. Persons thus affected have the appearance of delicate health and are commonly regarded as phthisical, although they have often considerable strength and can pursue some occupation. In true chronic interstitial pneumonia there is less *fever* than is present as a rule in phthisis but the recognition of the tubercle bacillus is the crucial test, for otherwise the symptoms are very similar. In both conditions there is paroxysmal cough with copious expectoration of muco-purulent matter. The resemblance is still more close if there is bronchiectasis, when the usual emptying of the cavity by cough takes place, commonly

in the morning, sometimes twice a day, and even oftener. The expectorated matter of the bronchiectatic cavities may be fetid from decomposition. There is usually less dyspnœa than in true phthisis, and except where the disease is the sequel of true pneumonia the fatal termination is longer deferred than in tuberculosis, it may be for years.

Physical Signs.—The chest is more or less retracted, its circumference diminished. Its movements are restricted and its topography altered. When the left lung is extensively affected a pulsation is often seen in the second, third, and fourth interspace, very similar to what is sometimes seen to the right of the sternum, when a pleuritic effusion on the left side pushes the heart over to the right. In high degrees of the disease the shoulder is drawn down and the spinal column laterally curved, just as in recovery after empyemic pleurisy. The unaffected side is more bulky than in health. The tactile fremitus may be diminished or increased according as the pleural membrane is thickened or not. The same is true of vocal resonance. Percussion generally elicits impairment of resonance. Though there may be high-pitched tympany and even amphoric resonance over a dilated bronchus, the lung on the sound side furnishes hyper-resonance. To auscultation the breathing sounds may be feeble, but there may be broncho-vesicular or bronchial and even amphoric breathing of the most intense kind.

There is usually sharp accentuation of the second pulmonic sound because of the forcible effort of the right ventricle to push the blood through the contracted lung, and when the right ventricle begins to yield cardiac murmurs may develop at the tricuspid valve.

Diagnosis.—Chronic interstitial pneumonia is mainly to be distinguished from fibroid phthisis, which is often impossible without an examination of the sputum for bacilli. The history and duration of the case may be of assistance.

Prognosis.—Recovery is impossible, yet cases last many years, ten, fifteen, and even longer.

Treatment.—As intimated, treatment for the fibrosis is unavailing; intercurrent bronchitis may be helped by the usual remedies for that disease. Anti-spasmodics, belladonna, and hyoscyamus are often useful adjuvants to the cough medicines. Patients are generally better in summer and in a warm climate, where they should dwell, if possible. They should be fed with an abundance of rich, nutritious food, and surrounded by the most favorable hygienic conditions.

BRONCHO-PNEUMONIA.

Synonyms.—*Catarrhal Pneumonia; Capillary Bronchitis; Suffocative Catarrh; Lobular Pneumonia; Aspiration Pneumonia; Deglutition Pneumonia.*

Definition.—Broncho-pneumonia is an inflammation of a circumscribed area of lung tissue caused by an irritant that finds its way to it through a bronchus. Usually it succeeds on a bronchitis of the terminal bronchus leading to the part. Some would consider broncho-pneumonia and capillary bronchitis one and the same thing, but the latter term is best restricted to what it actually indicates—inflammation of the smallest

bronchioles. It often precedes and is even associated with broncho-pneumonia. Parts of a lobule, a whole lobule, or scattered groups of lobules are thus affected, and may unite to form larger areas. Thus, while a broncho-pneumonia is primarily lobular, we may have even a lobar broncho-pneumonia if all the lobules of a lobe are simultaneously affected. Aspiration pneumonia is a broncho-pneumonia caused by the irritation of inhaled or indrawn particles. Tubercular broncho-pneumonia is one variety of this. Syphilitic broncho-pneumonia is a rare but possible affection.

Etiology.—The recognition of broncho-pneumonia as a separate disease is usually credited to Barthez and Rilliet. Broncho-pneumonia is divisible according to its origin into two distinct varieties—simple or non-specific, and specific, including tubercular and syphilitic forms. The consideration of tubercular broncho-pneumonia is appropriately relegated to the chapter on Tuberculosis of the Lungs.

Broncho-pneumonia cannot be said to be caused by a specific organism, but in addition to the simple mechanical causes to be mentioned the streptococcus and staphylococcus, as well as the tubercle bacillus, are to be considered causes.

Simple broncho-pneumonia is preëminently a disease of the very young and the old. In the young it occurs as an idiopathic affection, though it is also a frequent complication of the infectious fevers, measles, whooping-cough, scarlet fever, diphtheria, and small-pox. In adults, especially the old, it occurs during influenza, erysipelas, typhoid fever, and all debilitating affections, including Bright's disease and organic disease of the heart. The inhalation variety especially occurs in comatose states, however induced. William Pepper lays especial stress on vesicular emphysema as a predisposing cause. In both young and old it may succeed a simple bronchitis from cold, but it is as a complication of the infectious diseases named that it becomes during the first five years of life a very common, serious, and fatal disease, causing, it is said, more deaths among children than any other disease except infantile diarrhœa. Diarrhœa itself and rickets are also to be included as predisposing causes. All influences depressing to life, such as overwork, fatigue, the air of badly ventilated and crowded houses, insufficient food, and defects of hygiene act similarly. Collapse of the lung is at once a cause and a consequence of broncho-pneumonia.

Another cause of broncho-pneumonia more common in adults and the aged is the inhalation of fine irritant particles or the aspiration of particles of food. In comatose states from any cause the sensibility of the larynx is benumbed, and minute particles of food are permitted to pass beyond the rima glottidis to enter the larynx and thence the smaller bronchial tubes, where they excite inflammation. Hence the term aspiration or deglutition pneumonia. Glosso-pharyngeal palsy is often associated with deglutition pneumonia, and it often follows tracheotomy and cancer of the larynx and œsophagus. The inflammation thus excited is sometimes so intense as to cause suppuration and even gangrene. Stone-cutting, steel-grinding, and coal-mining become causes through the irritating particles inhaled in these occupations. Francis Delafield, in the section on broncho-pneumonia, in his "Studies in Pathological

Anatomy," says the extension is not from the bronchus to the air vesicles which are connected with it, but to those which surround it. Thus he says, "It is as if a red-hot needle were thrust through the lung, making a track of charred tissue around it." He refers more particularly to the broncho-pneumonia which succeeds bronchitis.

Morbid Anatomy.—The morbid anatomy of simple broncho-pneumonia is quite definite, yet somewhat complex and difficult of description. The lungs are large and heavy. On the exterior, especially at the base, may be seen a mottled appearance, due to an alternation of dark blue or bluish-black depressed areas with projecting areas more natural in hue. The depressed areas represent portions of collapsed lung, and can, for the most part, be reinflated. In places they are continuous, forming large patches. Where there is much of this diffuse pneumonia, corresponding patches of fibrin may be seen on the pulmonary pleura.

On section the surface of the lung is dark red in color and from it project reddish-grey spots representing areas of broncho-pneumonia. These may be separated by tracts of uninflamed and collapsed tissue, or may unite to form more extensive inflamed areas. A section made transverse to the lobule will be found penetrated by a central bronchiole filled with muco-pus, while if the section is parallel with the length of the bronchiole, the central alveolar passage with its alveoli opening into it may be readily recognized, being rendered distinct by the same muco-purulent contents. Around the bronchus to the extent of from $\frac{1}{8}$ to $\frac{1}{5}$ inch (three to five mm.) or more is an area of greyish-red consolidation elevated above the surface, usually slightly granular to the touch, but still lacking the hard, shotty feel of croupous pneumonia. On pressure, a mixture of pus and desquamated cells may be squeezed out which, at a later stage, becomes almost pure pus, appearing as white points in the non-depressed tissue. Surrounding the imperfectly hepatized areas and at a lower level is a smooth, dark, airless tissue, representing collapsed lung, which may be the seat of beginning inflammation. At a later stage, if the patient survives, especially in adults, the inflammatory foci may assume a darker hue, even that of grey hepatization. Still later in the persistent forms the areas may resemble miliary tubercles, from which they may be always distinguished by the fact that the white droplets can be squeezed out, while the tubercle remains firm. These areas may be converted into cirrhotic patches. During the progress of a broncho-pneumonia the air cells in the adjacent lobules are found dilated, and the edges of the lung and upper portions have also become emphysematous. The bronchioles themselves are also dilated in places. The uninflamed areas are generally congested.

The contents of the bronchioles and air vesicles are pus cells and swollen exfoliated epithelium. The walls of the bronchiole and of the air vesicles are thickened and infiltrated with leucocytes. Rarely do they contain blood or the fibrin-network characteristic of lobar pneumonia. Occasionally, however, minute extravasations of blood may be found.

The phenomena in the aspiration form of broncho-pneumonia are more intense in every respect than in the other forms, the infiltration of the air vesicles with leucocytes leading sometimes to suppuration or even to gangrene.

Symptoms.—The initial symptoms vary with the precursory disease. In a child, and here the disease has its greatest practical interest, there may have been measles or whooping-cough or diphtheria, in which convalescence may or may not have set in. To incipient or aggravated *cough* decided *fever* is added, a temperature of 102° F. (38.9° C.) and higher being attained, the cough becomes more severe and painful, the *breathing becomes rapid*, and an easily visible, distressing *dyspnœa* supervenes. Auscultation reveals fine râles at the bases of or throughout both lungs, but the percussion note may remain clear. Later, however, impairment of resonance may be noted. The embarrassed breathing grows worse, the fever higher, the *lips* and *face become cyanosed*, the short, incessant cough is ineffectual in the raising of expectoration, and the little sufferer is a picture of pitiable distress. For such a state of affairs the term *suffocative catarrh* given by the older authorities is well chosen. Happily, as the disease advances and the blood becomes charged with carbon dioxide, sensibility wanes, the suffering abates, and the cough grows less, but the frequent breathing, often 60 to 80, the lividity of face, and frequent pulse show that the fury of the disease is not spent, but will probably terminate only in the death of the little sufferer, which is directly due to exhaustion of the muscle of the right ventricle. At times, however, and even sometimes when least expected, a favorable turn takes place and a convalescence surprisingly rapid sets in.

In fatal cases in children death may occur within twenty-four hours. When recovery takes place, the disease lasts from five to ten days, and as many more are required for complete restoration to health. More rarely a chronic interstitial pneumonia, what Delafield calls a persistent broncho-pneumonia, develops, which may last for months or years and finally give rise to miliary tuberculosis. In adults, as in children, the symptoms vary with the mode of origin. In the idiopathic form, which is recurrent in some old persons, there is fever, a burning spot in the cheek, and shortness of breath, but a cough less troublesome than would be expected. The physical signs rather than the symptoms determine the diagnosis. There are fine moist râles associated with harsh breathing rather than bronchial breathing, and relatively clear percussion.

The symptoms in a case of deglutition pneumonia are very similar. In the inhalation pneumonia of miners, stone-cutters, and steel-grinders the symptoms are slower in their development and resemble more those of tubercular phthisis.

Physical Signs.—These are by no means as distinctive as those of croupous pneumonia. Though I think it best to separate capillary bronchitis from broncho-pneumonia, the association is so close that, given the fine subcrepitant râles of the former unaccompanied by impairment of resonance, we may infer that broncho-pneumonia is at hand. Further signs, however, of actual involvement of the lung substance are moderate impairment of resonance and *harsh* breathing, rather than true bronchial breathing, though more rarely the latter may be present, especially when the bases of the lung are involved. Inspection may recognize retraction of the cartilages and lower sternum during inspiration, indicating defective expansion of the lung.

Diagnosis.—The diagnosis of broncho-pneumonia is usually easy. High fever, cough, mucus expectoration, fine râles, and slight impairment of resonance following one of the infectious diseases in a child under five years, and developing gradually, admit of but one interpretation. When a number of small foci unite to form a large area corresponding to the whole or a portion of a lobe, the physical signs are more like those of a lobar pneumonia, and the absence of expectoration in children increases the difficulty of diagnosis. Lobar pneumonia develops more suddenly and resolves more rapidly.

The similarity in the morbid anatomy of persistent broncho-pneumonia and tuberculosis has been referred to, and the clinical resemblance is even greater, so that it may be impossible to say of a given condition in a child which it is. The presence of signs at the apices is to be sought for, and if found tuberculosis may be suspected, but the correct diagnosis is sometimes only made on the autopsy table.

Prognosis.—The prognosis varies with the etiology, but broncho-pneumonia is always a serious disease. From 30 to 50 per cent. of children perish from it. Yet, as mentioned under symptomatology, some remarkable recoveries take place. In adults it is about as serious as croupous. The deglutition variety is almost always fatal and is the usual cause of death in glosso-pharyngeal palsy. Some cases pass into tubercular consumption, even in children.

Treatment.—The indifference of parents and the carelessness of nurses are responsible for many cases of broncho-pneumonia occurring during convalescence from measles, diphtheria, and whooping-cough which, with proper care, might have been averted. Among the causes thus responsible are exposure of children with uncovered heads at open doors and windows, insufficient clothing during sleep, overheated rooms, and draughty corridors.

Restorative measures are indicated in this disease from the outset. Nauseating expectorants are rarely demanded and often do harm by lowering the vitality of the young patient. Blood-letting, undoubtedly useful in some cases of croupous pneumonia, is not called for in catarrhal. Opiates to quiet the cough and relieve the pain are the strongest indications in the earlier stages of the disease and sometimes throughout it. They should be associated with diaphoretics and febrifuges, among which the solution of acetate of ammonium, the solution of citrate of potash, and sweet spirit of nitre are the best. The tincture of aconite in small but often repeated doses is extremely valuable if the temperature is high and the pulse full and rapid.

When secretions become free and a stimulating expectorant is required there is none better than the aromatic spirit of ammonium, which fulfils every indication and spares the stomach more than the chloride or carbonate of ammonium. If the accumulation of mucus becomes troublesome it may be dislodged by a mineral emetic such as alum, of which the dose for a child is a heaping teaspoonful; or sulphate of zinc, in doses of 10 to 30 grains (0.65 to two gm.); or the syrup of ipecac, more likely to be at hand, may be used. At this stage alcohol, in the shape of whiskey or brandy, becomes an important adjuvant. They should be added to the nourishment, of which the best form is milk, although nourishing

broths are also indicated. As digestion is apt to be feeble, the milk is better peptonized. Quinine, and especially strychnine as a respiratory stimulant, are useful tonics.

In the way of local treatment counter-irritation by mustard and turpentine is especially useful. The former should be used in the shape of a weak plaster, one part of mustard to five or six parts of flour or flax-seed meal. If white of egg and glycerine are used to mix it with instead of water, the plaster is less painful and may be kept on continuously. One of the best modes of applying turpentine is by the St. John Long liniment, consisting of a teacupful of vinegar, a wineglass of turpentine, and one egg beaten up together. This may either be rubbed thoroughly on the chest or it may be applied on flannel. It may be that the turpentine is absorbed and acts as an expectorant. Blisters are not to be recommended.

The poultice is a measure of treatment for catarrhal pneumonia which is variously valued. It is undoubtedly useful in children if properly used, but great care should be taken that it does not become cold. It should be lightly made and changed often, and when changed it should be done rapidly, a fresh, hot poultice being at hand to replace the one removed. Where poultices are not used the cotton jacket should be substituted, as it ensures a uniform temperature of the body. This should be further favored by maintaining a uniform room-temperature of 68° to 70° and averting draughts by screens.

If the temperature be very high it may be reduced by sponging, or better by the wet pack at a temperature of 75° F. (25° C.). The child does not, however, die of the effects of high temperature, but rather, finally, of a failing right heart. The bath is, however, very calming to the nervous system.

The same measures may be used with appropriate modifications in the catarrhal pneumonia of adults, and also in the variety known as deglutition pneumonia. As this form of pneumonia is, however, generally the beginning of the end in some other serious condition, treatment avails but little.

EMBOLIC PNEUMONIA.

Embolic pneumonia is either non-septic or septic.

Embolic Non-septic Pneumonia.

Synonym.—*Hemorrhagic Infarct of the Lung.*

Etiology.—The non-septic hemorrhagic infarct of the lung is the result of embolism, more rarely thrombosis of the pulmonary artery. The emboli come from the right side of the heart, where they either originate as fragments of thrombi or have entered from the systemic veins. Emboli usually lodge at the bifurcation of the branches of the pulmonary artery. The usual transudation of blood takes place in a cone-shaped area. Not every embolus is followed by an infarct. An embolus may be so large as to cause death before an infarct can be formed. Nor is every hemorrhagic infarct followed by a pneumonia. The ultimate consequences of

non-infectious emboli depend on their size. A large embolus and a corresponding infarct with free extravasation of blood is apt to be followed by gangrene of the lung, which may excite intense reactive inflammation in its neighborhood, and the aspirated blood may cause pneumonia. When the lodged particle is small the hemorrhagic infarct is small and the transudate is a diapedesis rather than a hemorrhage. From this true embolic pneumonia results only when there is no collateral circulation, that is, when it is supplied by an end-artery.*

Morbid Anatomy.—The infarct thus caused is conical in shape with its base toward the pleura and varies in size from that of a cherry-stone to that of a hen's egg. The pleura over the infarct projects above the surrounding surface and is at first smooth, but later is roughened by a film of lymph. The infarct when recent is dark reddish-brown in color, and on section rises also above the surrounding surface.

This transudation is the preliminary of a peculiar reactive inflammation, the embolic pneumonia under consideration. Succeeding a slight preliminary contraction there takes place an immigration of leucocytes from the contiguous vessels which accelerates the reabsorption of the blood. To the disintegration and absorption of the red blood discs succeeds a more rapid paling and contraction until no color remains, or there may be a hardening of the pulmonary tissue with a cicatricial-like contraction into which the pleural membrane is drawn, producing fibroid thickening with radiated prolongations. Such hardening is partly due to a condensation of the lung and partially to an organization of the cells in the infiltrated alveoli and alveolar walls. Such remnant is slate-grey from the remnant of hæmatin which remains, or it may be dark red, due to hæmatoidin crystals throughout it. If the infarct is large a part may break down into reddish inodorous pulp, which may be absorbed, or a part may make its way into a bronchus and be expectorated. In such event the residue of cicatricial tissue is larger. Caseation and calcification of the remains are possible results.

The embolus itself is in like manner removed, a few filaments or slight wrinkles in the wall of the vessel being the sole residue.

Symptoms.—There may be no symptoms, or they may be confined to a transient *pleuritic pain* in the pleura covering the embolus. With the increase in size of the infarct such pain increases and may be associated with some *shortness of breath*, due to destruction of the aërating surface. To this may be added *expectoration of blood* if the effused blood gets into the bronchus. If the infarcted area is sufficiently large there may be dulness on percussion, increased vocal fremitus and resonance, crepitant and subcrepitant râles, bronchial breathing, and bronchophony. Further characteristics are the absence of fever and suddenness of onset and the presence of intra-vascular disease. It has been mentioned that the embolus may be so large and cut off so large a supply of blood to the lung that death will take place before an infarct can form.

Diagnosis.—Embolic non-septic pneumonia is often overlooked. The above symptoms, suddenly occurring in connection with states lead-

* All the large branches of the pulmonary artery are end-arteries, and many of the smaller branches also.

ing to thromboses in the veins or the right heart, may be suspected to be due to non-septic embolic pneumonia. Infarcts which form from non-infectious emboli arising in the left heart or arterial system must be so small as to escape detection, since the emboli themselves must be so small as to pass through the capillaries into the veins, thence into the right heart, and thence to the lung.

Prognosis.—The prognosis of non-septic embolic pneumonia is favorable unless the embolus is so large as to stop up a large vessel, producing a correspondingly large infarct. An embolus plugging one of the largest branches of the pulmonary artery is fatal before an infarct can form.

Treatment.—Nothing can be done to actively relieve an embolic pneumonia of this kind. A patient in whom it is suspected must, of course, be kept absolutely at rest. Counter-irritation may be applied to the chest wall over the area involved. Anodynes should be used to the extent required to relieve pain.

Embolic Septic Pneumonia.

Synonym.—*Metastatic Abscess.*

Etiology.—The cause of septic pneumonia or metastatic abscess of the lung is a septic embolus. Such a septic embolus may originate in a thrombus at a seat of putrid inflammation or suppuration, such as the wound of an operation or a compound fracture or in the uterus after childbirth. The veins of such a focus are filled with thrombi, which extend into the larger branches, where they soften and break up into fragments, some of which may pass into the right heart, thence into the pulmonary artery and its branches, until one is reached small enough to resist its further transit. Such an embolus, which is probably swarming with bacteria, is an intense irritant, and inflammation sets in which invariably terminates in abscess, as contrasted with the simple indurative irritation caused by a non-septic embolus. Thus caused, septic pneumonia is one of the anatomical features of pyæmia.

Morbid Anatomy.—Should it be our fortune to see this form of pneumonia in its earliest stages, the same dark red color as that seen in the hemorrhagic infarct of non-septic pneumonia may be noted, except that the blood extravasation is more copious. Such extravasation is a further irritant, and soon an intense inflammation sets in which may also be divided into two stages. The connective tissue of the alveolar and infundibular walls is the seat into which the pus cells wander. The latter furnishes a white-grey ground on which may be seen, with the naked eye, delicate red lines and circles which represent alveolar septa whose vessels are still pervious to blood. This constitutes the first stage. In the second stage abscess formation rapidly succeeds when the hepatized area rapidly melts into a creamy pus, in which float a few fragments of elastic tissue representing broken-down alveolar walls and blood-vessels. The abscesses thus produced may be multiple, but mostly of small size. If the abscess is subpleural there will be suppurative pleuritis with empyema, and possibly perforation of the lung.

In case a very large vessel is obstructed and a corresponding part of lung cut off, say a fifth of a lobe, the area thus cut off from pulmonary

arterial blood is rapidly filled from the veins, and a condition analogous to a hemorrhagic infarct occurs, to the border of which the inflammation is confined, where finally the necrotic mass is dissected loose.

Symptoms.—The symptoms are those of pyæmia, of which the lung abscesses form a part. A *chill* after a surgical operation or injury after confinement, followed by *sweating* and *high fever,* are significant symptoms. Successions of these are even more conclusive.

Treatment.—Treatment should be supporting and stimulating. Quinine should be administered in large doses, and whiskey as in a low fever. The physician should watch for an opportunity of surgical interference, although such opportunity rarely occurs.

PULMONARY TUBERCULOSIS.

Synonyms.—*Phthisis pulmonalis; Pulmonary Consumption; Consumption of the Lungs.*

The Greek term φθισις is an admirable word, meaning literally wasting, which is almost, if not quite, the most characteristic symptom of the disease known technically as *phthisis pulmonalis.*

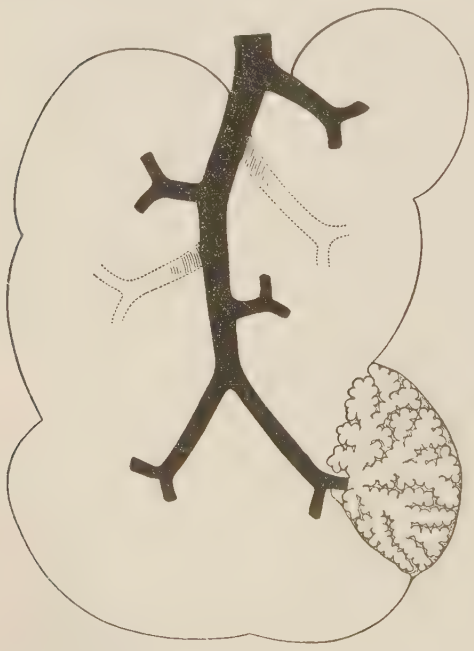


Fig. 36.—Lobule of Lung, Showing Acini and Alveolar Passages.—(*After Rindfleisch.*)

Definition.—Pulmonary tuberculosis is an infectious disease due to the lodgment and proliferation of the tubercle bacillus in the lung substance.

Etiology.—The dependence of tubercular consumption on the tubercle bacillus and its various favoring elements has been fully considered under the head of General Tuberculosis, p. 181. There are two possible routes of invasion of the lungs, one by the air-passages,—inhalation tuberculosis,—the other by the blood. The first is by far the most common for ordinary forms of consumption, while the latter produces usually miliary tuberculosis.

Morbid Anatomy.—*Inhalation Tuberculosis.*—The bacillus, notwithstanding its probable detention at various points in its journey, rarely obtains a fruitful soil until it reaches the ultimate ramifications of a bronchus or its termination in the alveolar passages, infundibula, and air vesicles. It is in the septa forming these that it locates itself by preference, multiplies, and excites secondary

inflammatory processes, the sum of which constitutes the tubercular nodule.

A correct understanding of what is to follow may be facilitated by a review of the appended drawing, showing a single lung-lobule 1.5 cm. long and one cm. broad magnified ten times. The principal bronchus is seen entering the lobule and dividing into seven smaller bronchioles, and each of these into two still smaller ones. These smaller bronchioles open directly into a group of from three to five branching alveolar passages with their infundibula beset with air vesicles. These form the equivalent of an acinus in a racemose gland, and may be termed lung acini, a more constant unit of lung structure, as Rindfleisch truly says, than the lobule, at least as far as size is concerned, since so few as two of these may unite to form a lobule, or as many as 20 to 30. The figure in the text is made up of 14. In pathological processes other than tuberculosis, the lobule is the more important element, since they are determined by the distribution of the blood-vessels and interstitial connective tissue. Emboli, infarcts, and abscesses therefore light upon the border of the *lobules*, while the miliary tubercle is found between the bronchioles and within the *alveolar* septa, where the bacillus secures lodgment and the tubercle its growth. This favorite position may be seen by making a section across one of the smaller bronchioles after it passes into the acinus, when we will meet the circular edge of a bronchiole and an entire system of partitions between three to five alveolar passages.

Under the irritating influence of the bacillus, these septa undergo cellular infiltration, which results in their thickening and encroachment upon the lumina of the air-passages. By this process is produced a little granulation which corresponds at first in size to an acinus, and enlarged by the implication of other acini until a lobule is finally involved, producing an irregularly rounded or oval body assuming somewhat the shape of a lobule, ranging in diameter from a millimeter to six millimeters ($\frac{1}{25}$ to $\frac{1}{4}$ inch)—the *tubercle granulum*. On section such a granule is found perforated by one or more minute openings or slits corresponding to the air-passages, one for each of the roundish subdivisions which make up the nodule. In vertical section the appearance is more definite, having a central stem with branches. The area is whitish-yellow in color, surrounded by a ring of hyperæmic tissue. The microscope shows the periphery of the bronchiole or air-passage infiltrated with concentric layers, of which the external is made up of small lymphoid, the middle of large epithelioid cells, and within this again a third zone in which no cells are differentiable, these having lost their contour and become fused into a homogeneous mass—in a word, having become caseous. The lumen of the tube itself is plugged with cheesy matter. The blood-vessels stop short at the edge of the tubercle, as it is thoroughly avascular.

By suitable staining methods, tubercle bacilli may be demonstrated among the cells of the tubercle granule in moderate numbers at its periphery, but not in the cheesy centre, where they do not seem to thrive until softening takes place, when they are found in great numbers.

Thus begin most cases of pulmonary phthisis as a localized tuberculosis of the smallest air-passages at the apex of one of the lungs. The apices are attacked first, because here the unfolding of the lungs, in the act

of breathing, is more limited, the blood moves less freely and tends rather to stagnate, conditions which favor the retention of secretion, favor the lodgment and encourage the growth of the bacillus.

On the other hand, the view so long entertained that the left apex is more frequently affected than the right seems to be erroneous in the light of modern studies. Thus William Osler out of 427 cases found the right apex involved in 172, the left in 130, both in 111. Pension examinations furnish an opportunity for obtaining information on this subject, and my friend, Theo. G. Davis, of Bridgeton, N. J., took occasion, as examiner in a pension board, to note the cases of tubercular phthisis which passed before his board, with the following results: Out of 897 males whose ages ranged from forty-five to seventy-one years, 94, or about 10½ per cent., had pulmonary tuberculosis, more or less pronounced. Of these 39 were markedly worse on the right side and 29 on the left; both sides were affected in 26, proportions very like those of Osler.

From this usual starting-point the disease spreads with varying rapidity to other parts of the lung. Two principal varieties, however, result, based upon the rapidity of the spread of the disease, while a third variety, also slow in its development, is characterized by a special involvement of the connective tissue. The first is the ordinary chronic form of consumption or chronic ulcerative phthisis, the second pneumonic phthisis or galloping consumption, and the third fibroid phthisis.

Chronic Ulcerative Phthisis.

Synonym.—*Slow Consumption.*

Morbid Anatomy.—This most usual form of consumption, beginning with the tubercle-granulum and associated with more or less catarrh of the apex, extends thence slowly downward. The deposit in the beginning is not actually in the very apex, but a little below it, and usually the first point at which physical signs are found is on the middle of the clavicle or just below it. Sometimes, however, the extension is rather backward, so that the physical signs are first manifested in the supra-spinous fossa, whence the importance of always insisting on the posterior examination.

From this initial focus, usually toward the anterior face of the lung, the disease extends more or less throughout the lobe, or it may pass to another lobe. If the disease be on the right side from the upper it may extend to the middle lobe and thence into the lower lobe about an inch below its apex, corresponding also to a point on the surface opposite the 5th dorsal spine. On the left side the extension is directly from the upper to the lower lobe. From its previous focus the tubercular infiltrate travels centripetally along the bronchi from smaller to larger as a tuberculous peri-bronchitis. As Rindfleisch aptly expresses it: "The white berries acquire a stalk of the same nature and color. The stalks unite with one another and thus form a radiating or rudely stellate focus of larger extent." These stalks are bronchioles, whose walls are infiltrated with tubercle. Larger and larger branches become implicated with the intermediate parenchyma, but usually it does not extend beyond the cartilage-ringed bronchi of the second order, forming tubercular masses of corresponding size.

The infiltration is not limited to peri-bronchial tissue. It extends also inward toward the lumen of the tube, invading the submucous tissue, where it may be seen as whitish or cloudy patches on slitting up the bronchi and washing off the adherent muco-pus. Thus uncovered, the mucous membrane is found also red and inflamed, contrasting strongly with the whitish patches referred to. As we penetrate deeper, these enlarge and intrude upon the lumen of the tube, while the hyperæmic areas grow smaller. Such intrusion becomes finally complete occupation, associated, sooner or later, with an excoriation or rupture of the mucous membrane. This is the beginning of ulceration, which assumes an important place in facilitating subsequent destructive process, and is the foundation of the term adopted by this form of phthisis, *chronic ulcerative phthisis.*

The pathological processes referred to, and the destructive effects of which they are the cause, give to the lung in a state of chronic phthisis a varied picture which is not always found in a single case, nor, indeed, would the lesions of two or more cases always cover this picture. They include the following :—

(1) The *caseous tubercular masses*, formerly called crude tubercle. They embrace single or compound peri-bronchial foci perforated by the central bronchiole, itself plugged with cheesy matter. Thus constituted they form greyish-yellow masses from a couple of millimeters to four or five centimeters ($\frac{1}{12}$ to two inches) in diameter. They have the composition already described. Though usually massed toward the apices of the lung, they may also be disseminated through the remainder of the lung, and around them there may also be found scattered true miliary tubercles.

(2) The second anatomical feature of the phthisical lung is the *cavity.* As soon as a tuberculous area reaches a certain size, the tendency to break down is increased, though such tendency does not depend altogether on extent. The bronchial wall, weakened by the tubercular infiltration and the ulceration referred to, is the initial invitation. The wall yields to the pressure which it formerly easily resisted—the inspiratory and expiratory strain incident to coughing,—the bronchus dilates, the gap of the ulcer widens, and the texture of the bronchus gradually yields. The free access of air to the already necrotic caseous matter causes it to soften, break down, and a cavity results. Small foci unite with others and thus larger cavities form, occupying the greater part of a lobe or even a whole lung in very rare instances.

Large cavities have usually smooth walls and are lined by the so-called pyogenic membrane, into which, however, often protrude blood-vessels of large size, as thick as a crow-quill, and exhibiting also at times aneurismal dilatations. Rarely such vessels may pass directly across a cavity, and when eroded they may give rise to fatal hemorrhage toward the end of a case of chronic phthisis. On the other hand, these vessels may also become thoroughly occluded by an obliterating endarteritis. The surface of these smooth-walled cavities is constantly producing pus, while muco-pus is being added by communicating bronchi. Such cavities may be more or less completely emptied by expectoration. They are also surrounded by a consolidated lung tissue, which gives a dull percus-

sion note and thus often prevents the tympany natural to a cavity. Small cavities have rough and ragged walls, from which there is constant breaking down, adding elastic tissue, pus, granular debris, and bacilli to the matter expectorated. There may be a number of these small cavities separated, and if under the pleura one may rupture into the pleural sac, producing pneumothorax.

Other cavities form by the softening of the centre of a caseous area. Others still may be purely bronchiectatic, being limited by bronchial walls. It is more particularly the bronchi of medium size that are thus involved, weakened also by tubercular infiltration. The form of dilatation may be cylindrical or globular. The small tubes especially may be the seat of cylindrical dilatation.

(3) *Pleurisy* is constantly associated with tubercular phthisis. It is found in four forms :—

(*a*) As an adhesive pleurisy in the immediate neighborhood of tubercular infiltration, causing a collateral hyperæmia and inflammation of the pleura.

(*b*) There may be perforation from a cavity into the pleura exciting a purulent pleurisy and a pyopneumothorax.

(*c*) A pleurisy may be lighted up by cold in a favorable focus of collateral hyperæmia.

(*d*) Finally, the pleura may be the seat of a tubercular pleurisy resulting in a thickened membrane which may be limited or may encase the whole lung and cement the lobes in a continuous inseparable mass.

(4) *Pulmonary concretions* are also found in the phthisical lung, usually about half as large as a pea, smooth or lobulated. They represent calcareous infiltration of alveoli * of the lung, filled with tubercular broncho-pneumonic products. They are a medium of one form of healing of tuberculosis. Those retained in the lung are commonly surrounded by a ring of hyperplastic connective tissue. At times they are expectorated, being released by a sequestrating suppuration, by which they are liberated into an adjacent bronchus, whence they are brought up by coughing. Sometimes a good many are coughed up. They are something different from *bronchial* calculi, which are always smooth, spherical, or elliptical, and are found in small bronchiectatic cavities.

(5) Other evidences of *attempts at healing* seen in the phthisical lungs are of the nature of reactive inflammation. They may occur :—

(*a*) In the initial stage as the result of treatment and favorable hygienic surroundings, when the initial granule is replaced by a cicatricial-like puckering of fibrous tissue or a hard cartilaginous mass of connective tissue.

(*b*) There may be a sequestration or encapsulation of a cheesy nodule which may or may not undergo calcareous infiltration.

(*c*) Even a cavity of moderate size may heal, in which event, the cavity being cleared out, its walls unite by adhesive inflammation and thus a band of cicatricial tissue takes the place of the cavity. Larger cavities may be reduced in size by a contraction of the cicatricial tissue

* If macerated in hydrochloric acid the lime salts can be dissolved out, and the actual elastic tissue frame-work of an alveolus with its infundibula and attached air vesicles be left.

surrounding them, or several small cavities may be thus surrounded. Quite small cavities surrounded by connective tissue and communicating with a bronchus were called *cicatrices fistuleuses* by Laennec.

(6) The neighborhood of a tubercular infiltration is often the seat of a pneumonia which may be simply reactive or specific, *i. e.*, a *tubercular broncho-pneumonia*. The area is hyperæmic, hard, consolidated, and the air vesicles filled with exfoliated epithelium. The latter may exhibit various stages of fatty degeneration. It may be complete when an appearance indistinguishable from that of tubercular infiltration is present. In fact, it is tubercular infiltration plus catarrhal pneumonia.

(7) Where a subject dies of tubercular phthisis, *other organs* should be searched for *tubercles*. Tuberculosis of the larynx is common and is not infrequently associated with destruction of the cords and epiglottis. The bronchial glands are usually involved, swollen, inflamed, or tubercular, and when tubercular may become caseous and sometimes calcareous. Other glands are also affected, such as the cervical, mediastinal, and post-peritoneal. It is now recognized that the so-called "scrofula" of the neck is a tuberculosis of lymphatic glands. After the bronchial glands the organs most affected are the intestine, next the spleen, kidneys, and brain in nearly equal proportion, then the liver and the pericardium.

The only remaining morbid states which may be considered as having any essential relation to tubercular consumption are the amyloid and fatty infiltration. The former is found affecting the kidneys, liver, spleen, and mucous membrane of the intestines; the latter, especially the liver and kidney.

Symptoms.—The onset of tubercular consumption is by no means uniform. Notwithstanding the fact that its insidious nature is well recognized, its initial stadium is often overlooked. The victim is scarcely appreciably ill. Yet he may lose flesh and strength continuously. He may even say that he has no cough, while close questioning will discover that he has had a *slight hacking cough* for some time, worse in the morning. Soon the symptoms are plainer, there is *evident wasting*, an *intermittent fever*, a *bright eye*, and the *cough with expectoration* is a conspicuous symptom. Yet during all this the patient is cheerful and denies that there is much the matter with him.

In another instance an individual "is subject to cold;" he takes cold repeatedly, and each attack, while passing away, yields more stubbornly than the previous one, and finally one comes which persists. There is *daily fever* which abates to return again, emaciation is evident, and the bright eye and burning cheeks and *night sweats* again attest the arrival of the dread disease.

Again, after a stubborn attack of bronchitis in a person previously healthy a *hemorrhage of the lungs* unexpectedly makes its appearance, or such a hemorrhage may set in without previous warning, although, again, careful inquiry may find that cough has been present for some time. The patient has, perhaps, previously been overworked, or lived under unfavorable hygienic surroundings, or may possess a hereditary tendency.

In still another instance a patient may consult the physician without suspecting that he is very ill, and the signs of advanced disease of the apices will be found present, and there may be but a few more months of

life remaining to the unsuspecting victim. Another case may begin with *hoarseness*, due to probably tubercular laryngitis, not infrequently the initial symptom.

A certain number of cases of consumption begin as *tubercular pleurisy*, which invades the lung by contiguity or by blood infection. One of the most convincing facts in favor of that form of infectious theory, which seemed established prior to the discovery of the bacillus, was the frequent occurrence of pleurisy as a forerunner of phthisis. It was held that the caseous product of the pleurisy furnished the infectious virus, which, entering the blood, caused tubercle formations in various parts of the body. Thus, one-third of the 90 cases of pleurisy followed up by Bowditch terminated in phthisis.

Inveterate dyspepsia is associated with many cases and is as often a predisposing cause as a symptom. A great loss of appetite and indisposition to take food are often symptomatic, and their presence does much to diminish the efficiency of remedies and nutriments so essential to successfully combat the disease.

Physical Signs.—Given the suspicion of the existence of tubercular consumption from the presence of the above symptoms, whatever others may be superadded, or whatever modification may occur in them, the diagnosis is completed by a physical examination. These, therefore, will be next studied. While it is not always easy to separate the clinical history of a case of consumption into three sets of symptoms corresponding to the three separate stages in the morbid anatomy, the physical signs corresponding with these stages are tolerably definite. They are :—

(1) The incipient stage, or beginning deposit.
(2) Stage of complete consolidation.
(3) Stage of softening and cavity-formation.

(1) *Inspection*, in the *incipient stage*, is as often negative as not. A slightly diminished expansion in the infraclavicular space, as compared with the opposite side, may be present, and more rarely a slight flattening of the same region. The clavicle becomes correspondingly conspicuous. The body may continue well nourished or slightly emaciated, or the heart-beat in the normal position may be somewhat accelerated, while the respirations are likely to be more frequent than in health.

Palpation may recognize increased vocal fremitus in the same situation, although not always, while the physiological difference in favor of the right side is to be remembered. *Percussion* in this stage gives slightly higher pitch and impairment of resonance, which may be noted above, on, or below the clavicle. Dulness may sometimes be brought out by directing the patient to hold his mouth open during percussion or to hold his breath at expiration.

To *auscultation* above or below the clavicle, we have the first evidence of abnormality in a prolongation of the expiratory murmur and harshness in the inspiratory sound—in a word, broncho-vesicular breathing. Theoretically, this should be preceded by a diminished intensity in the inspiratory sound, owing to the interference of the newly-deposited tubercles with the movement of the air into the air vesicles, but practically this is scarcely encountered, and if encountered is of such indistinctive significance as to be of little value.

Increased vocal resonance is a constant accompaniment of these modifications in the normal breathing sounds, but it, as well as the vocal fremitus, may be masked by a pleuritic thickening, and the physiological difference so often referred to must be remembered. J. M. Da Costa also calls attention to the fact that in a certain number of cases, at this stage, there is a blowing sound in the subclavian or pulmonary artery, and that a murmur is sometimes present in the subclavian or pulmonary artery before any other physical sign is present. There are frequently concurrent with these signs those of a bronchitis more or less acute.

(2) In the *second stage* the changes discoverable by *inspection* are more easily recognized. There is evident loss of flesh, depression of surface, and impaired range of respiratory movement. The hectic flush is intermittingly present. *Palpation* may even discover an increased warmth of skin. The increased vocal fremitus is now plainly recognized unless obscured by a thickened pleural membrane. Dulness on *percussion* is positive and easily elicited.

To *auscultation* there is increased vocal resonance. The bronchial factor in the breathing now becomes conspicuous, showing itself by the harshness and relative shortening of the inspiratory element, with the decidedly prolonged and blowing expiration ; also a gradual diminution of the vesicular factor, until the latter disappears entirely, when we have the typical bronchial breathing of extended areas of tubercular infiltration. This sign will now be found in the supra-spinous fossa posteriorly as well. The high degree of vocal resonance known as bronchophony is also superadded as a valuable confirmation of the presence of complete consolidation. The auscultation signs of a concurrent bronchitis may also be present in this and the next stage.

(3) In the *third stage* the information furnished by *inspection* is still more positive. Emaciation is marked, and breathing and the pulse are rapid, the face often flushed. There is flattening over the affected area, and the excursion of respiratory movement is still more limited. In this stage the superficial veins over the involved area may be prominent, partly from emaciation and partly from obstructed circulation. There may be visible pulsation in the second, third, and fourth interspaces to the left of the sternum because of the retraction of the lung, while the heart may even be drawn up if this retraction be of the left upper lobe. This is seen more particularly in the variety known as fibroid phthisis. To *palpation* the vocal fremitus is still more marked, and even remains distinct over cavities, because of the consolidation around them, unless there be some obstruction to the entrance of air in the bronchus leading to the involved area. Rhonchal fremitus may be added if adventitious sounds are present. The skin is hot and dry, unless succeeding one of the sweats which characterize this stage, when it may be moist and clammy.

Dulness on *percussion* is always to be found in the third stage, but to it is often added some one of the varieties of tympanitic note referred to, viz., pure tympany, the "cracked-pot" sound, or amphoric resonance, due to cavities. These require sufficient size and superficial situation on the part of the cavity. On the other hand, resonance may even be normal over a cavity some distance from the surface, especially if the percussion be lightly made, while the consolidated tissue which almost invariably

surrounds a cavity often permits only a dull sound to be elicited. Wentrich's change of note should be sought—a change of note produced during percussion over a cavity on opening and closing the mouth, the pitch being higher when the mouth is open.

Auscultation in this stage may continue to recognize the bronchial breathing of the second, but to it are superadded first small bubbling sounds or subcrepitant râles indicating liquefaction. Later may be added the distinctive signs of a cavity. These signs are cavernous breathing, cavernous voice, pectoriloquy, either whispering or loud-speaking, amphoric breathing, and amphoric voice. To these are often added the large bubbling sounds known as gurgling, caused by the air bubbling through the fluid in a cavity. Metallic tinkling may be added to these phenomena, caused by the bursting of bubbles in a cavity with the amphoric conditions.

"Cavernous breathing," strictly speaking, is any modification of the normal breathing sounds due to the air passing in and out of a cavity. When high pitched it becomes tubal or amphoric. The amphoric sound is supposed to occur in cavities with firm walls, which best secure the "echoing," which is the condition of amphoric breathing and amphoric percussion. Over more yielding walls the breathing is lower pitched, and to this the term "cavernous" is especially applied.

Special Symptoms.—The *cough* of consumption varies greatly. It is at first very slight, even in advanced stages. As a rule, however, it grows in severity with the progress of the disease, reminding us of Virgil's classic description of Rumor: "*Mobilitate viget, viresque acquisit eundo*" ("Flourishes in its rapidity, and gathers strength as it goes along"). It is caused by the irritation of current bronchitis or broncho-pneumonia or the accumulated contents of cavities. When a cavity becomes more or less filled with secretion it must be emptied, and a spell of coughing comes on and continues until it is emptied, whence the paroxysmal character the cough so often assumes when this stage is reached.

The *expectoration* of tuberculosis varies with the stage of the disease. At first scanty, and in no way characteristic, it grows more copious and becomes puriform as the disease progresses. A circumscribed circular shape is finally assumed, which is more or less distinctive, and is called "nummular," from its resemblance to a coin. The quantity of expectoration varies greatly, from half an ounce (15 c. c.) to half a pint (250 c. c.) in the twenty-four hours. It generally has a sweetish, unpleasant odor, but is rarely offensive. It is sometimes tinged with blood.

Minutely, the expectoration is made up chiefly of pus corpuscles, among which may, however, be found epithelial cells from the mouth and lung alveoli, elastic tissue from the air vesicles, more rarely from the bronchial tubes or blood-vessels, oil drops, particles of food, and generally innumerable tubercle bacilli, and at times blood discs. The elastic tissue is most easily demonstrated by boiling the sputum in a test-tube with an excess of solution of potash or soda, the effect of which is to thin the sputum and permit the elastic tissue to fall to the bottom of the tube; whence it is easily carried by the pipette to the glass slide and recognized under the microscope by its wreath-like or circular shape, if derived from the air vesicles. Care must be taken to eliminate fibres of

elastic tissue which may be derived from food. To this end the mouth should be carefully rinsed out before collecting sputum for examination, and it is further to be remembered that particles of food containing such tissue may remain in the mouth for two or three days. The elastic tissue from the bronchi occurs in the shape of elongated or reticular fibres. That from blood-vessels is similar; more rarely it is fenestrated membrane. The alveolar epithelial cells are round and oval, mononucleated, highly granular, nearly twice the diameter of a pus corpuscle.

The bacilli, which are an unfailing sign of tuberculosis, are demonstrable only by special staining methods, of which that by carbol-fuchsin with or without Gabbet's counter-stain of methyl blue (see p. 181) is recommended.

One of the most unpleasant consequences of the cough is the *vomiting* which it induces, more especially in the last stages of the disease. It is not unusual to throw up a meal immediately after it is taken as the result of a paroxysm of coughing. Such vomiting is probably a reflex act excited by irritation of the pharynx in coughing. Fortunate is the patient who can immediately thereafter take another meal, since this meal is generally retained, because the accumulated muco-pus which caused the coughing spell is also thrown up with the food in the first act of vomiting, and the cough ceases for a while.

Pain is not inherent to tuberculosis, that is, the seat of a tubercular infiltration is not usually a seat of pain. Pain is, however, a frequent secondary symptom. It is most severe as the result of a concurrent pleurisy, when it is of a sharp and cutting character at the site of the pleurisy. Pain also results from inveterate cough. Such pain is usually in the lower part of the chest and is mainly caused, I think, by the motion to which this part of the thorax and the diaphragm is subjected in the act of coughing.

Fever is a symptom of all stages of pulmonary consumption. At the onset there is fever of an irritative kind, due to the deposition of the tubercle and to inflammation. This is a fever of a continued type with slight evening increments, often overlooked, until it becomes associated with hectic fever, which is a septic fever occurring during softening and cavity formation. *Hectic* fever is one of the most interesting symptoms of consumption, adding often a picturesqueness which increases the sadness of the situation. Coming on usually toward the end of the day, the maximum point reached is at no fixed hour, but generally occurs between 2 and 6 P. M., though it may be reached as late as 10 P. M. The minimum usually noted, between 2 A. M. and 6 P. M., may occur as late as twelve noon. Hence frequent observations of temperature should be made during the day and night, two in twenty-four hours being inadequate. Once in four hours is not infrequently desirable and where careful study is desired once in two hours may be necessary. The appended chart shows extreme range of temperature in hectic fever.

There is, however, no greater mistake than to suppose that every case of consumption must have fever throughout. It probably always has fever in the beginning—the fever of onset; but with the disease once established it frequently happens that there is no fever in any part of the twenty-four hours. Appended is a chart of such a case.

In the course of a case of consumption it constantly happens that periods occur of various duration, from one to seven days, in which the fever is higher than usual with moderate remissions, say of a degree, and attended with increased localized pain. These are explained by the occurrence of new patches of broncho-pneumonia which may be either simple or tubercular.

The *pulse* is always frequent in tubercular consumption, and gradually grows feebler as the disease progresses.

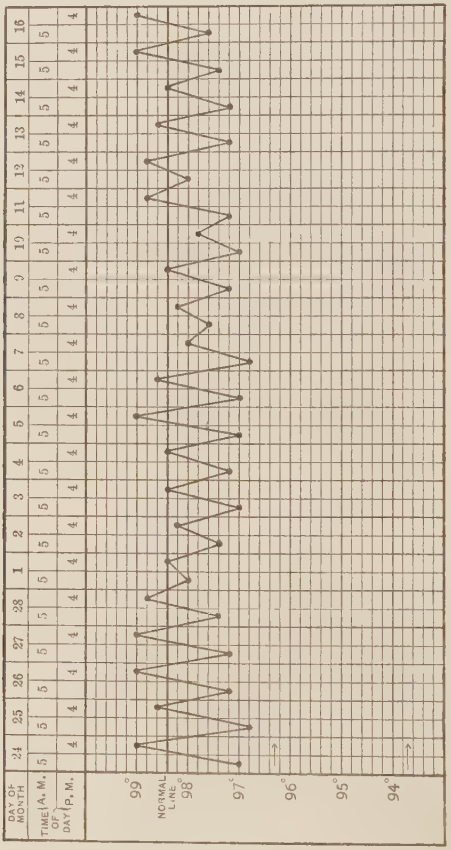


FIG. 37.—TEMPERATURE CHART OF A CASE OF TUBERCULAR CONSUMPTION WITHOUT FEVER, long under treatment at the Hospital of the University of Pennsylvania.

The fever of hectic is generally followed by *sweating*, sometimes limited to the head or the neck. The occurrence of sweats in the night, or rather toward morning, has given rise to the term, "night sweat." They are not, however, confined to the night, but may occur at any time, especially during sleep.

Hemorrhage from the lungs is a symptom everywhere associated with the idea of consumption. There are two periods in which it occurs —one early and one late. The early hemorrhages are usually moderate

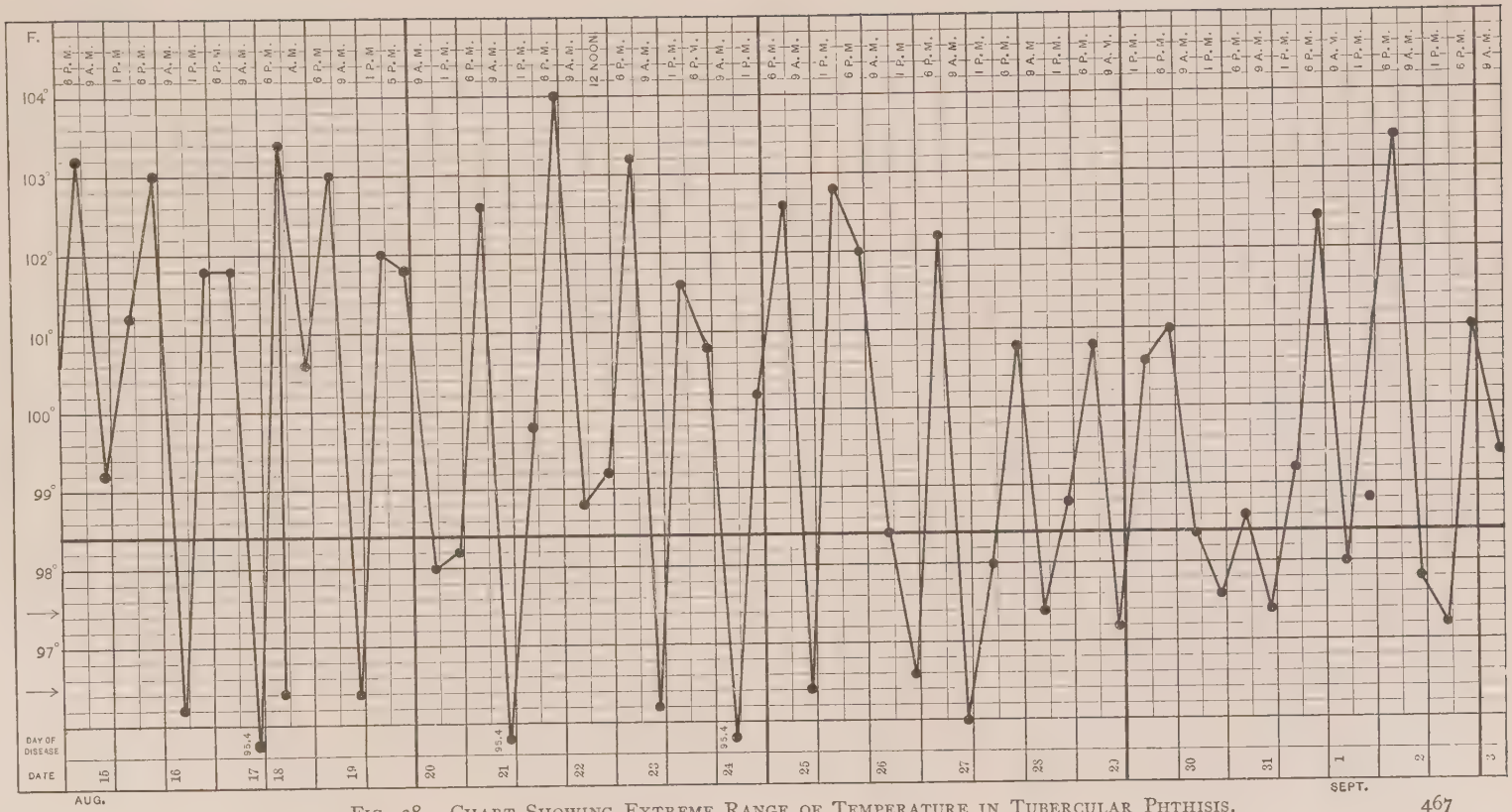


FIG. 38.—CHART SHOWING EXTREME RANGE OF TEMPERATURE IN TUBERCULAR PHTHISIS.

and are due to the rupture of blood-vessels weakened by tubercular infiltration. They are sometimes the very first announcement of the presence of the disease, at others they are a means of relief to a certain feeling of oppression in the chest which precedes them. Their greatest danger is production of an insufflation pneumonia by the inspiration of small particles of clot, which act as irritants. When the hemorrhages are small the blood is often admixed with mucus, constituting the true hæmoptysis. In such cases the blood probably comes from the mucous membrane of the bronchial tubes. The hemorrhages late in the disease are commonly large, sometimes enough to cause immediate death. They are due to ulceration into a large blood-vessel, often one of those described as traversing the wall of a cavity or bringing it from side to side.

Diarrhœa is a frequent symptom late in the disease. It is commonly due to tuberculosis of the bowel and is often exceedingly obstinate. Not every diarrhœa, however, in tuberculosis is tubercular.

The *club-finger* was noted by Hippocrates, and has long been associated with consumption—though not peculiar to it. It is a condition found in other chronic diseases, as emphysema, chronic bronchitis, chronic cardiac disease, and aneurism. The end of the finger is bulbous, quite like a club, and the nail curves over the end. It may involve some of the fingers only.

Tuberculous meningitis may be added toward the close of the disease. In it there is extension of tuberculosis to the membranes of the brain, producing symptoms such as *pain in the head, delirium, acute mania, vomiting, fever,* and finally *convulsions and coma.* The symptoms vary a good deal with the seat of the involvement, and will be further considered in treating of tubercular meningitis. If in the fissure of Sylvius, there may be aphasia and even hemiplegia ; if at the base, retraction of the head and palsies of the cranial nerves from pressure, also optic neuritis ; if on the convexity, delirium is more decided, and there may be local convulsions with hemiplegic weakness. Ventricular effusion—acute hydrocephalus—adds little to the specialization of symptoms. There may be co-involvement of the membranes of the brain and spinal cord, producing symptoms of cerebro-spinal meningitis.

The relation of pulmonary consumption to *cardiac disease* has always been an interesting one. It is commonly thought that affections of the heart and lungs are never concurrent. Occasionally such concurrence is observed, but whether such relation is any but an accidental one is doubtful. Osler reports 12 instances of endocarditis in 216 autopsies. The rarity of lung tuberculosis succeeding chronic valvular heart disease must still be admitted. It has been ascribed to the hypertrophy of the unstriped muscular structure about the smaller bronchioles and their acinous terminations, which keeps the alveoli evacuated of such secretions as favor the development of phthisis.

Albuminuria is often present in advanced stages. It may be simple febrile albuminuria, or it may be the result of chronic parenchymatous nephritis or amyloid disease of the kidney, when it is more copious. Or it may be due to pus if there is tuberculosis of the bladder or kidney. Tubercle bacilli should be sought for in purulent urine if present.

Diagnosis.—The diagnosis of chronic tubercular consumption may be difficult in the early stages, but later, when the physical signs have developed, it is easy. Even in the early stages the finding of the bacillus removes all doubt. Occasionally, however, the sputum is very scanty and difficult to get. If such an examination is not possible, or furnishes negative results, some days may elapse before a positive diagnosis is obtained. For the physical signs in the early stages cannot always be relied on, while there occur cases in which even months after bacilli have been found in the sputum the physical signs are confusing and inconclusive. Due regard must be paid to the fact that in health the expiratory sound below the right clavicle is longer and rougher than correspondingly on the opposite side, while the percussion note may also be somewhat higher pitched. The presence of fever more or less constant, the bright eye, and crimson flush in the cheek, with or without emaciation, should excite suspicion and lead to careful physical exploration and examination of the sputum, if not already made. The search of the sputum for elastic tissue is relatively less valuable, because bacilli are usually found much earlier.

I cannot refrain from adding a word on the importance of securing the physical examination under favorable conditions early in the study of a case. Especially is this true of cases in which there is a hereditary disposition. It goes without saying, that the incipient physical signs of a consumption may easily escape detection when an examination is made with the clothing on, while they would be easily recognized if the patient were stripped to the skin. Too frequently, also, an examination is deferred because of a fear that the patient will be needlessly alarmed thereby. So-called "hemorrhages from the throat" should be carefully investigated, as should also any continued hacking cough. Many of these coughs are now known to be due to tonsillar trouble, but this should not be taken for granted, and a careful examination of the throat should be associated with a physical examination of the chest. A habitually frequent pulse and rapid breathing should also excite suspicion. We should not omit either to examine the posterior part of the chest in the supraspinous fossæ, for it sometimes happens that physical signs are here detected before they are recognizable in front.

Prognosis.—The prognosis of chronic ulcerative phthisis varies greatly with different cases. Its duration extends over periods of from a few months to years.

A more important practical question is that of its possible curability and our power to defer the unfavorable end. That occasional cures from consumption take place cannot be denied; that there is such a thing, even as spontaneous recovery must also be admitted. That such recoveries are infrequent and even rare does not alter the fact that they do occur. That much may also be done to put off the fatal ending of this very sad disease admits of even less dispute. Sooner or later, however, it is usually fatal. But we should not be deterred by this fact from using our best endeavors, not only to put off the end, but also to seek an ultimate cure.

Acute or Pneumonic Phthisis—Broncho-Pneumonic Phthisis.

This more unusual form of tubercular phthisis constitutes one variety of "galloping consumption," or *phthisis florida.* In it the tubercular infiltration is by a rapid peripheral invasion inciting to active inflammation. This is manifested as a broncho-pneumonia, by which the air vesicles and bronchioles are variously blocked with cheesy matter. The result is the dissemination through extensive areas of lung tissue of opaque, white foci $\frac{1}{5}$ to $\frac{1}{2}$ inch (five to 12 mm.) in diameter. These areas are usually separated by areas of a more or less congested but still crepitating tissue, contrasting strongly with the white of the tubercular broncho-pneumonic foci. These tend to soften with varying rapidity, resulting sometimes in numerous little abscess cavities throughout the lung. At other times the broncho-pneumonic foci are more widely separated or may be limited to the apices. In more rare instances the condition may succeed on croupous pneumonia, forming continuous areas which may also extend throughout a lobe or entire lung. The process is truly pneumonic; the results resemble, indeed, more a lung in the second stage of croupous pneumonia. As in it, too, the lung is heavy and airless, sinking rapidly in water. There is, however, a greater tendency to disintegration than in croupous pneumonia, and cavities form rapidly in the apices and elsewhere.

There may also be enlargement of the bronchial glands in any of these forms, but more particularly in the first—the rapid peripheral extension.

Symptoms.—The *broncho-pneumonic* form of consumption occurs most frequently in children as a sequel to measles or whooping-cough. In such what seems to be an ordinary case of bronchitis, with *fever, obstinate cough,* and *shortness of breath,* physical examination will reveal submucous and subcrepitant râles throughout the chest with or without limited areas of consolidation. Tubercle bacilli and elastic tissue appear in the sputum. The fever continues, may become hectic, with sweats. The child emaciates rapidly, and death ensues in from three to eight weeks. Other cases originate more suddenly and with less apparent cause as cases of simple bronchial catarrh, which assume the graver picture described. Such children may inherit a predisposition to phthisis.

In adults the attack begins as an ordinary cold in a person with a predisposition to tuberculosis, though apparently healthy or run down with over-work. The *cough* is harassing, and soon becomes loose, *expectoration* muco-purulent. There are *high fever* and *rapid wasting*, and *hemorrhage* may set in to the surprise of every one concerned. Then there may be a lull in the storm, but for a short time only. The symptoms, and especially the burning fever, wear out the patient.

Bacilli and elastic tissue will now be found in the sputum and the diagnosis is settled. Even adults may perish in three weeks. On the other hand, a reactive effort toward improvement may take place and after a time be followed again by decline and perhaps again by improvement, with the effect of prolonging the disease but not of altering the termination.

The pure *pneumonic* form succeeding croupous pneumonia is more

an affection of adults. More rare still than the broncho-pneumonic form, it may be also rapid in its course. It begins with a chill followed by fever, often after exposure to cold, with pain in the side, cough, dyspnœa, mucous and rusty sputum, impairment of resonance, bronchial breathing, increased vocal fremitus—in fact, all the symptoms of a pneumonia of the whole or a part of a lung, which may be an upper or lower lobe. If the lower lobe, it is probably regarded as a pneumonia until the absence of the signs of resolution call attention to the fact that something unusual is going on. Later, softening and the signs of a cavity may present themselves at the apex and bacilli and elastic tissue be found in the sputum. The case may last for three weeks or three months.

Diagnosis.—The diagnosis in the pneumonic form of phthisis can never be made in the beginning, because the symptoms of the first and second stages of this form are identical with those of the first and second stages of true pneumonia, and it is only when the type of the latter disease is departed from that phthisis can be suspected. The fever in true pneumonia should abate by the ninth day or twelfth day at latest, and if it continues after that time pneumonic phthisis should be suspected and the expectoration should be examined for bacilli.

In the broncho-pneumonic form it is even more difficult to make the diagnosis early from simple bronchitis and broncho-pneumonia. The temperature in phthisis is probably more irregular and higher. Where the disease lasts more than three weeks, the sputum should be examined carefully for bacilli.

Prognosis.—The prognosis is very unfavorable in this form of consumption, death being inevitable in from a few weeks to a few months. Rarely do patients live a year.

Fibroid Phthisis.

Definition.—This term is applied to a form of pulmonary consumption in which the lung, in addition to being the seat of tuberculosis, is permeated by an overgrowth of fibroid tissue. Its course is much slower, and while it often begins as an inhalation bronchitis in those exposed to the inhalation of fine particles of dust from various sources, it may also begin as an ordinary ulcerative or catarrhal phthisis.

Symptoms.—Its symptoms on the whole are less aggravated than those of ordinary phthisis. The cough is less severe, less exhausting, though more apt to be paroxysmal, and the patient has less fever and emaciates less rapidly. He is often able to pursue some occupation. Bacilli are less numerous and are found with greater difficulty. Expectoration is often, however, as copious, often proceeding from cavities or dilated bronchi, and is more frequently fetid. It may contain fat crystals and Charcot's acicular crystals. There may also be hemorrhage. With the addition of these symptoms, and the presence of bacilli in the sputum, the clinical history is scarcely different from that of simple non-specific cirrhosis of the lung, from which it is, indeed, often separated with difficulty. As in this affection there may be hypertrophy of the right ventricle, induced by the extra effort demanded of the right heart to move

the blood through the fibroid lung. Fibroid phthisis is especially characterized by its prolonged course, which may extend over years.

Physical Signs.—The degree of retraction as noticed by inspection is greater, more easily recognized, and not always confined to the apices of the lungs. The heart is frequently dislocated and its apex correspondingly awry, sometimes to an extreme degree. If on the left side, owing to retraction of the lung, there may sometimes be seen a distinct cardiac pulsation in the 2d, 3d, and 4th interspaces. The intercostal spaces are often narrowed, the diaphragm may be drawn up. Modifications of vocal fremitus as revealed to palpation are not nearly so constant, being masked by the retraction and pleuritic complications, and may be absent. There is little or no elevation of temperature.

Percussion is more constant in its results, there being marked dulness and a wooden-like resistance. The hypertrophy of the right ventricle referred to may extend the normal cardiac dulness toward the right edge of the sternum.

Auscultation most frequently notes bronchial breathing and exaggerated voice sound, but both of these may be lessened in intensity by a thickened pleural membrane. A dilated bronchus is a frequent result, yielding the signs of a cavity, which may be in the middle or even at the base of the lung, and furnishes copious expectoration which may be characterized by the peculiar fetor.

To the signs of the fibroid state in one part of a lung are frequently added those of emphysema in the remainder, or in the other lung.

Prognosis.—This is perhaps no better, so far as cure is concerned, than for the chronic ulcerative phthisis, but, as has already been stated, the duration is much longer and under favorable circumstances much more can be done for the patient.

Treatment of Tubercular Phthisis.

There is no disease of like importance in which treatment must for various reasons differ so much in different cases. This is owing partly to the fact that curative measures must be adapted more or less to the circumstances of the patient, and partly to the varying peculiarities of the patient himself. In the following pages I will advise first, regardless of the patient's circumstances, such treatment as experience has shown to be most efficient, then recommend such measures as are useful or necessary under any circumstances.

The fundamental principle of a successful treatment of a case of tubercular consumption is *early diagnosis and corresponding promptness in the application of remedial measures, supported by the belief that consumption is not a hopelessly incurable disease.**

I. **Climate Treatment.**—Immediately that it is recognized that tubercular consumption is established, or even, if possible, when the disease is threatened, the patient should be sent to a suitable climate, provided always that other necessary conditions of a wholesome and happy

* For evidence of the correctness of this principle see a paper by S. Edwin Solly, "Neglect of the Early Diagnosis and Treatment of Pulmonary Tuberculosis," Medical News, Feb. 4, 1893.

life can be secured. To discuss at length the relative value of such places would occupy more space than is justified in a treatise of this kind.

The following may, however, be laid down as truths reached by those who have given special study to this subject:*—

(1) Tuberculosis is relatively rare in the following localities in the order named, viz.: On certain sea-coasts; on certain islands enjoying a nearly pure ocean climate; in desert places of wide extent, such as are found in the interior of continents; in polar regions; and, finally, rarest at high altitudes, increasing in rarity with increasing altitude.

(2) Animals successfully inoculated with the bacilli of tuberculosis develop the disease rapidly when confined, while those kept in the open air may escape entirely.

(3) Damp, especially *cold* and damp, soil favors the development of tuberculosis, as do also variations in dampness when conjoined with changes in temperature.

(4) Moist heat has no influence in producing the disease, but cases originating in tropical countries where this prevails progress more rapidly.

(5) Dryness of air is a positive advantage to the consumptive, while variability in a comparatively dry air has no prejudicial influence. While humidity of the air does not in itself apart from other factors apparently produce phthisis, Solly believes that "where benefit has been received in a humid or sea climate it would appear probable that it is mainly due to greater purity of the air or the elimination of unsanitary conditions and hurtful occupations, as when an overworked citizen takes a sea voyage, or a Bostonian is sent into such a climate as the Isles of Shoals, or a Philadelphian to Atlantic City." On the other hand, whatever the cause, the beneficial influence of a sea voyage to the consumptive is undoubted.

Where low climates are characterized by rarity of phthisis it is by reason of dryness and uniformity of temperature, as is the case with Lower Egypt and the valley of the Nile in Central and Upper Egypt, and in the interior of Algiers as contrasted with the coast belt of that country, with Java, the Gulf States of America, Mexico, Guiana, and some of the West India Islands.

That elevation is unfavorable to the development of consumption and favorable to its cure is abundantly attested, but there is some difference in opinion as to the degree of altitude at which these properties are manifested, some placing it as low as 1500 feet, the majority at 2500. The latter is probably the more correct, though there is reason to believe that different individuals as well as different stages of the disease may be differently influenced in this respect. But for the most part dryness goes with altitude, so that the two effects are commonly associated. How altitude operates independently of dryness is not easy of explanation, although it is probable that everything is more or less dependent on diminished atmospheric pressure, the rationale of which, for want of space, it is impossible to discuss here. The immediate effect is increase in the breathing pulse-rate; next an increase in the depth of

*See S. E. Solly, article "Climate," in Hare's "System of Therapeutics," Vol. 1, p. 415, Philadelphia, 1891.

each respiration followed by cardiac dilatation and by hypertrophy; and later by a fall in the rate of breathing and the pulse to the normal, as the depth of the respirations and the amount of blood passing through the heart at each contraction is increased.

The principle of the beneficial treatment of consumption by climate therapy is therefore to withdraw the subject from the influence of high degrees of atmospheric pressure and moisture, from great thermometric variations, and to associate with these such accompanying conditions as sunlight and warmth, without extreme heat or depressing cloudy weather, cool nights, and opportunity for exercising and rest in the open air.

The following are the conclusions of F. I. Knight as to the indications and contra-indications for altitude treatment:*—

The age at which altitude is most likely to be of service is under fifty; temperament of the phlegmatic rather than the nervous, with irritable heart, frequent pulse, and inability to resist cold. In the latter, however, are not to be included those who show nervous irritability from disease, as they are generally benefited in high places.

As regards disease, those with—

(1) Early apical affection with little constitutional disturbance are most benefited.

(2) Cases with more advanced disease, showing some consolidation but no excavation nor any serious disturbance, do well. When both the apices or much of one lung is involved, and the pulse and temperature are both commonly over 100° F. (37.7 C.), it is best to begin with a low altitude.

(3) Hæmorrhagic cases, early cases with hæmoptysis and without fever or much disease, are benefited.

(4) Patients with advanced disease, those with cavities or severe hectic symptoms, should not be sent to high altitudes. A small, quiet cavity is not a contra-indication; hectic symptoms are a contra-indication.

(5) Patients in an acute condition should not be sent.

(6) Cases of fibroid phthisis are not suitable.

(7) Convalescents from pneumonia or pleurisy are usually well suited to elevated regions.

(8) Advanced cases of tubercular laryngitis, if good local treatment and freedom from dust can be had, may do no worse than elsewhere.

(9) In cases complicated by other diseases there is much care needed. Cardiac dilatation precludes high altitudes; so also does hypertrophy for the most part, though with exceptions. A cardiac murmur, the result of old endocarditis, with no signs of enlargement or deranged, circulation, should not deter. Nervous derangements of the heart are usually contra-indications. In renal disease and in chronic hepatitis, the local physicians claim that benefit is often obtained. Intestinal ulceration is not barred out, but benefit is doubtful. Heredity to phthisis does not contra-indicate high altitudes. Diabetes is a contra-indication. Syphilis is no contra-indication, though the combination with phthisis always makes the prognosis bad.

* Paper by Dr. Knight, published in Transactions of the American Climatological Association for 1888.

The following classification, by G. A. Evans,* of the climates resorted to by consumptives may be found useful in making a selection of climate for a particular case :—

(1) Climate Cool and Moderately Moist, general elevation 2000 feet.—Western slope of the Appalachian chain, Adirondacks, Catskill, Alleghany, and Cumberland Mountains.

(2) Climate Moderately Warm and Moderately Moist.—Western North Carolina, Asheville, elevation 2250 feet; Western South Carolina, Aiken; Georgia, Marietta and Thomasville.

(3) Climate Warm and Moist.—Florida (equable), Southern California, coast region (equable).

(4) Climate Warm and Moderately Dry, elevation about 2000 feet.—Southwestern Texas, Southern California, inland.

(5) Climate Cool and Moderately Dry, elevation about 1000 feet.—Minnesota, Nebraska, Dakota.

(6) Climate Cool and Dry, elevation from 4000 to 7000 feet.—Montana, Wyoming, Colorado, Northern New Mexico, and Western Kansas. In this group are to be placed Davos and San Moritz, in Europe.

(7) Climate Warm and Dry, elevation 3000 to 5000 feet.—Southern New Mexico and Southern Arizona.

The usefulness of sanitaria for consumptives has of late been conclusively demonstrated. As yet these resorts have been provided only at low or moderate elevations, but statistics show that twice as many cures are reported in such sanitaria as in open resorts at the same altitude, and that the patient treated in an open resort at a high altitude has three times as good a chance of recovery as one treated in an open resort in a low climate, and twice as good a chance as one in a sanitarium in a low climate. The usefulness of sanitaria and special hospitals at ordinary altitudes is, however, established. It is reasonable to suppose, therefore, that when sanitaria are established at high altitudes still more satisfactory results may be expected. As to the permanence of the curative results of treatment at high altitudes, it is a common impression among the laity that persons to retain the advantages gained in such climates must remain there. Of this Solly says: "I am firmly of the belief that persons cured in elevated countries have at least as good a chance of keeping well after returning home as those cured at sea level, and owing to the decided increase in general and pulmonary vitality imparted by the climate, probably a much better one."

II. **Hygiene and Dietetic Treatment.**—The following should be carried out as far as possible in every case, whether enabled to make the change of climate advised or compelled by the force of circumstances to remain at home: Secure a habitation wholesomely located, free from dampness, avoiding low ground. The apartments occupied should be those accessible to sunlight for as many hours of the day as possible. In this latitude a south and west exposure obtains this condition. Provide the best possible ventilation for day and night. Especially at night should sleeping chambers be thoroughly ventilated, as during the day the patient

* "Hand-book of Phthisiology," New York, 1888.

secures the effects of change of place, while at night he is compelled to remain in a single room. Ventilation should be secured without subjecting the patient to draughts of air. A low temperature at night may be rendered less harmful than draughts of warmer air, since its effects may be counteracted by extra covering.

In addition to availing himself of a proper location and ventilation, the patient should spend as much time as possible out-of-doors, except during active fever, in the practice of moderate exercise, due regard being had to its effect on the heart and breathing. No sudden or forced efforts should be made. For the most part the patient should be kept moving, although if the weather is suitable he may also sit for a time. Sunlight rather than shade should surround him in his out-door life, and the temptation to sit down long for rest in cool, shady places should be resisted.

Daily bathing of the most thorough kind should be insisted upon, better with tepid, certainly not with very cold water. It should be followed by active friction, so as to maintain the skin functions at their highest point. Cold sea-bathing is not to be recommended, because the reactive power of consumptives is very feeble, and a chill of the body may be followed by permanently harmful results.

The body should be clothed in wool next the skin by day and by night, winter and summer. At night nothing is better or more convenient than a long flannel nightgown extending almost to the feet.

III. Food should be abundant and of the best and most nutritious kind. Meats, including especially fats, poultry, game, oysters, fish, rich animal broths prepared in the most tempting way should be provided, because the quantity taken should be as large as can be digested and assimilated. Milk and cream, cheeses, and the like are eminently suitable. Koumiss may be substituted for milk.

What shall we say of alcohol? It is in the majority of cases an efficient adjuvant in consumption, if properly used. That it is at times abused and that the alcoholic habit is sometimes acquired does not alter the fact that it is useful. The physician should watch its use as he does that of morphine. A moderate amount with meals in the shape of whiskey improves digestion and increases appetite, while combined with milk and cod-liver oil it helps the assimilation of the latter and contributes to fat production—an acknowledged advantage to the phthisical patient. He should be limited to a couple of glasses of sherry or as many tablespoonfuls of whiskey at dinner, while a half-ounce morning and evening with a glass of milk will be as useful as a larger amount.

The whole purpose of the measures recommended under this heading is the production by improved nutrition of a soil prejudicial to the growth of the bacillus of tuberculosis. The effect of unhealthy location, dampness, bad ventilation, darkness, deficiency in fresh air and sunlight, filth of body, chilling influences, colds, improper clothing, and insufficient food is to favor such growth.

The treatment of consumption by *suralimentation*, as suggested by Debove, may be considered at this point. By it is meant surcharging the stomach with food through the stomach-tube. While it is true of the victim of consumption, as of no other disease, that he should be fed,

the method does not seem reasonable. The introduction of food far beyond what the appetite calls for must be attended sooner or later by a rebellion of the stomach. At the same time some happy results are reported. The method, as recommended, is to wash out the stomach with cold water and then introduce a liter (two quarts) of milk, an egg, and 100 grams (about 3½ ounces) of very finely powdered meat. This is done three times a day. It is much more rational to secure a natural appetite by fresh air and out-door life. If, on the other hand, there be reason to believe gastric catarrh is present, an occasional washing out of the stomach may stimulate the appetite wholesomely. Or the vegetable bitters may be used for this purpose. Of these the tincture of nux vomica in 20 to 30 minim (1.3 to two c. c.) doses before meals in cold water is one of the best. The compound infusion or tincture of gentian in two-drachm (eight c. c.) doses is also excellent.

IV. **Medicinal Treatment.**—As to medicines, the remedy which has undoubtedly been of more use in the treatment of consumption than any other is really a food—cod-liver oil. When cod-liver oil is well borne it should be administered to every such case of consumption. When it is not well borne, that is, when unpleasantly eructated or causing indigestion, loss of appetite, or diarrhœa, it should at once be discontinued, and if a cautious attempt to return to it is met with a similar experience no further trial should be made. In my hands the best method of administration is to place in a wineglass from two teaspoonfuls to a tablespoonful of whiskey and overlay it with the same amount of cod-liver oil. It is then "tossed" into the back part of the throat, and after a little experience this is accomplished with great facility and nothing is tasted but a pleasant residue of whiskey. The maximum dose should be a tablespoonful and twice a day. The best time is immediately after breakfast and on retiring at night, although experience may determine more suitable seasons.

The various compound preparations and emulsions, consisting of cod-liver oil, other tonic substances, gums, and flavors to cover up the taste do not meet with much favor with me. At best they are but half oil, they are costly, and as a rule, in my experience, are no better borne than the pure oil. Occasionally they are better tolerated, and under such circumstances they should be administered. It is to be remembered that the chief purpose of the whiskey is not so much to cover the taste of the oil and to render easy its administration as to favor its assimilation and efficiency.

After cod-liver oil, more frequently in conjunction with it, I value creasote. Creasote is not a specific for consumption, but it relieves the catarrhal symptoms and diminishes the cough and expectoration. There are various modes of administering it. One drop, as dropped from an ordinary bottle—not a dropper—equals very nearly half a minim, and a minim weighs almost exactly a grain. A convenient shape is a gelatine-coated pill, of which ½ grain (0.03 gm.) pills and one grain (0.065 gm.) pills are made. Beginning with one grain after each meal and increasing ½ grain a day, six to seven grains (0.39 to 0.45 gm.) three times a day is very easily attained, as a rule. I do not often exceed five grains (0.32 gm.) or ten drops three times a day, lest the stomach be upset. One

should seek, however, to reach at least this dose and keep it up with occasional intermissions. Another excellent mode of administration is in hot water immediately after meals, beginning with two drops or a minim at a dose and increasing up to ten drops, which correspond very nearly to five grains (0.32 gm.). It may also be given in one of the bitter tinctures, or in any mixture with alcohol, or in emulsion, or with sherry wine. Cod-liver oil and creasote may be given conjointly, that is, the creasote may be incorporated with the oil before using.

I have never been able to secure happy results from the use of creasote by inhalation. It may, however, be employed in combination with chloroform and alcohol, to which tincture of conium is sometimes added to mitigate the irritating qualities of the vapor. A mixture of equal parts of each may be made and a few drops placed on the sponge of a Burney Yeo's inhaler, and inhaled as long at a time as possible; or ten to 20 drops (six to 1.3 c.c.) may be added to seven drachms (26.25 c.c.) of water and one drachm (four c. c.) of glycerin, and used in one of the numerous excellent forms of nebulizer now in use. Or it may be placed on the surface of steaming water with the vapor of which it may be carried to the mouth by a suitable appliance. A little glass tube open at both ends and filled with small pieces of pumice on which the substance to be inhaled is dropped also serves the purpose fairly well.

Iodine has long been a popular remedy employed by inhalation. A good way is to dissolve a few grains in an ounce of ether and to inhale the vapor with the mouth or nose over the vial for a few minutes at a time. The following combination may be used in the little pumice-loaded tube referred to: Compound tincture of iodine, glycerole of carbolic acid, tincture of conium, each a drachm (four c. c.); spirit of chloroform enough to make an ounce (30 c. c.). The carbolic acid may be omitted, if desired, and other changes made. S. Solis-Cohen recommends the use of ethyl iodide placed simply in an ounce vial, over which the patient places his mouth or nose and inhales for five minutes at a time. Or glass capsules containing five minims of the drug may be crushed in a cloth and then inhaled. He regards it as especially useful in ulcerative laryngitis and as assisting in the disinfection and healing of pulmonary cavities.

Iron is indicated in all consumptive cases, and it is generally well borne, but it should be given in much smaller doses than is usual. The bane of iron is its constipating effect, and this counteracts all the good it otherwise does, and in my experience the different preparations of iron do not differ materially in this respect. Such effect is not produced, however, if a proper dose is given, and if it constipates in the dose administered, this should be reduced until no such effect results. When this is attained it should be kept up with occasional intermissions. Five or six drops of the tincture of the chloride of iron thus administered and kept up for a long time go a great way toward keeping up the strength and counteracting the tendency to anæmia so characteristic of consumption. Other preparations of iron are reduced iron, carbonate of iron, which may be given in the shape of Blaud's pills, and the sulphate of iron. The vegetable salts of iron, the citrates and malates, are elegant preparations, and the same principle should be observed in their administration.

Fowler's solution of arsenic is often a useful preparation in consumption and may be combined with iron or alternated with it. It is not desirable to give very large doses, and five minims are a sufficient maximum dose. It is especially useful where there are gastric symptoms, and may be continued in moderate doses for a long time.

Strychnine is a drug which is very valuable in pulmonary consumption, more especially as a heart tonic. It should also be continued over long periods in doses of $\frac{1}{40}$ to $\frac{1}{30}$ grain (0.00165 to 0.0022 gm.) three and four times a day. Quinine is also at times very useful, especially when there is fever.

V. **Serum Treatment of Tuberculosis.**—J. T. Whittaker * correctly says, "The discovery of tuberculin established the first real epoch in the treatment of tuberculosis, as it constitutes the first actual address to its cause." This is none the less true in view of the fact that the first essays with it appeared to be absolute failures. There is reason to believe, however, that the continued use of it by certain courageous therapeutists—Whittaker, of Cincinnati, just quoted; Dennison, of Colorado; Trudeau, of the Adirondack region in New York, and von Rusk, of Asheville—may be followed by results which promise more than the earlier trials immediately succeeding Koch's announcement. This expectation is reasonable in view of the acknowledged efficacy of the serum treatment of other diseases, notably diphtheria. The prospect of benefit to be derived from it is the greater the earlier its use in the disease, the more localized the process, and the less general the infection. It is contra-indicated in cases with decided fever, also when there is hemorrhage. The dose administered should be short of that sufficient to produce a febrile reaction. The primary dose should be 0.0002 gm. or 0.2 mg. on alternate days hypodermically, increased with every other administration 0.0001 gm. until a 0.002 gm. dose is attained, when it may be increased more rapidly, say 0.0005 gm. every two or three days. After 0.015 gm. dose is attained the increase may be more rapid according to the effect produced until 0.05 to 0.1 gm. dose is attained, when decrease by halving the dose at every injection and discontinue altogether at 0.010 milligrams.

The injection is best given in the back between the shoulders. The general guide as to dose is the body temperature, which should be taken for a week before treatment as a basis for comparison. A slight rise (½° to 1° F.) six to twelve hours after the injection is the signal that enough has been given, and the dose should not be repeated until the temperature again falls to the standard determined, after which the dose last given is repeated until it produces no fever.

The object aimed at is to get in as much tuberculin as possible, say up to 0.100 gm., so gradually as to produce only a little local and as little general reaction as possible. It is to be remembered that tuberculin can influence favorably only the tubercular element of phthisis and is powerless and probably injurious where any extensive and generalized complicating streptococcus infection has taken place.

VI. **Pneumotherapy.**—Where for any reason the advantages of high altitude are not available, some benefit may be derived from artificial

* "Theory and Practice of Medicine," New York, 1893, p. 158.

pneumotherapy, by which it is sought to modify the air breathed, more especially as to density, although such therapy may also include modifications in temperature, humidity, and chemical composition. The simplest application as applied to density is the producing of conditions by which the patient may be immersed in a compressed or rarefied air which he likewise breathes. The more usual application at the present day is, however, that of "pneumatic differentiation," by which the patient inhales air different in density from that which surrounds him.

In the differential method the object is also to facilitate inspiration or expiration, or both. Inspiration of compressed air favors the former as does also expiration into compressed air. Expiration, on the other hand, is favored by inspiration of rarefied air and expiration into rarefied air. These objects are accomplished by the pneumatic cabinet, and very satisfactory results are claimed by some observers. The treatment is truly rational. But whether it be the result of inherent difficulties in the use of the apparatus or failure to accomplish what was expected, the use of it does not seem to grow in favor, and I doubt whether as many cabinets are in use to-day as five years ago. To be efficient the apparatus should be used two or three times a day, with intervals of rest between, and unless the patient have it at his own home or be in a hospital provided with one, it becomes almost impossible to avail himself of it.

VII. **Treatment of Special Symptoms.**—Naturally, the first of these is cough, and there is no symptom which requires more judgment in its management. A slight cough is best let alone as a rule, because it is an effort to remove secretion, the retention of which may be harmful. If a cough becomes harassing, so as to keep the patient awake or otherwise wear him out, it should be controlled. This should be done, if possible, by counter-irritation. A simple capsicum plaster, or painting with iodine, or iodine with a little croton oil added, or a mustard plaster, or turpentine stupe may answer the purpose when the cough is not too severe.

As to cough medicines, creasote may be classed among the curative measures for this symptom, as it diminishes secretion and thus relieves cough. Moderate cough is often easily controlled by simple syrupy remedies, such as syrup of wild cherry and syrup of tolu, to which some dilute hydrocyanic acid may be added, two to four minims (0.12 to 0.24 c. c.) to the dose. If these measures are not sufficient, an opiate becomes indispensable. It does not matter much what preparation is used. A teaspoonful of paregoric in the beginning is often sufficient, acting like a charm, or deodorized tincture of opium, if a stronger preparation is needed, will answer better because of its smaller bulk. For this reason, too, sooner or later, the alkaloids of opium are indicated. Codeine is the best of these to start out with in doses of ¼ grain (0.0165 gm.) increased. Morphine, however, becomes ultimately the best remedy in the majority of cases. When this stage is reached the wiser course is not to order it at stated intervals, but at such times as the cough needs especially to be controlled, as at night on going to bed, or once during the night. In the morning the patient should be allowed to cough for a time to get up the accumulated secretion. The dose essential for this purpose must vary, anything from $\frac{1}{24}$ to $\frac{1}{4}$ grain (0.00275 to 0.0165 gm.). Sometimes it may be combined with advantage with a syrupy prepara-

tion, which facilitates expectoration, and to this may be added a few drops of a mineral acid, as the aromatic sulphuric. A cough medicine of this kind, long in use in Philadelphia, is as follows :—

℞. Morphinæ Sulph., ... gr. ss–ij (gm. 0.033–0.066)
Potass. Cyanid., ... grs. iij (gm. 0.198)
Ac. Sulph. Aromat., ... ʒj–ij (gm. 3.96–7.92)
Syr. Prun. Virginian., . q. s. ad. . . fʒiij. (gm. 95.04).
M. et S.—Teaspoonful as often as necessary to quiet cough.

In the morning when a patient has to contend with a cavity full of pus it is better to give him a tablespoonful of whiskey or a milk punch to aid in coughing up the accumulated matter than to give a sedative cough mixture.

The ammonium preparations, chloride and carbonate, are rarely useful in the cough of consumptives, while their effect is to derange the stomach and destroy the appetite. Sometimes, however, where there is much loose phlegm, the use of the former for a short time may be beneficial. Under the same circumstances terebene is one of the best medicines, given in doses of five to ten minims (0.3 to 0.6 c. c.). It taxes the stomach, however, somewhat severely.

The fever of consumptives rarely demands special measures. Should the temperature exceed 103° F. (39.4° C.) there is no more satisfactory or harmless measure than sponging, allowing a thin film of water, the evaporation of which produces the refrigerating effect. Or three grains of antipyrin or acetanilide or five of phenacetin (0.198 to 0.33 gm.) may be given, the effect watched, and the drug repeated two or three times if necessary. The high fever of phthisis rarely lasts long and of itself does little or no harm. It is merely a symptom of a more uncontrollable septic process.

Night sweats do demand special measures. By far the most reliable therapeutic agent is atropine ; $\frac{1}{100}$ to $\frac{1}{60}$ grain (0.00066 to 0.0011 gm.) at bedtime usually suffices. It may be combined with morphine, if the latter is necessary. Sponging at bedtime with a saturated solution of alum in alcohol is often efficient when atropine fails, or sponging with simple hot water may answer. Agaricin or agaric acid in doses of $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.00825 to 0.0165 gm.). Camphoric acid, 20 to 30 grains (1.32 to two gm.) in a capsule at bedtime, is another remedy highly recommended ; so are muscarin, five minims (0.3 c. c.) of a one per cent. solution, and picrotoxin, $\frac{1}{60}$ grain (0.0011 gm.). An old remedy is the aromatic sulphuric acid, and it is certainly a good tonic, which, administered in doses of ten to 20 drops (six to 1.3 c. c.) before meals, may also aid in checking the sweats.

Hemorrhage is an alarming symptom and must be treated, although it is probable that most hemorrhages stop of their own accord. The patient should be immediately put to bed at rest, with the shoulders raised. Ice, suitably encased, may be applied to the chest, or cloths wrung out in cold water. A hypodermic injection of $\frac{1}{4}$ grain (0.016 gm.) of morphine to an adult is a useful measure to secure quiet. Indeed, I almost always begin treatment with it. Gallic acid may be given in doses of 15 grains (one gm.) every half hour while the hemorrhage lasts. The domestic remedy, common salt, is probably useful by exciting reflex con-

traction. A teaspoonful swallowed is the dose. When the hemorrhage persists the hypodermic use of ergot is to be recommended. The best preparation for this purpose is a good quality of the fluid extract, of which 30 minims or a drachm (two to four c. c.) may be injected at one time, twice in the twenty-four hours. What is known as ergotin is probably a solid extract, of which one grain (0.065 gm.) is equivalent to five minims (0.3 c. c.) of the fluid extract.

The diarrhœa of consumption does not generally become troublesome until tuberculosis of the bowel develops. Slight degrees seem often to relieve the cough. When there is tuberculosis of the bowel it is exceedingly difficult to control. Sufficient doses of bismuth are on the whole the best remedy—sufficient, because at first the smaller quantities, say ten grains (0.66 gm.) answer, while later much larger doses are necessary. Opium is, however, often necessary, and sometimes the mineral astringents, as the acetate of lead, nitrate of silver, and oxide of zinc, act well in combination with it. Tannic acid is also efficient in combination with opium, and changes must be rung on these various remedies, as any one is apt to lose its effect.

VIII. **Prophylaxis against Tuberculosis.**—Accepted views as to the nature and causation of tuberculosis have raised the question of prophylaxis into one of paramount importance. Careful analysis of accumulated evidence in favor of the communication of tuberculosis goes to show that sputum dried and disseminated with dust in the atmosphere is by far the most important medium. After this the meat and milk of tuberculous cattle, though most recent studies, already referred to on p. 184, go to show that it is doubtful whether tuberculosis was ever caused by the drinking of milk. The perspiration of the affected subject must be acknowledged to be a possible medium, since inoculation of animals by it has resulted in tuberculosis, while the sweat collected after washing and the use of proper antiseptics failed to produce the result. Kissing and the use of wind instruments and pipes previously used by tubercular subjects are possible media. It is claimed of meat and milk that they infect through the alimentary canal and the form of tuberculosis resulting from them is usually glandular, especially of the adjacent mesenteric glands. In like manner the tuberculosis traced to kissing has been in the glands about the neck. The discharges from skin tuberculosis or lupus are also vehicles of infection. Mainly, however, we have to guard against sputum as an agent of infection, the other causes being comparatively easy of escape.

The first and most important measure is, therefore, the disinfection of the sputum. To this end a spit-cup should always be used when possible and it should contain a germicide which will destroy the bacillus. The best of these germicides is corrosive sublimate, dissolved in water in the proportion of one to 1000 or half grain to the ounce, and a small quantity of this solution should be placed in the spit-cup. In consequence of the fact that corrosive sublimate coagulates albumin the tartaric or citric acid sublimate should be used. Next in efficiency is carbolic acid in proportion of one to 30 or 24 grains (1.6 gm.) to an ounce (30 c. c.) of water. A strong solution of soda or potash may be used. As already stated, sputum becomes practically active only when

dried, pulverized, and carried into the air as dust. It is evident, therefore, that even water in the cup will render it harmless for the time being, while if scalding water is substituted its permanent destruction is secured. The first-mentioned methods are most efficient and should be practised when possible. Such vessels should be further washed with scalding water and more germicide solution at least once a day.

Under no circumstances should the patient be allowed to expectorate upon the floor, in cars or other public conveyances, or even, if possible to prevent it, in the street. In order to meet these necessities as well as those of other situations in the house where temporarily the use of sterilizing cups is impossible, the handkerchief is indispensable, but it should consist either of old pieces of muslin or linen which can be burned after use, or of porous paper to be similarly disposed of. The so-called Japanese handkerchiefs answer the purpose admirably.

Dettweiler's pocket spit-cup, invented for use in the street or elsewhere as a substitute for the handkerchief, is an admirable invention. It is made of blue glass, is flat, and holds about three fluid ounces, or 90 cubic centimeters. There are two openings, one at the top and one at the bottom, both provided with metallic screw-caps. The upper and larger opening receives a polished metal funnel extending half way down into the flask, and the whole is closed tightly with a spring cover or cap. The funnel acts like a similar appliance in certain ink bottles and prevents the spilling of the contents of the flask even if the cap is left open. The lower opening is intended to facilitate the thorough cleansing of the flask. It is said that it can be made at a cost of less than 50 cents, and can be easily kept clean. The pasteboard spit-cups, supported in a rim of steel, recommended by the New York City Health Department, intended to be burned after use, are correspondingly inexpensive and answer the purpose very well.

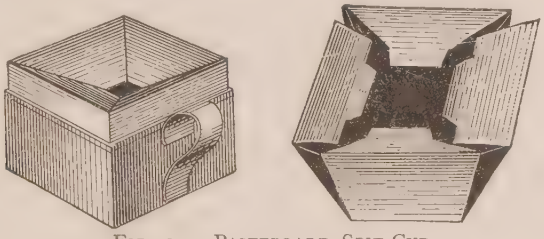


FIG. 39.—PASTEBOARD SPIT-CUP.

To the same end, diminution of the possibility of harboring dried bacilli, unwashable curtains and superfluous upholstering should be banished from the rooms occupied by tuberculous patients. There should either be no carpets, or they should be replaced by rugs which can be frequently taken up and shaken. The sleeping-car, with restricted air space *per caput*, its costly upholstery and curtains, used year after year, becomes a possible source of infection, especially in routes toward health resorts, but is less serious than it might be because of the short time that it is generally occupied by the tuberculous and healthy alike. The stateroom of the ocean steamer stands a greater chance of being a medium of infection from its longer occupation.

When it is remembered how easy it is with ordinary intelligence and simple means to render completely innocuous the bacillus of tuberculosis, I do not myself believe it can be any more efficiently accomplished by the assistance of Boards of Health, and I see nothing to be gained by reporting tuberculosis as an infectious disease, like scarlet fever and diphtheria. I am opposed, therefore, to reporting consumption to Boards of Health, because I think it unnecessary, that nothing is gained by it, and that needless inconvenience, to say the least, is occasioned to victims and their families.

The second source of infection, the milk of the tuberculous cow, if it be a source, is avoided by boiling the milk, which is thus rendered thoroughly sterile. There are, however, objections to boiling milk. In the first place, the taste of boiled milk is not always agreeable, but of greater importance is the fact that it is constipating, especially when it is the only food, as in the case of children. It is desirable for this reason, therefore, to be able to use milk unboiled. That this is possible without harmful results was shown by some interesting experiments of Gebbard, who ascertained that the virulence of tuberculous milk is destroyed by dilution with the milk of other cows. Thus milk from the udder of tuberculous cows was found to have lost its virulence when diluted in one instance 40 times, in another 50, and in a third 100 times. On the other hand, the dilution of sputum 100,000 times was found not to affect its virulence, while pure cultures do not lose virulence when diluted 400,000 times. An important practical conclusion is deduced from these experiments of Gebbard, viz., that a time-honored practice as to hand-fed babies, of using only the milk from one cow is more dangerous than the mixed milk of a herd. For the chances of infection with such are much greater. Practically the use by adults of raw milk mixed with other food cannot be regarded as dangerous, but with children fed exclusively on milk precautions should be taken to render it sterile by cooking, or if it must be used uncooked it should be the mixed milk of a number of cows. The milk of a cow known to be tuberculous should be invariably condemned and the animal slaughtered. The products of milk, that is, butter and cheese, are, of course, not amenable to the treatment to which milk can be subjected. Safety from infection from these sources can only be secured by a rigid inspection of cows, and by measures to prevent the development of tuberculosis in these animals.

Infection by tuberculous meat is still rare. In the first place, the flesh of tuberculous animals may not itself be tuberculous, and, in the second place, the cooking to which meat is subjected must kill bacilli. On the other hand, that the communication of tuberculosis by tuberculous meat when carelessly used is possible is shown by the fact that tuberculosis has been produced in animals by the introduction of the juice of the meat of other tuberculous animals and even from tuberculous human beings. The use of raw or half-cooked meat should therefore be prohibited.

In consequence of what has been said of the experimental production of tuberculosis by the inoculation of sweat as well as the increased possibilities of getting into the mouth portions of tuberculous sputum, no one should sleep with a tuberculous patient. Dishes and utensils used

by such patients should not be used by others unless first scrupulously cleaned, and this is best accomplished by thorough boiling. The patient should himself be taught to prevent his hands, face, and bedding from becoming smeared with sputum.

Precautions against auto-infection are scarcely less important than those against infection to others. It has been said that if it were not for auto-infection most cases of tuberculosis, except those within the cranium, would get well. Be this as it may, it is certain that new foci of tuberculosis are constantly being developed in the same patient, which aggravate his complaint and hasten his death. Such a focus is tuberculosis of the intestine, which probably often has its origin in swallowing sputum. Patients should therefore be enjoined against the practice of swallowing sputum, a practice too frequently followed.

The close dependence of tuberculosis upon predisposition, hereditary or acquired, chiefly the former, has long been recognized. As to whether this or infection is the more important factor in the production of the disease cannot be regarded as settled. Thus one authority, Volland, has recently (1892) declared that the greatest amount of good will be done by such treatment early in life as will correct any possible constitutional taint. Behrend, on the other hand, claims that our principal efforts are to be directed against the dangers of infection. Under the circumstances, a due amount of attention paid to both factors cannot be amiss.

It goes without saying that a tuberculous mother should not nurse her infant, but what should the child or adult predisposed to consumption do to avert the evil. The residence is the first consideration. If possible, the person should be reared in a country of high altitude. Such a course is much more likely to prevent tuberculosis than to cure it if once acquired. Above all, he should avoid residence in houses situated in low, damp, and shaded localities. Bowditch's observations many years ago showed conclusively that consumption is favored by these conditions. Further, such person should not reside in a house where many cases of consumption have preceded. And if it is impossible to avoid this the walls and floors should be thoroughly cleaned with the germicide solutions already mentioned. The rooms of the house should be large, airy, and well ventilated. He should sleep at night with windows and even doors open, due precaution being taken against draughts.

Out-door life should be sought under all circumstances, avoiding, however, especially, damp, cold exposure. Riding and driving should be practised. Judicious athletics, such as develop all parts of the body in good proportion and especially such as secure expansion of the lungs, should be encouraged. Frequent inflation of the lungs should be practised several times a day. Practice with dumb-bells and clubs of moderate weight is preëminently calculated to empty the deeper recesses of the lungs of retained mucus and to cause the blood to move into the more remote parts where the circulation is naturally sluggish.

The treatment of *acute* or *pneumonic phthisis* is restorative and stimulant, symptomatic and palliative. There is no advantage to be derived by taking the patient away from home. Food and stimulus are required to combat the exhausting effect of the disease and its fever. The fever itself may

be lowered by sponging and the cautious use of such apyretics as phenacetin, acetanilid, and the like, because in this form of the disease it is more apt to be continuous and exhaustive in character. The cough must be controlled by opiates and such other measures taken as will make the patient comfortable and mitigate the sadness with which an inevitable fatal prospect is more or less associated. If it should happen that the disease assumes an unexpected chronicity, it may fall into a class of cases in which the treatment laid down for the more chronic forms of consumption is available.

TUMORS OF THE LUNG.

The lungs are subject to morbid growths classified as tumors, though owing to their situation they rarely present the macroscopic, tumor-like qualities.

They include carcinoma, sarcoma, and many of the histioid tumors.

Etiology and Morbid Anatomy.—*Carcinoma* occurs rarely as a primary growth, but is not infrequent as a secondary new formation. Primary cancer presents itself usually in the shape of a white or yellowish nodule two to four inches (five to ten cm.) in diameter. It is found in the upper lobe of one lung, posteriorly and externally, more seldom in other parts. It probably originates in the alveolar epithelium and causes secondary infiltration of the bronchial glands and pleura. It may be represented by any of the three principal forms, scirrhous, encephaloid, or epithelioma, also by colloid and melanotic. It occasions a reactive pneumonia in the lung tissue about it and furnishes often the physical signs of this affection.

There also occurs in the lung a primary *peri-bronchial* cancer disseminated in nodules throughout the lung along the bronchi, smaller nodules on the smaller bronchi, and larger, irregular masses on the larger tubes, varying in size from that of a pea to a walnut. It produces also infiltration of the lymph glands at the root of the lung. *Sarcoma* is also a rare form of primary tumor in the lung.

More frequently both carcinoma and sarcoma are found in the shape of secondary nodules invading both lungs. From three to 20 opaque, white nodules, ½ inch (1.25 cm.), more or less, in diameter, are found irregularly scattered through each lung. Every variety of primary cancer may be thus represented secondarily in the lung. Their origin is probably embolic and they may be secondary to cancer anywhere, but most frequently the breast.

As elsewhere, these growths generally present themselves after middle life, primary cancer affecting either sex about equally, while secondary is more common in women, consistently with the more frequent occurrence of cancer elsewhere in women.

The histioid tumors are represented by a sub-pleural enchondroma, occurring rarely primarily as large as a walnut; more frequently secondary to occurrence elsewhere, when it may attain a large size.

Other histioid tumors are myxoma, adeno-sarcoma, dermoid cysts, hydatid cysts, fibromata, osteomata, and gummy tumors.

Symptoms.—Carcinoma and sarcoma may both be latent, or at

most produce such vague symptoms that it does not occur to physician or patient to locate them. There may, however, be *pain, oppression, cough, expectoration,* and superficial signs of *vascular obstruction*, such as lividity of the face and swelling of the upper extremities. The encroachment of the larger cancerous masses upon the pleural cavity may be considerable. Pressure on the trachea and bronchi may occur and occasion great *dyspnœa,* while the heart may be dislocated. The pneumogastric and recurrent laryngeal nerves are sometimes involved, occasioning the various forms of paralysis of the vocal cords and aphonia. The reactive pneumonia referred to may present the physical signs special to this disease, and it is probably thus that the prune-juice expectoration, thought to be quite characteristic of cancer of the lung,—ten times out of 18, as elaborated by Stokes many years ago,—originates. This complication, too, may occasion the fever which is occasionally present.

The external lymphatic glands, as those in the neighborhood of the clavicle, may be involved and exhibit enlargement.

Sooner or later, if the patient lives long enough, that is, if his life is not destroyed by some encroachment on the breathing or vascular function, he emaciates, becomes cachectic and debilitated. The more usual duration of the disease is six to eight months, but death is liable to occur suddenly from the causes named.

Physical Signs.—These, of course, are indefinite, and it is probably their indefinite and irregular manifestation, with the symptoms named, which will suggest the nature of their cause. Physical signs of pneumonia and pleurisy, either alone or combined, may be present, the voice and breathing sounds and percussion-note being affected accordingly.

Diagnosis.—This, in the case of primary cancer elsewhere, is suggested whenever any of the symptoms named occur in a pronounced degree and are sufficiently long-continued. In the case of primary growths the diagnosis must remain doubtful longer, and we must study and wait the development of the more distinctive symptoms.

The non-malignant tumors present no signs by which they can be distinguished from the malignant, except that their course is less rapid and they develop no cachexia.

Treatment.—This consists only in measures calculated to relieve symptoms and make the patient comfortable.

DISEASES OF THE PLEURA.

ACUTE PLEURISY.

Definition.—Inflammation of the serous investment of the lung or of its reflection on the ribs and diaphragm.

Etiology.—I still believe that pleurisy may be caused by simple chilling of the body during exposure to cold. Doubtless fewer cases are thus caused than was formerly supposed, but it does not seem likely that the large number of cases which appear thus to arise, exhibiting the physical signs of pleurisy, and from which recovery is apparently complete, can originate in any other way. At the same time it must be admitted that the proportion of cases which are tubercular in origin is very much larger than was at one time supposed, and, moreover, that many cases of tuberculosis supposed to have succeeded upon pleurisy have really been primary tubercular pleurisy. In addition to tuberculosis as a primary cause of pleurisy, we must mention rheumatism and chronic Bright's disease as predisposing causes at least. It should be said, also, of the latter that a certain proportion of them have been relegated to the tubercular pleurisies. The pleurisies which go to make up the sum of the pyæmic state are of undoubted microbic origin. If rheumatism be microbic, then, too, the pleurisies which occur secondarily to it must, of course, be referred to the same category. In addition to these instances of primary and secondary pleurisy must be mentioned those which are the result of extension of inflammation by continuity and contiguity, as from the adjacent lung to the pleura over it or from the diaphragm to the pleura above it; also pleurisies of traumatic origin.

Morbid Anatomy.—The morbid anatomy of pleurisy will be best understood by supposing every pleurisy to begin, as it probably does, with a dry stage, a *pleuritis sicca*, whatever may be its subsequent course. Thus considered, the earliest stage of all pleurisies has a hyperæmic basis, succeeded immediately by a roughness of surface due to loosening and detachment of the epithelium, a roughness which is increased by the addition of fresh inflammatory lymph composed of transuded fibrin and wandered-out leucocytes from the blood-vessels of the pleura. Further progress of such pleurisy is—

First, toward *resolution*, in the course of which the product described liquefies and is re-absorbed.

Second, toward *organization* and *adhesion*, in which vascularization and fibrillation take place and the two surfaces of the pleura are more or less permanently glued together over an area corresponding to the extent of inflammation. This is the probable explanation of the little patches of adhesion which are so frequently found at autopsies, some of which may have formed without the consciousness or discomfort of the patient while others have succeeded upon a stitch in the side which has been passed by as of little consequence. Other instances of this primary adhesive inflammation are found between the opposed surfaces of pleural membrane covering tubercular deposits in the lung, or limited pneumonic areas or morbid growths, such as gummy tumors, cancers, and sarcomas.

Third, toward *serous accumulation* constituting *exudative pleurisy*, in which varying quantities of fluid are transuded into the pleural cavity. In this usually clear, straw-colored exudate may be suspended shreds of the yellowish plastic lymph already described, which accumulates also most abundantly where the movement of the pleural surfaces is least, as in the chinks and corners of the pleural cavity. This effusion also, in a large number of cases, is absorbed, allowing the pleural surfaces to approach each other and again unite by what is known as secondary adhesive inflammation, organization taking place as before, producing either continuous fusion or bands of new tissue attaching different parts of the pleural surface. The question as to how the process of exudation is stopped is an interesting one, which cannot perhaps be satisfactorily answered, though it is probable that pressure of the accumulated fluid and the contraction incident to organization, as well as the suspended operation of the cause, may be a part.

Fourth, toward *pus formation*, in which either primarily, from the outset, or secondarily, that is, some time after the process has commenced, the microbes of suppuration get in their work and produce a purulent product or an empyema. The onset of this wandering out of numberless colorless cells is often announced by a chill. Even such an accumulation of pus may in rare instances disappear without active interference, permitting the approximation and union of the two pleural surfaces. The pleural surfaces thus apposed are, however, comparable to an ulcer, and the union and repair take place by a formation of cicatricial tissue. This is subject to the contraction usual to such tissue, dragging not only the contents of the thorax, including the heart and lungs, out of place, but also in extreme cases the ribs and vertebræ, producing slight lateral curvature of the spine.

The ordinary serous fluid which more commonly fills the sac in sero-fibrinous pleurisy is a highly albuminous liquid, sometimes coagulating spontaneously, in which are found also a few leucocytes, exfoliated endothelial cells, shreds of fibrin, and sometimes a few red blood discs. Modifications are those in which the red blood corpuscles are much increased producing a bloody fluid, or in which leucocytes are variously numerous short of a number sufficient to justify the term pus. Urea, uric acid, and sugar are sometimes found in pleural exudates. The quantity of fluid ranges from half a liter to four liters (a pint to four quarts).

Various displacements of adjacent organs are caused by the liquid effusion. In right-sided pleurisies the liver is depressed. Recently a very striking case came under my notice, in which the liver was pushed so far forward and downward as to produce the appearance of an abdominal tumor to the left of the epigastrium. In left-sided pleurisies the heart may be displaced as far as the midsternal line, without, however, being rotated. The displacements from traction after organization are difficult to describe, but the heart may be dragged so that its apex is much higher than normal or further to the right than normal, while the parts of lung adherent are drawn in various directions, with the production at times of bronchiectatic cavities. If the patient die during the presence of large liquid effusions the lung is also found compressed into the back part of the pleural sac.

Symptoms.—The initial symptom of pleurisy is usually *pain*—at first in the side. It may, however, be preceded by a *chill*, and at times there may be a short *prodrome of discomfort* in no way peculiar. The pain in bad cases is of the severest kind, and among the pains most difficult to relieve. It is sharp and cutting in its character, aggravated by breathing, so that the patient takes the shortest breath possible, and the breathing is made up of short, hurried gasps. Cough likewise causes agonizing pain and it is accordingly restrained. Nor is the pain in these cases always confined to the chest, but may shoot down into the abdomen and back. The latter probably implies that the diaphragmatic pleura is involved. *Fever* is also a constant symptom, but is not, as a rule, as high as in pneumonia. At the beginning the temperature may be 102° or 103° F. (38.9° or 39.4° C.), but it very early subsides to a lower figure, even though the other symptoms may abate only partially, and under any circumstances it falls much lower after a week or ten days unless there is purulent exudate, when it assumes a hectic type. The *cough* is peculiar enough to require special allusion. It is a short cough attended with little expectoration, and is a much less conspicuous feature than in pneumonia. Its characteristic of shortness is due to the pain caused by the act of coughing, on account of which the act is cut short. The *decubitus* of pleurisy is quite constantly on the affected side, in order that the unaffected side may be free to expand. The patient also has less pain when he lies on the affected side because the range of its motion is restricted. This pertains to pleurisies associated with copious effusions as well as the dry pleurisies.

While the majority of pleurisies begin in this way, a certain number also begin insidiously, and for days and even weeks the patient, while feeling uncomfortable, and would doubtless be found to be feverish and slightly dyspnœic if thoroughly studied, continues his occupation, and even when the physician is called scarcely mentions symptoms which suggest an examination of the thorax. Such pleurisies are known as latent pleurisies. They are latent only to superficial observation. Closer investigation promptly reveals the physical signs of a pleural effusion.

It has already been mentioned that purulent pleurisies may be primary or secondary. They are in any event most frequently tubercular, and an examination of the pus from such a pleurisy not infrequently discovers the tubercle bacillus in it.

Physical Signs.—Acute pleurisy is also resolvable clinically into three stages, each of which is characterized by physical signs more or less distinctive. They include a dry stage, a stage of effusion, and a stage of resolution or absorption.

The *first or dry stage* is characterized anatomically by the presence of the so-called lymph or exudate on the pleural surfaces. During this is revealed to inspection a retrained expansion of the affected side, often thrown into jerks or catches because of the pain suffered in a continuous inspiration. The expansion on the opposite side is full and unhampered. The patient lies on the affected side. Palpation may recognize a fremitus corresponding to the friction of the two pleural surfaces. Percussion in this stage is negative, except that it may cause pain, but auscultation recognizes the friction sound, which will be further charac-

terized in treating diagnosis. It may be at a single spot in the inframammary or infra-axillary space, and hence be overlooked. At other times it may be noted over a considerable area. According as the inflammatory process stops here with resolution or continues into the second or stage of effusion there may or may not be other signs.

The signs of the *second stage* vary with the amount of liquid in the sac. With a small amount, the lungs are slightly floated up, and there may be no signs unless it be a vesiculo-tympany above the line of the fluid, a Skodaic resonance by mediate relaxation of the air vesicles. The effusion, however, rarely remains so trifling, but commonly rises to the mid-chest. In the upright position of the patient, inspection discovers in a spare person shallowness and perhaps obliteration of the lower intercostal spaces. The motion of the chest wall is lessened both in the vertical and transverse directions.

To palpation vocal fremitus is diminished over the area of effusion, but may be increased in the lung above it. To percussion there is absolute flatness over the area of effusion, but the line of demarcation is not everywhere at the same level, being higher behind than in front. The late Calvin Ellis first called attention to an S-like curve in the line of demarcation which is said to be diagnostic. Very important in the diagnosis is the fact that the fluid changes its level when the position of the patient is changed, and correspondingly the line of dulness is altered. There is also an abnormal sense of resistance to the finger in percussing over the area of effusion. Above the effusion, especially anteriorly, there is again Skodaic resonance by mediate relaxation, and even rarely a cracked-pot sound. Tympany may also be due to the proximity of a distended stomach. Measurement discovers that the affected side is a centimeter (0.4 in.) more in circumference than the other side.

To auscultation the breathing sounds are inaudible or very feeble, as compared to the corresponding portion of the opposite side, but vocal resonance, though diminished, is still well heard where the collection of fluid is moderate. Above the line of dulness there is occasionally a friction sound, and close to the root of the lung bronchial breathing may be heard. This is, however, more apt to be the case when the effusion is larger and the lung is further compressed. Egophony is also sometimes heard.

When the effusion is larger, filling up two-thirds or three-fourths of the pleural sac, the effects described are increased, while new ones are added. Inspection notes that respiratory movement is still more hampered, the intercostal spaces are widened and even bulging, while fluctuation may sometimes be recognized through them. The heart is displaced by the accumulated fluid, and if the fluid be in the left sac, the apex is often found far over to the right of the median line, and if in the right, the apex may be pushed further to the left. The heart sounds are not, however, altered. On the opposite side the breathing movements are supplementally increased. There is complete absence of vocal fremitus on the affected side.

Percussion is absolutely flat all over the effusion, and Skodaic resonance is not now obtainable because the lung is too thoroughly compressed up into the apex of the sac. Resistance to pressure is marked.

On auscultation bronchial breathing may be heard at the upper posterior of the lung, because the large tubes are still pervious to air, and the compressed lung intensifies the sound. Sometimes bronchial breathing is heard in more peripheral parts of the chest, probably conducted hither along a band of adhesion or along a rib. Elsewhere there is absence of breath-sounds. Vocal resonance and whispering voice are alike absent, or the former is very feeble. In certain situations, too, high up, where there is but a thin film between the chest-wall and the lung, there may be egophony, but this is more apt to be present as the fluid is being absorbed.

In the third stage, if resolution takes place with a gradual retrocession of the fluid and the reëxpansion of the lung, we have a return to normal physical signs. There may be, too, a *friction redux*. A considerable time is, however, required for absorption, and it is often many days before the normal breathing sounds are heard with their usual intensity or the natural fremitus is felt. Often, on the other hand, resolution is not complete, and there then remain the symptoms and sequelæ of a chronic pleurisy.

In connection with the heart, pleuro-pericardial friction may be heard if the pleura covering the portion of the lung over the pericardium is involved. The apex beat may not be discoverable if it is so dislocated as to be covered by the sternum, and it often happens that the heart must be located by its signs.

Varieties of Pleurisy.—*Tubercular pleurisy* has been alluded to. It is a pleurisy due to the invasion of the pleura by the tubercle bacillus. It manifests itself—

(1) As an acute primary inflammation characterized by a sero-fibrinous or purulent exudate. The onset of such an inflammation may be like that of ordinary acute pleurisy or it may be insidious in its development, like that of the latent form described. It may immediately precede pulmonary tuberculosis, be associated with it, or succeed it.

(2) As an acute pleurisy the result of extension from an adjacent tubercular lung, and as such may be a circumscribed, adhesive, or an extensive sero-fibrinous or purulent pleurisy.

(3) A chronic, adhesive, tubercular pleurisy characterized by great thickening and adhesion of the pleuræ with tubercular infiltration of the thickened product.

The symptoms and physical signs are in no way different from those described in connection with the more usual forms of pleurisy.

Diaphragmatic pleurisy is a painful form of pleurisy in which the pleural covering of the diaphragm is involved either alone or along with the remaining pleura. It is usually dry, plastic, but may also be exudative with a sero-fibrinous or purulent product. The pain is low down in the thorax in the zone of the diaphragm, and is often aggravated by deglutition as well as by breathing. Because of the pain in breathing the diaphragm is fixed and the patient breathes by the upper thorax. Of diagnostic value is the fact that the pain may be increased by pressure at the insertion of the diaphragm on the 10th rib.

Hemorrhagic pleurisy, characterized by bloody effusion, is found in asthenic states however induced, in tubercular pleurisy, in which event the hemorrhage occurs from the young blood-vessels, and in cancerous

pleurisy ; also sometimes in persons otherwise healthy. It is, of course, not to be confounded with blood-stained serum, caused by wounding a blood-vessel in the act of tapping or with a hæmothorax from rupture of an aneurism.

Encysted or circumscribed pleurisy is a form of purulent pleurisy in which adhesions form so as to produce loculi or spaces which are filled with pus. They are quite difficult to recognize during life—in fact, they are commonly found when exploring the chest with the needle. More rarely they are revealed to physical examination, dull percussion-areas being found in alternation with clear areas. Such physical signs should suggest the use of the needle to clear up the diagnosis. These collections sometimes pulsate and become *pulsating pleurisies.* Pulsating pleurisies are almost invariably on the left side and receive in some way the impulse of the heart, which in turn is communicated to the eye or hand of the observer. The possible confounding of these with aneurism will be again referred to.

In *interlobular pleurisy* the opposed surfaces of two lobes of the lung are glued together and sometimes a pocket of pus is pent up between them, forming a variety of encysted pleurisy. Such an abscess may break into a bronchus. It is not usually recognized before autopsy.

Diagnosis.—The certain diagnosis of pleurisy depends almost entirely upon the physical signs, for however severe the other symptoms there is nothing in them by which the disease can be surely recognized. In the majority of cases of pleurisy the diagnosis is easy by the aid of these signs. It is true, there is a certain resemblance between pleurisy and pneumonia in the first stage of each, and in that stage a diagnosis is often difficult, especially where the physical signs are not distinct. The resemblance of the friction sound to the crepitant râle is well recognized. The usual distinctive features are the superficial situation and the intermittent character of the friction sound, its presence during expiration as well as inspiration, and if confined to one of these acts, rather to expiration, while the crepitant râle is heard only during inspiration. The friction sound is also usually more circumscribed, while it may sometimes be heard better with the stethoscope. Pain is very apt to be elicited in pleurisy if the stethoscope is pressed hard upon the chest. As the pleurisy becomes dry and adhesions form, the friction sound resembles more that of creaking leather.

In the second stage of pleurisy, too, furnishing as it does a dulness on percussion like that of the same stage of pneumonia and frequently bronchial breathing, we have also a resemblance in the physical signs. But it is true of the bronchial breathing of pleurisy that it is commonly best heard at the upper border of the dulness and least where the dulness is most marked ; whereas in pneumonia the bronchial breathing is most intense where the consolidation is greatest. *Above all, in pleurisy with effusion there is diminished vocal fremitus and diminished vocal resonance ; in pneumonia increased vocal fremitus and increased vocal resonance.* There is commonly, further, in pleurisy a change of level of the dulness with a change of the position of the patient, which is not the case in pneumonia. The œgophonic voice is also often here present in pleurisy ; whereas we have only broncophony in pneumonia. Finally, in the differ-

ential diagnosis between acute pleurisy and pneumonia, the trifling cough and absence of expectoration in the former are valuable signs, though it must not be forgotten that in old persons there is sometimes very little cough in pneumonia.

As to further differential diagnosis, pleurisy in the dry stage has been mistaken for muscular rheumatism, intercostal neuralgia, periostitis and caries of the ribs, and even gastralgia and ulcer of the stomach. The absence of fever in the first two, the circumscribed situation of disease of the ribs, and the associated history of gastralgia and ulcer of the stomach serve to differentiate them.

The confusion of mediastinal tumors arising from the pleura itself with pleurisy is a natural result, especially since such tumors in their turn produce pleurisy. In pleurisy the physical signs are commonly limited to one side, while in mediastinal tumor the fremitus is less diminished, the dulness extends upward, is more irregular, more circumscribed; while symptoms of compression of nerves and vessels and of encroachment on the œsophagus sooner or later make their appearance. Repeated exploratory punctures may be necessary to settle the diagnosis, which, after all, may require some time to settle it.

The impulse of a pulsating empyema sometimes very strongly suggests an aneurism, but the empyema furnishes no murmurs or pressure symptoms, while the location is usually different from that of aneurism.

Prognosis.—The prognosis of acute pleurisy depends largely upon its cause. The simple pleurisies which are the result of cold always get well, and this is the termination in most cases even when there is large effusion if the exudate remains serous. It has already been said that a purulent pleurisy is, in the vast majority of instances, tubercular. We have learned, however, that a tubercular pleurisy is not necessarily fatal, and it is more than likely that some of the healed empyemas with which we are familiar are instances of such recovery. Others are cured by the introduction of drainage-tubes and exsection of ribs, but more often the patient slowly succumbs to the exhausting effect of the illness or to tuberculosis of the lungs. Not a very rare event is the spontaneous rupture of such a pleurisy outward, an event better anticipated by paracentesis. Very stubborn, too, are the somewhat rarer cases where perforation takes place from the pleural sac into the lungs, adding the symptoms of a pneumothorax to those of the pleurisy. Yet even such sometimes heal spontaneously.

Though not a frequent event, sudden death when least expected is sufficiently so to make it important that one should be on his guard for it. It is not alone when the chest is full or during a tapping that it occurs, but it may happen several days after a large part of an effusion has been removed. Pulmonary thrombosis is probably the most frequent cause. A recent case of my own terminated thus, when convalescence was thought to be established, and the patient expressed himself better than on any day during his illness. At the necropsy was found a white chickenfat clot in the right ventricle extending as a red clot into the pulmonary artery. The chest was partly filled with sero-fibrinous fluid. Œdema of the opposite lung and degeneration of the heart muscle are probable causes, suggested by Weil. Obstruction to the circulation by dislocation of

the heart or twisting of the great vessels has also been suggested as a cause.

Treatment.—Many simple pleurisies doubtless get well of themselves, with, perhaps, more or less adhesion of the lung, which may be the cause of certain unexplained restrictions in expanding the chest. For very severe cases of pleurisy local blood-letting is the promptest measure of relief, and there is no condition in which so delightful an effect comes to the suffering patient, gasping for breath and racked with pain. I am confident, too, that the duration of many pleurisies would be shortened by such a treatment. In its absence the next best measure is a blister, which seems at once to suspend the process as well also as to relieve the pain in the less severe cases. Succeeding the blister a cotton-jacket or a poultice should be applied for a time at least. Anodynes—morphine hypodermically is the best—are often necessary to relieve the pain and sometimes have to be repeated, while I have even known repetition to be inefficient and unsatisfactory when a blood-letting produced prompt relief.

Even where the effusion is considerable it often passes away without any very active measures. The blister aids in its absorption, however, and the iodide of potassium may be used in coöperation, five to ten grains (0.32 to 0.65 gm.) every six hours. If there is much delay, however, in the absorption of fluid, paracentesis thoracis should be practised as soon as the fever has subsided. It is an operation every physician should be ready to do without calling on the surgeon. I prefer for it the line of the angle of the scapula between the 8th and 9th ribs, and while it is true that the chest wall is a little thicker here and sometimes perforation is not immediately easy, it is a point freer from danger to adjacent organs than the side where the chest walls are really thinner. The point for tapping preferred by others is the 7th interspace in the midaxillary line. The interspaces are made wider and the operation easier if the arm of the side to be operated is carried over to grasp the opposite shoulder. The needle should be introduced close to the upper margin of the rib, so as to avoid wounding the intercostal artery. Local anæsthesia should be obtained by the application of ice and salt. It is particularly in the insidious forms of pleurisy that the tapping of the chest becomes necessary, because they seem to be as slow to disappear as they are slow to make their presence known. A further indication for paracentesis is aggravated dyspnœa. The operation is usually well borne, though sometimes faintness results. It is therefore well to fortify the patient in advance with an ounce of whiskey and if faintness results to desist. Sudden death has happened in rare instances during the operation. On the other hand, sudden death has occurred more frequently in cases of full pleura without operation. When this accident occurs it is more than likely that the heart was previously damaged.

Empyemas almost never get well after a simple tapping. The pus re-accumulates and the symptoms and physical signs are renewed. Free opening should be made without delay, and a good, large drainage-tube passed into the chest through an upper and out through a lower opening in the chest wall. The drainage-tube is often very much too small. In the event of failure of the drainage-tube to secure a cure, which at best requires weeks with daily washing out of the chest cavity, exsection of a

part of one or two ribs is sometimes practised with better results. A cure means thorough union of the pulmonary and costal pleuræ and complete obliteration of the pleural sac.

CHRONIC PLEURISY.

Definition and Pathogeny.—Under the term chronic pleurisy are included several morbid states the result of inflammatory processes of longer duration than a few weeks. These include both *exudative* and *dry* or *plastic* pleurisies.

(1) *Exudative pleurisies* characterized by liquid product include—

(*a*) The condition already spoken of as *latent pleurisy* associated with effusion.

(*b*) *Suppurative* pleurisies, all of which, though they may originate acutely, are always of long duration and may therefore be appropriately classified as chronic.

(*c*) *Plastic pleurisies* characterized by a dry product. These originate in two ways: First, they are plastic from the beginning; that is, the so-called lymph first deposited becomes permanently organized as a more or less thick layer uniting the pleural surfaces. Such primary adhesions are more usual in circumscribed areas of pleural surface. Second, the same result takes place when the surfaces separated by the more copious sero-purulent transudate re-approach each other as the latter is absorbed, producing secondary adhesions.

Finally, we have a most distinctive product of chronic pleurisy in the cicatricial tissue which succeeds the healing of the extensive suppurative surfaces forming the walls of an empyema and which also closely cements the lung to the costal pleura. Mention should also be made of the form of chronic pleurisy resulting in a thick interpleural deposit of slow formation, also tubercular in origin, which extends its new formation from the pleura into the interlobular tissue of the lung, dividing it or dissecting it in extreme cases into distinct areas well shown upon section, which has given rise to the name *pneumonia dissecans*, or pleurogenous pneumonia. This form of pneumonia has its type in the pleuro-pneumonia of cattle. I have met one striking instance of this form of chronic pleurisy of tubercular origin in man. Any one of these varieties of chronic pleurisy may originate as a tubercular pleurisy and probably most of them are of this kind.

The morbid product of chronic pleurisy requires no further description than has been given above and in the description of the morbid anatomy of acute pleurisy, which necessarily included to some extent that of its frequent termination in the chronic form. The adhesion between the lungs and the ribs is variously close and the product variously thick, insomuch that while usually the two surfaces are easily dragged apart, sometimes it is impossible to do this without lacerating the lung. Attention may again, however, be called to the displacement of viscera, the retraction of the chest wall, and curvature of the spinal column, which sometimes take place as a consequence of the extreme contraction of the plastic product of chronic pleurisy in its most aggravated form, that with empyema.

Treatment.—It need only be added to what has already been said in the treatment of acute pleurisy, that in chronic pleurisy especially chest gymnastics, consisting in systematic inspiratory efforts and massage of the thoracic walls, must be availed of. Operative procedures must be considered in conjunction with the surgeon. Mild local measures, such as counter-irritation by iodine and counter-irritating ointments, may be useful to relieve pain, which sometimes annoys the subjects of chronic pleurisy. Nothing more can be accomplished by active counter-irritation by blisters.

HYDROTHORAX AND HÆMATOTHORAX.

Etiology.—The term hydrothorax is applied to any accumulation of clear serum in the pleural sacs. It is the result mainly of resistance to the free circulation of the blood through the vascular basis of the pleural membrane. It occurs as a part of general dropsy, however caused, but Bright's disease or valvular heart disease are the most frequent causes. Hence the chest should be frequently examined in these diseases, as hydrothorax may be the first symptom of a dropsy. Hydrothorax is usually bilateral in renal diseases; more commonly unilateral in heart affections. The serous fluid in hydrothorax is characterized by the small amount of albumin as compared with that exuded in pleurisy.

Symptoms.—The symptoms are those of pleuritic effusion, both as to subjective symptoms and physical signs. Crepitant râles are sometimes heard in the lung above the effusion due to its retraction and to partial atelectasis.

Treatment.—This is considered under that of the diseases causing the hydrothorax.

Hæmatothorax is a term applied to any accumulation of blood in the thorax however caused. It may be due to the wounding of vessels, malignant disease, or aneurismal rupture. The symptoms and physical signs and treatment are those of pleural effusion.

PNEUMOTHORAX.

Synonyms.—*Hydro-pneumothorax; Pyo-pneumothorax.*

Definition.—Pneumothorax means air in the thoracic cavity, but the term is limited to the condition in which there is air in a pleural sac. It is almost always accompanied by a liquid inflammatory exudate, usually purulent or sero-purulent, whence the terms pyo-pneumothorax and sero-pneumothorax. The effects of pneumothorax are compression of the lung, almost always dislocation of the heart toward the opposite side, and in some instances displacement of the liver and spleen. Pneumothorax is almost without exception one-sided, though it is not impossible for a double pneumothorax to occur.

Etiology.—The most frequent cause is perforation of the pleura over a phthisical cavity, a hemorrhagic infarct, or in septic broncho-pneumonia or gangrene of the lung. Other causes are perforating wounds of the lung, perforation of the diaphragm due to malignant disease in the abdomen,

especially cancer of the stomach or colon or the œsophagus. Perforation into the lung from the pleural side may occur in empyema. Rupture of the lung due to straining has caused it. The opening may be valvular, so as to admit air intermittently.

Symptoms.—*Sudden pain* and *increased dyspnœa* usually usher in a perforation causing pneumothorax, though the effect may be more gradual. Sometimes the symptoms are more severe, constituting those of *collapse*,—*faintness*, *frequent pulse*, and *lowered temperature*. Pneumothoraces have also been found post mortem when unsuspected before death, having occurred without producing symptoms. The patient may be orthopnœic or may lie upon the affected side for the same reason as in pleurisy. Pleurisy is a frequent but not invariable consequence, and superadds its own symptoms.

Physical Signs.—These are the most distinctive symptoms. Inspection recognizes commonly a bulging half chest with the intercostal spaces obliterated or prominent as compared with the opposite side. The breathing is frequent and short. Palpation recognizes absent or very indistinct vocal fremitus, the lungs being no longer in contact with the chest wall, which is also in a state of tension interfering with vibration. The percussion note is usually ringing and amphoric over the upper part of the side, that containing air, while over the area below, containing the fluid, there is absolute dulness. On the other hand, there may be dulness over the air-containing space instead of tympany on account of the extreme high tension putting a stop to all vibration. We also meet here that interesting modification of tympany known as Biermer's change of note, based upon the fact that with a given tension the larger an air-containing cavity, the lower the pitch of the percussion note. If the patient with pyo-pneumothorax sits in the upright position, the pleural air-containing space is enlarged as contrasted with the recumbent position, because the weight of the fluid pushes the diaphragm downward, whereas in the horizontal position the fluid flows into the gutter between the ribs and spinal column, the diaphragm rises, the cavity becomes smaller, and the pitch of the percussion note is raised.

Auscultation recognizes feeble or absent vesicular murmur in the situations where it is present in health, while amphoric breathing may be substituted—bronchial breathing of a metallic character. Ringing amphoric bronchophony is also heard when the patient speaks. An interesting auscultation sign is the so-called "metallic tinkling," a sound ascribed to the dropping of liquid from the seat of perforation into the fluid below. Here also is produced in its typical expression the "coin clinking" sound conveyed to the ear of the auscultator listening at the back of the chest while a coin placed against the chest in front is being tapped by another coin. This is a sign usually limited to pneumothorax, though it may also be produced over bronchiectatic cavities. Here, too, may be produced the well-known Hippocratic succussion sound by shaking the body of the patient, the splashing being intensified in the air-distended cavity.

Diagnosis.—Almost the only condition with which pneumothorax may be confounded is diaphragmatic hernia, the physical signs of which very closely resemble those of pneumothorax. The causes of diaphragmatic hernia are usually severe traumatic agencies, such as compression

between cars or under masses of earth, yet occasionally more trifling causes produce it, as in the case referred to on p. 429. If such a condition be suspected, all doubt may be settled by passing a stomach-tube or sound, which will discover the exact position of the viscera. A distended stomach itself is named as a source of confusion with pneumothorax, and it is true that succussion and metallic tinkling can be elicited in it in great perfection. The absence of distention of the thorax itself, the limitation of the physical signs to the neighborhood of the stomach, their association with movements of the stomach quite independently of breathing, point to the proper source. Pneumothorax is scarcely likely to be confounded with large cavities, for while the latter furnish amphoric signs over them the vocal fremitus is increased or at least remains distinct, while with pneumothorax vocal fremitus is diminished or absent. Further, over cavities there is at least no prominence while there is often depression, and succussion signs cannot be brought out. Finally, cavities are circumscribed. Bronchiectatic cavities furnish signs behind and below the scapula, and therefore more in the situation of those of pneumothorax, but there is dulness instead of tympany, no bulging, and vocal fremitus probably remains distinct, while there is often pectoriloquy, never present in pneumothorax.

Treatment.—This is mainly symptomatic. Sudden pain and extreme dyspnœa must be treated by morphine, preferably subcutaneously; embarrassing accumulation of fluid by thoracentesis and draining of the sac, and in extreme cases the air may be liberated in a similar manner. Often pneumothorax gives surprisingly little inconvenience, and it is by no means impossible for spontaneous healing to take place. Potain suggested replacing the air and fluid by sterilized air, but such air would soon be substituted by impure air.

MORBID GROWTHS OF THE PLEURA.

These are rare and will be considered to some extent in treating mediastinal disease. The pleura is subject to *carcinoma* and to *sarcoma*, the clinical phenomena of which are identical. Most cases of carcinoma of the pleura arise by contiguous growth from primary cancer of the lung. Secondary cancer of the pleura occasionally arises by metastasis from the mammary gland or lungs.

Sarcoma occurs as a primary growth in the shape of the so-called endothelial carcinoma of Wagner, which starts from the endothelial cells of the lymphatics and connective tissue. It also gives rise to secondary deposits in the lungs, lymphatic glands, the liver, and muscles.

The symptoms of any one of these forms of growth are those of chronic pleurisy, varying in intensity with the extent of the growth, single secondary nodules often giving rise to no symptoms, while the diffuse forms, spreading from the lungs, cause all the symptoms described as belonging to chronic pleurisy, the lung symptoms being relatively insignificant. In the meantime, we may long remain in the dark about the true nature of the disease, its real nature being determined with the development of cachexia toward the end, the decline of strength, and

probably secondary deposits in discoverable localities. The bloody character of the effusion is a sign pointing to malignant disease of the sarcomatous or carcinomatous type.

The *prognosis* is altogether unfavorable and *treatment* is palliative only.

There are also sometimes found in connection with the pleura *chondroma* and *lipoma*, while *calcification* sometimes takes place in chronic inflammatory products.

Echinococcus or *hydatid disease* is occasionally found in the pleural cavity. Of this the first clinical symptom is hydrothorax, the fluid from which is non-albuminous, differing in this respect from that of pleurisy and to a less degree from that of ordinary hydrothorax. The only unmistakable evidence of hydatid disease is the presence of hooklets and fragments of the hydatid cysts in the aspirated fluid. Here, also, the product may be purulent.

MEDIASTINAL DISEASE.

Under mediastinal disease are included all anatomically morbid conditions seated in the mediastinal space, except diseases of the heart, aorta,

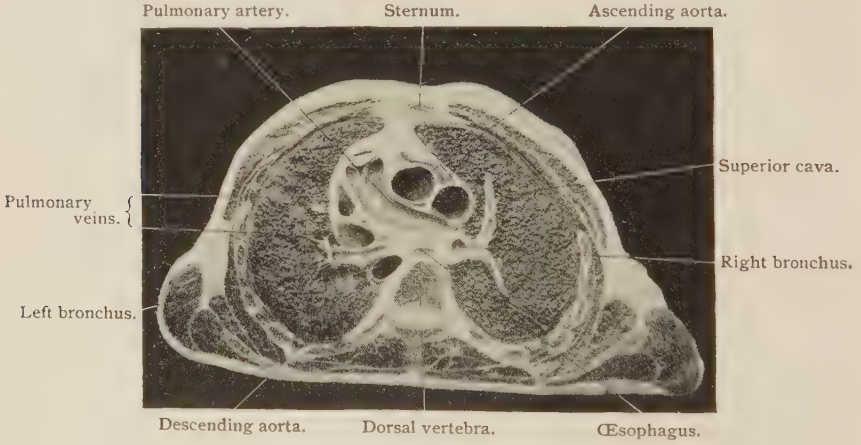


FIG. 40.—SECTION THROUGH FROZEN THORAX AT 2D INTERSPACE IN FRONT, LOOKING FROM ABOVE DOWNWARD.

trachea, and œsophagus. By far the greater number of these are tumors, but simple lymphadenitis, abscess, and hemorrhage are also included.

Anatomical.—In consequence of the difficulty attending the conception of the mediastinum and its contents, I precede the consideration of mediastinal disease by a brief anatomical description of the mediastinum and its spaces.

The mediastinum is bounded in front by the sternum, posteriorly by the vertebral column from the lower edge of the 4th dorsal vertebra downward, and laterally by the two pleuræ. It contains all the viscera in the thorax except

the lungs. Clinicians are in the habit of subdividing this space into the anterior, middle, and posterior. But the anatomists (notably Struthers and Gray) whom I follow, divide it first into two parts, an upper or *superior* and a lower, and subdivide the lower into three, an *anterior*, *middle*, and *posterior*.

The *superior* mediastinum is that portion of the interpleural space above the upper level of the pericardium, between the manubrium sterni in front and the upper dorsal vertebræ behind, and bounded below by a plane passing from the junction of the manubrium with the body of the sternum backward to the lower border of the 4th dorsal vertebra. It contains the origins of the sterno-hyoid and sterno-thyroid muscles, and the lower ends of the longus colli; the transverse portion of the arch of the aorta; the innominate, the left carotid, and the subclavian arteries; the superior vena cava and the innominate veins, and the left superior intercostal vein; the pneumogastric, cardiac, phrenic, and left recurrent laryngeal nerves; the trachea, œsophagus, and thoracic duct, and the remains of the thymus gland with lymphatics.

The *anterior space* is bounded in front by the sternum and posteriorly by the pericardium, and laterally by the pleuræ. It is wider below than above,

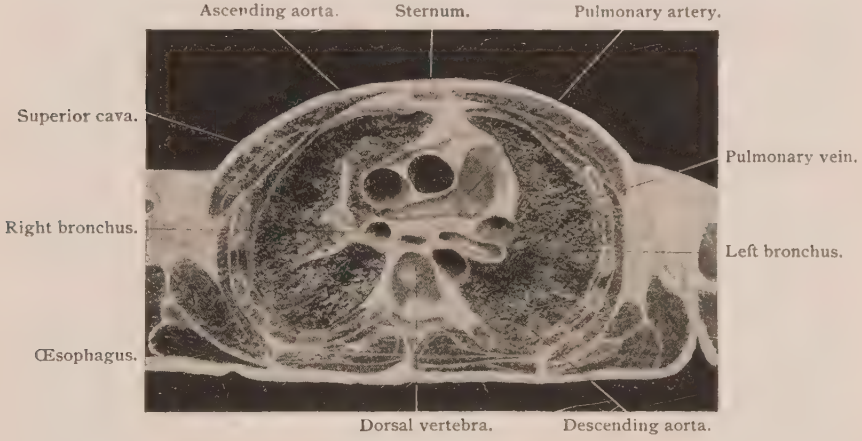


FIG. 41.—SECTION THROUGH FROZEN THORAX AT 2D INTERSPACE IN FRONT, LOOKING FROM BELOW UPWARD.

and is narrowest in the middle, since at this point the two pleural edges approach each other, while in some cases they are actually in contact. The anterior mediastinum contains the origins of the triangularis sterni muscles; the internal mammary vessels of the left side; a quantity of loose areolar tissue; a few lymphatic glands, with lymphatics from the upper surface of the liver and two or three lymphatic glands called anterior mediastinal glands.

The *middle space* contains the heart in its pericardial sac, the ascending aorta, the superior vena cava, the pulmonary artery and veins, the phrenic nerves, the bifurcation of the trachea, and the roots of the lungs, with numerous lymphatic glands. It is broader than the anterior or posterior mediastinal space.

The *posterior space* is triangular in form and is bounded behind by the vertebral column. Its anterior boundary is the pericardial sac and the roots of the lungs; its lateral walls, the pleuræ. It contains the descending portion of the arch and the descending thoracic aorta; the greater and less azygos veins, the thoracic duct, the pneumogastric and splanchnic nerves, the œsophagus, and some lymphatics.

MEDIASTINAL TUMORS.

Historical.—The celebrated English physician and acute observer, Thomas Willis (1621–75), seems to have been the first to have made an observation on mediastinal disease. H. Boerhaave recorded a steatoma of the anterior mediastinum in 1742. Joseph Lientaud (1703–80) described several cases. Boyle published others in 1812, and J. G. C. F. M. Lobstein gave the first text-book description in 1835, after which cases multiplied, although the number which came under the notice of any single observer was always few. F. Strauscheed analyzed 112 cases in 1887, and Hobart A. Hare collected 520 cases in his Fothergillian Essay in 1889. Out of 7566 autopsies at the Marine Hospital at Cronstadt, 158 subjects were found to have tumors of the mediastinum said to be malignant.

Pathology and Morbid Anatomy.—The varieties of growth consist mainly of sarcoma, including lympho-sarcoma, carcinoma, simple lymphadenoid tumors, and more rarely cysts, dermoid and hydatid, fibroma, lipoma, gumma, and chondroma; also the teratoma myomatoides of Virchow. Sarcoma and carcinoma and lymphadenoid tumors make up the larger number. Most observers have found more carcinomas than sarcomas, but in the light of the fact that many tumors formerly described as cancerous are at the present day acknowledged to be sarcomas, and that the majority of recently described tumors in this region are sarcomas, it is more than likely that the latter have always predominated. Hilton Fagge and Douglas Powell were the first to announce this, and William Pepper and Alfred Stengel, in a recent monograph (1895), come to the same conclusion.

The majority of tumors in the anterior mediastinum start from the remnant of the thymus gland and are lympho-sarcomata. The lymphatic structures in the anterior mediastinum furnish a few. In the middle mediastinum the lymphatic glands are the principal starting-point of the relatively frequent lympho-sarcomas. The carcinomas are usually primary, but secondary carcinoma is not infrequent. The breasts, lungs, and stomach are among the primary seats named. The secondary cancers do not usually attain a large size. Cancer may extend from the abdomen to the glands of the chest by vascular embolism, by direct spread of the disease to the under surface of the diaphragm, through which it may penetrate along the lymphatics into the chest and glands, or by embolism through the thoracic duct to the chest and then by retrograde embolism to the glands.

The pleura is also a frequent starting-point of mediastinal growths. Among these are the so-called endothelioma of Wagner and Schulz starting in the endothelium of lymphatic vessels and sometimes the surface endothelium. They are sarcoma or carcinoma according as the endothelium is counted mesoblastic or endodermic in origin. The cases of primary cancer of the pleura are probably endothelioma. Fibrous, fatty, and calcareous tumors of the pleura are rare events. The lungs also contribute tumors to this locality, carcinoma primary and secondary, and sarcoma primary and secondary. Of the primary tumors carcinoma is the more common, but primary sarcoma of the lymphatic glands surrounding the bronchi and within the lungs near the root is not very rare. The clinical symptoms are the same as where the glands around the

bronchi outside of the lung are affected. The cancers may start from the surface epithelium of the bronchi, from the mucous glands, or the alveolar epithelium of the lung. Finally, from the œsophagus, also, start cancerous tumors, invading the mediastinum, usually small, though not always. From these the posterior mediastinum and lungs may also be invaded.

Symptoms.—Mediastinal tumors may be latent. Their symptoms when present are, in a word, those of *pressure.* Such pressure may involve the lungs, the trachea, the bronchi, the œsophagus, the heart, the vessels and the nerves of this locality. They include symptoms, subjective and objective, of the usual kind, and also physical signs. It will be remembered that the symptoms of aneurism are also those of pressure largely, and it is chiefly from aneurism that mediastinal tumor is to be distinguished, often a matter of some difficulty. The division by Pepper and Stengel into three groups affords the most convenient mode of studying these symptoms. These groups are :—

(1) Those in which the anterior mediastinum is the seat of the growth and in which physical signs are likely to be more prominent.

(2) Those involving the middle and posterior spaces, and in which symptoms predominate over physical signs ; and

(3) In which the pleura or superficial portion of the lung is involved, and in which the symptoms and physical signs are of equal prominence. Between these divisions are also intermediate sets of symptoms.

I. *Intrathoracic Tumors Situated in the Anterior Mediastinum and in which the Physical Signs are Prominent.*—The symptoms are mainly those arising from pressure exerted on the venous trunks, the superior vena cava, the right and left innominate. The yielding walls of these vessels as contrasted with the firmer adjacent arteries easily suffer compression, and may even be penetrated by the growths which may proliferate within them, sometimes causing occlusion by thrombosis. The consequence is distention of the veins of the upper extremities, the head, neck, and upper chest. Coldness, lividity, œdema, and clubbing of the ends of the fingers result, while the superficial venous channels may be dilated and tortuous. From pressure on the arteries may result inequality of the radial pulses. Of the nerves, the inferior laryngeal is especially liable to compression with resulting hoarseness and aphonia. The sympathetic is also sometimes compressed with consequent inequality of pupils, the pneumogastric being less frequently involved than where the tumor occupies a more posterior situation. As the tumor enlarges and the breathing passages are intruded upon dyspnœa makes its appearance. Dyspnœa is usually of the inspiratory kind. Pericarditis and pleurisy with pain, hydropericardium, and pleural effusion may be present. With the prolongation of the disease, the patient wastes, but it is said that cachexia is less apt to develop than in malignant growths of the posterior mediastinum. Pain is not always present, indeed, is said to be less marked than in aneurism.

Physical Signs of Growths in the Anterior Mediastinum.—To inspection the sternum is frequently pushed forward and in a few instances eroded. Vocal fremitus may be either increased or diminished. Percussion elicits abnormal dulness characterized by more or less irregular shape. Pulsation may occur, but is rare, while the sharp diastolic shock of aneurism is wanting. If the tumor extends upward sufficiently, it may be felt

in the supra-sternal fossa. Auscultation over the area of dulness may be negative, but sometimes the breath sounds and heart sounds are well transmitted, while a distinct systolic bruit may be produced by pressure on the aorta or the pulmonary artery. Secondary enlargement in the cervical lymphatic glands sometimes makes its appearance.

II. *Intrathoracic Tumors in the Middle and Posterior Portions of the Spaces Around the Bronchi, Œsophagus, Aorta, and Nerves.*—Dyspnœa is an important and early symptom in tumors in this situation and the inspiratory effort is extreme. Pressure here is especially exerted upon the vena cava, whence results œdema of the abdominal walls and lower extremities. The effect of pressure on the arteries is not serious. From pressure on the vagus nerve comes peculiar cough, paroxysmal and whooping. Sometimes it is loud and ringing, at others constant and hacking. This is said to be due to the joint involvement of one vagus and the pulmonary plexus; whereas experimentally two pneumo-gastrics are required to be cut to produce this symptom. The explanation is in the involvement of the pulmonary plexus. Muco-purulent and even blood-stained sputa may attend the cough. The latter is sometimes a sign of perforation of the bronchial wall. Dysphagia from pressure on the œsophagus is also a symptom in this group, sometimes, indeed, the only one. It is not, however, invariably present. Vomiting, cardiac palpitation, with irregularity, and syncope when present are also ascribed to the pneumo-gastric involvement. Pressure upon the azygos veins may cause œdema of the upper part of the abdomen and serous effusion in the chest, while pleural effusions are also due to complicating inflammations or neoplasms of the pleura. Fever may also be a symptom of tumor of the posterior mediastinum. It is usually moderate, but is sometimes high and irregular followed by sweating. On the other hand, there may be lowered temperature, as in tumor of the anterior mediastinum from impeded circulation.

Cachexia is much more frequent in this group of symptoms, as might be expected from the greater severity and disturbing effect of the disease, including, as it does, destructive process involving bone and lung structure as well as severe and deep-seated pain.

It is evident that in this group the *physical signs* play a secondary rôle, and except as the result of modified breathing by pressure and impairment of resonance to percussion have little significance.

III. *Tumors Originating in the Pleura and Lung.*—The former is the more frequent starting-point, but the underlying lung is usually soon invaded and may be more frequently the actual starting-point than is commonly supposed. Naturally the symptoms first produced are those of pleurisy, and the disease is generally so regarded at first, being characterized by the comparatively sudden onset, sharp pain, cough, embarrassed breathing, and pleuritic effusion. Instead of abating ultimately, as is the course in pleurisy, these symptoms grow worse, especially the pain, which extends along the intercostal nerves and their distribution and to the neck and arms. The cough also persists while the expectoration may become bloody and include sometimes cells from the morbid growth.

Paracentesis, too, is successful, and often furnishes in the peculiarity of its product valuable aid in the diagnosis, because, instead of being clear or

nearly so, it is apt to be bloody or slightly chyliform from the presence of fatty matter. This fatty character has been found where there was cancer and sarcoma. The diagnostic importance of certain large, swollen cells of endothelial nature, which seem to become detached and transformed only in case of pleuritic disease of malignant character, is insisted upon by Fraenkel. To the information gained from the fluid obtained by tapping is added also unusual resistance to the trocar and imperfect relief to the dyspnœa. Rapid emaciation, anæmia, and cachexia complete the picture, while all doubt is removed if secondary growths make their appearance in the lungs, as not infrequently happens.

Diagnosis.—In view of the similarity of symptoms to aneurism, the history of the case in mediastinal disease becomes of the utmost importance, but shortness of breath, the bulging of the thorax, irregular outline of percussion dulness, the feebleness of breathing sounds, the dislocation of the heart and sometimes of the abdominal organs, the symptoms of venous engorgement, which are usually more marked in mediastinal disease, the more rapid course, and secondary metastatic deposits are strong points in favor of the latter as contrasted with aneurism. Laryngoscopic examination with a view to discovering any constriction of the trachea from pressure by the tumor may be availed of. The subjects of mediastinal disease are usually younger than those of aneurism. Bony erosion and pain are less frequent. Constitutional disturbance and emaciation are more marked.

Confusion with pleurisy and pericarditis is a natural error, when the symptoms involving the pleura and pericardium are recalled, and here the slower development of the symptoms associated with those of compression of the various mediastinal tissues and absence of tendency to improve should lead to suspicion of the true nature of the disease. Diastolic shock is never present in mediastinal disease, while the pulsation if present is not expansile.

The nature of the tumor may even be suspected from certain features. Thus to malignancy point rapid growth, metastatic deposits in the glands of the neck and apices of the lungs, cachexia, and tumors in other situations. Especially may sarcoma be suspected if the subject be a youthful one. Abscess may be suspected if there is a history of injury, caries, or pyemia, or if there is abscess of the lung or empyema attended by the supervention of pressure symptoms. Hemorrhage may be suspected also when there is trauma and the symptoms develop very rapidly.

Treatment.—There is no treatment for mediastinal disease, except such as may suggest itself for the palliation of symptoms.

MEDIASTINAL ABSCESS.—Separate mention should be made of mediastinal abscess, since it is relatively not such a very rare disease. Out of Hare's 520 cases of disease of the mediastinum, 115 were abscesses, as contrasted with 134 cases of cancer and 98 of sarcoma, 21 cases of lymphoma, seven of fibroma, 11 of dermoid cyst, eight of hydatid cyst, with isolated cases of gumma, chondroma, and lipoma.

The abscesses were found in the majority of instances in males, mostly in the anterior mediastinum, and most could be traced to traumatic causes. Other causes were tuberculosis, the eruptive fevers, and erysipelas. A few cases of mediastinal abscess also originate in the bronchial and

tracheal lymphatic glands, as tubercular lymphadenitis. In 54 cases the abscess was acute.

Of *symptoms*, *substernal pain*, sometimes throbbing, was the most conspicuous. To this was added *fever* in acute cases. Sometimes also *chills* and *sweats*. Erosion of the sternum and burrowing along a rib into the abdomen were noted, also rupture into the trachea and œsophagus. The pus may also become inspissated, cheesy, in chronic abscess. Suppurative lymphadenitis has been known to terminate thus, previous symptoms having been masked by the lung affection. Rarely are we able to detect fluctuation at the edge of the sternum and in the supra-sternal notch, where there may be pulsation. Only as the abscess becomes large enough to encroach upon the air-passages does it cause dyspnœa.

The *physical signs* are not distinctive. They are essentially those described in the general description of mediastinal disease. Fever, throbbing pain, fluctuation, and the history of trauma are symptoms, which, if added, aid the diagnosis.

As to *treatment*, given a correct diagnosis, operative interference is justified, and likely to afford relief if the pus is reached.

Simple Lymphadenitis.—This probably occurs, to a degree, in all inflammatory affections of the bronchi and of the lungs, but is rarely recognizable. The glands are mostly in the posterior mediastinum, and their enlargement may be appreciable to percussion in the upper interscapular region behind, though lymphatic enlargement may contribute also to dulness in the region of the manubrium. Tuberculosis may affect these glands.

SECTION IV.

DISEASES OF THE HEART AND BLOOD-VESSELS.

CARDIAC ASTHMA.

Definition.—The term cardiac asthma is applied to any shortness of breath which is the result of deranged cardiac action. Its frequent association with various forms of cardiac disease demand its preliminary consideration. It differs essentially from bronchial or spasmodic asthma :—

(1) In that there is no spasmodic contraction of the bronchial tubes, and

(2) In that the essential morbid change is an overfilling of the pulmonary capillaries, which, intruding on the lumen of the air vesicles, cuts off the access of air to the blood, causing an *ασθμα*, or panting, an effort by frequent and deep breathing to accomplish aëration. The overfilling of the pulmonary capillaries is most frequently due to a backing of blood from the left heart into the lungs because of valvular insufficiency.

The same results follow in the so-called *paretic* cardiac asthma due to dilatation of the left ventricle. This is a common condition of the senile heart in consequence of its imperfect nourishment and is especially prone to happen when such feeble heart is forced to overcome an unusual resistance. Such is the increased arterial tension due to arterio-sclerosis and chronically contracted kidney, both of which are often associated with shortness of breath. In both the blood does not pass as freely as it ought from the arteries into the veins. So long as the heart is well nourished it hypertrophies in these diseases, but as soon as its nutrition fails it slowly undergoes dilatation, the blood is backed into the lungs, and the asthma occurs and overcomes the resistance.

Again, the equilibrium of the circulation may also be lost by powerful contraction or over-action of the left ventricle, by which the blood is forced too rapidly into the capillaries and veins and thence into the pulmonary capillaries. This more unusual cause operates at times under the influence of mental emotion, possibly also in contracted kidney and arterio-sclerosis, while the heart is still strong,—it may be said, over-strong.

Diagnosis.—The recognition of cardiac asthma as contrasted with bronchial asthma is easiest made by auscultation, which notes the wheezing râles of the contracted bronchi in the latter, but finds at first no alteration in the vesicular murmur of the so-called cardiac asthma. Later, if œdema of the lungs occurs, there may be small, moist râles, crepitant

or sub-crepitant, and still later, if the air vesicles fill up, bronchial breathing with an impairment of resonance. Percussion in the beginning elicits resonance in both conditions. In bronchial asthma if there happens to be, as, indeed, is often the case, associated emphysema, there is hyper-resonance. Under these circumstances the normal areas of cardiac and hepatic dulness are diminished. In a less degree the same may happen with cardiac asthma. Bronchial asthma is an asthma of expiration, as spasm of the larynx furnishes an asthma of inspiration. In cardiac asthma both inspiration and expiration share in the over-effort to accomplish the perfect aëration of the blood. Cardiac asthma and bronchial asthma are sometimes associated.

Treatment.—The treatment of cardiac asthma is, of course, the treatment of the diseases which are responsible for it.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition.—An inflammation of the serous covering of the heart and of its reflection on the inner surface of the pericardial sac.

Etiology.—By far the larger number of cases of pericarditis are due to some poisonous substance present in the blood in the infectious diseases, or to some excrementitious matters which accumulate in the blood as the result of deficient elimination. Other cases arise *per contiguum*, a few cases are traumatic, and those that cannot be accounted for are called idiopathic. Acute articular rheumatism or its cause is by far the most frequent etiological factor, from 30 to 70 per cent. being ascribed to it. The greater the severity of the primary disease the more the chances that the complication, pericarditis, will occur; yet it arises also in the mildest case, and has sometimes even preceded the rheumatic attack. It may be that certain seeming idiopathic cases are due to the cause of rheumatism which spends itself on the pericardium instead of the joints. Other infectious diseases causing it are pyæmia, scarlet fever, typhoid fever, diphtheria, and even measles. Bright's disease is one of the best recognized causes of pericarditis, and it is probably the poisons which accumulate in the blood in this disease which are responsible for it. Such dyscrasic states of the blood as are represented by scurvy and purpura hemorrhagica play an important part in its production. Tuberculosis of the pericardium is a common cause of pericarditis. Tubercular pericarditis may be part of a general tuberculosis or a secondary infection from the lungs.

Adjacent diseases which cause pericarditis are chronic valvular disease, pleuro-pneumonia, pleurisy, especially tubercular pleurisy, morbid growths in the vicinity; ulcerative disease of the œsophagus, bronchial glands, and bronchi; disease of the vertebræ, ruptured aneurism, abscess of the heart, or invasion of the pericardium by suppuration through the diaphragm.

Morbid Anatomy.—The appearances vary with the stage of the disease. Ordinary acute pericarditis is met with in one of three stages. The first is represented by hyperæmia and alteration of the epithelium with transudation and exudation from the hyperæmic vessels. The initial phenomena are hyperæmic redness, followed by roughness caused first by loosening and detachment of the epithelium, further increased by deposits of fresh inflammatory lymph. This lymph is spread in yellow flakes over the surface of the pericardium or floats in the transuded serum.

From this point onward morbid appearances vary with the mode of termination. This may be by resolution, when the products described undergo fatty degeneration and are absorbed, restoring the normal state. More frequently there supervenes the second stage, in which the liquid transudate increases, separating the two surfaces of the pericardium and distending it. This transudate is a clear, straw-colored fluid, the quantity of which varies greatly, amounting sometimes to a liter (1 ¾ imperial pints) or more. In favorable cases it is re-absorbed, the two surfaces are re-apposed and more or less closely united. Sometimes this union extends throughout so that the two surfaces are with difficulty separable, or it may be partial by bands of varying length. At other times the second stage is represented by immediate organization without intervening transudation —primary adhesive inflammation.

Then, again, union being apparently prevented by the constant motion to which the two surfaces are subjected, organization takes place without union of the opposing surfaces and a peculiar villous product results, characterized by numerous projections uniform in size and shape, resembling closely the papillary projections on a sheep's tongue. These papillæ, composed of vascular connective tissue, originate in the usual way by an outgrowth and vascularization of the connective tissue of the serous membrane, and not by organization of the exuded lymph, as formerly supposed. This undergoes fatty degeneration and absorption.

The more unfavorable cases terminate in suppuration, which may also be primary or secondary. In the former instance there is at once a rapid outwandering of leucocytes and the formation of a purulent fluid in the pericardium—pyo-pericardium. In the secondary form the clear, serous transudate is substituted by pus, an event which is usually ushered in by a chill and is followed by hectic fever. The cause of the suppuration in either case is the access of the usual pus organisms, the streptococcus and staphylococcus. The contents of the pericardium may become cheesy, especially if the inflammation is tubercular.

Symptoms.—Clinically, as well as anatomically, we seek to separate the stages, first of roughening, second of effusion, and third of absorption, chiefly by aid of the physical signs.

Pericarditis is sometimes ushered in by a *chill*. More frequently a sharp *pain* in the region of the heart initiates the attack, previous to which there may, however, have been a sense of *discomfort* or distress about the organ, which may, indeed, be the only subjective symptom. The pain and discomfort may be referred to the epigastrium. To these symptoms may be added *dyspnœa*, or orthopnœa. There is also *fever*, which is not very high, temperature 102.5° F. (39.9° C.), unless there be previous disease with fever, when the pericardial complication adds an

increment. The *pulse* is frequent, the patient restless and uncomfortable. There is often *tenderness* over the region of the heart, which may be brought out by percussion. The *position* assumed by the patient varies. Sometimes he may prefer to lie on the affected side, at other times on his back or on the right side, or he may prefer to sit up. Finally there may be no symptoms additional to the primary disease, in which case the pericarditis must be discovered only by the physical examination, or may escape detection altogether until the necropsy reveals it.

As the effusion encroaches on the lung, the difficulty in breathing increases, dyspnœa becomes more marked, the action of the heart more disturbed, frequent, and irregular. When very large it may encroach upon the lower lobe of the left lung adjacent, and by compressing it influences the percussion note in the lower axilla and toward the angle of the scapula. Thus the normal resonance may be impaired or the note may be vesiculo-tympanitic (Skodaic) by relaxation, while the vesicular breathing is feebly heard. The natural state of the lung may be in part restored by changing the position of the patient, causing him to lean forward or to lie on his right side. Still larger effusions produce dysphagia in consequence of encroachment on the œsophagus. Aphonia may occur from pressure on the recurrent laryngeal nerve. The pressure of the full sac on the aorta may produce the *pulsus paradoxus* of Kussmaul, in which the pulse-beat is weakened and more frequent during inspiration.

Physical Signs.—The physical sign characteristic of the *first stage* is the friction sound. It may be associated with an impulse which may be stronger than natural. The friction sound is of the greatest importance in diagnosis. It is a superficial to-and-fro sound heard directly under the ear, commonly loud and rasping, never blowing, sometimes creaking. It is most loud over the middle of the heart. It is not synchronous with the normal heart sounds and not conducted in the direction of the blood current. It is, however, often influenced by changes of position or by breathing. The rub may sometimes be felt by the hand placed over the heart. It is generally of short duration and disappears with the filling of the pericardium by effusion. It may sometimes be brought out or intensified by having the patient lean over on the stethoscope.

The *second stage*, or that of effusion, has usually, but not always, signs discoverable to *inspection* or *palpation*, or both. There must be an amount of effusion sufficiently large to do this. The præcordium then may be bulging, the interspaces obliterated, and the impulse undulating, tumultuous, and indistinct. As the effusion increases the heart gets further and further away from the chest wall and assumes a more horizontal position, while the impulse, feebler and feebler to vision and touch, may disappear altogether. *Percussion* furnishes the most striking change. The area of dulness is enlarged, and peculiarly enlarged. It becomes rudely triangular, with the apex toward the inner end of the left clavicle and the base as low as the 7th rib, and extending in extreme cases from nipple to nipple, even pushing the diaphragm and liver downward. *Auscultation* confirms palpation. The friction sound is no longer heard. The impulse is feeble, indistinct, and often tumultuous. The heart sounds are indistinct and best heard at the top of the sternum.

The *third stage* represents a return to the normal state of affairs, which may be by the intermediation of a *friction redux* or not. Or adhesions may form between the heart and the sac, embarrassing its movements permanently, and producing retraction of the chest wall with systole. On the other hand, necropsy has often revealed close adhesions between the heart and the pericardium which were not suspected during life. Permanent roughening, represented by the "sheep's tongue" surface or other roughening or adhesions, may produce permanent friction sound, and the pericarditis is chronic.

Physical Signs of Chronic Adhesive Pericarditis.—These differ materially. They are most easily studied in children, in whom the condition is especially apt to occur after rheumatism. To *inspection* the impulse is more diffuse, extending sometimes from the 3d to the 6th interspace. It may be displaced in various degrees from its natural site; it may be to the right of its normal position and above it or down toward the epigastrium. It is sometimes multiple or spreads in a wave-like manner over the area named. At others the systole is associated with a retraction of the chest wall, which is especially evident in thin persons and is the most valuable sign of adhesion of the pericardium. This may be followed by a rapid rebound of the chest walls, known as the diastolic shock. It may be associated with a coincident collapse—the diastolic collapse of the cervical veins, due to a sudden emptying of these vessels consequent on the expansion of the chest wall, which was first described by Friedreich.

To *percussion* there is an increase in the normal area of cardiac dulness, usually upward and to the left, sometimes as high as the 1st interspace. Often the pericardium is adherent to the adjacent pleura, in which event the area of cardiac dulness is not influenced by deep breathing, a fact pointed out by C. J. B. Williams as of great value in diagnosis.

Auscultation may be entirely negative or there may be a modification of the usual friction sound which closely resembles the creaking of leather. A galloping or fœtal rhythm may be present, or there may be a loud systolic murmur at the apex which has often given rise to the erroneous diagnosis of mitral valve disease.

It is in adhesive pericarditis, too, that we sometimes have the *pulsus paradoxus*, described on p. 510. This happens when the aorta is dragged upon by cicatricial tissue during inspiration. It happens, too, in *mediastino-pericarditis*, when the traction on the mediastinal space during the inspiratory act drags on the aorta, compresses it, and makes small the pulse. This is more frequently demonstrable by the sphygmogram, but in extreme cases may be appreciated by the finger. It is not a pathognomonic sign of either event, but if associated with an inspiratory distention of the cervical veins it points strongly to adhesive pericarditis. Notwithstanding these signs chronic adhesive pericarditis is often recognized the first time at necropsy.

Diagnosis.—In all cases of acute articular rheumatism the heart should be frequently examined, because pericarditis often supervenes with feebly pronounced subjective symptoms. At the outset the distinction is to be made between pericarditis and acute endocarditis which even more frequently succeeds on rheumatism with subjective symptoms no more dis-

tinctive. There is not usually much difficulty. The to-and-fro rhythm, heard directly under the ear, usually most distinct over the centre of the heart, and the absence of transmitted sounds in accordance with the laws of transmission of the valvular abnormal sounds are distinctive features of the cardiac friction. If, however, one of the to-and-fro elements is wanting, the difficulty is greater and errors do occur. Close study must be made as to transmission. It is further characteristic of the friction sound that it is increased in loudness by pressing the chest wall with the stethoscope, while this is not the case in endocardial murmurs. This act is, however, often painful to the patient. In chronic valvular defects there are changes in the size and position of the heart which are not present in the first stage of acute pericarditis. Where both acute endocarditis and pericarditis are present the difficulty is greatly increased and one or the other condition is likely to be overlooked.

The pleuro-pericardial friction sound or "extra pericardial" friction sound is to be distinguished from pericardial friction. It is a sound similar in rhythm to the pericardial sound, but the primary conditions of its causation are in a pleuritis involving the opposed surface of the mediastino-costal sinus of the left side. It is more commonly heard, therefore, over the left border of the heart. It is the combined product of the respiratory and cardiac action, being usually louder during expiration. It generally ceases during a deep inspiration, because at this time the cardiac action cannot produce the required rubbing. On the other hand, sometimes this is the very condition under which the friction is loudest. Simply holding the breath may also stop it, though not necessarily because the heart motion produces it. This influence of the breathing one way or the other is, however, of importance in diagnosis, while other symptoms must also be taken into consideration. Thus if it be a pleurisy the pleural friction is probably heard elsewhere, and there are the other symptoms of a pleurisy present, while those of a pericarditis are absent. Unlike true pericardial friction the pleuro-pericardial friction is uninfluenced by bending the body forward, but is heard with equal distinctness in any situation of the body. Difficulties again increase when it is associated, as it sometimes is in a pleuro-pneumonia, with endocarditis. It also occurs in tubercular phthisis, where it is sometimes associated with a systolic click due to the simultaneous expulsion of a bubble of air from a portion of softened lung.

For diagnosis between pericarditis with effusion and dilatation of the heart, see p. 551.

Prognosis.—The course of pericarditis varies with different cases. In an ordinary uncomplicated case passing to recovery, the duration is one to three weeks, even where there is considerable effusion, which is often absorbed with surprising rapidity. In other cases, especially in cachectic subjects, the duration is longer. Relapses occur. Where adhesion results, convalescence is greatly prolonged, and in many cases the heart is permanently crippled. On the other hand, extensive adhesions are sometimes found at necropsy where no lesion was suspected. The pyo-pericardial cases are usually fatal.

Treatment.—Prompt treatment is of the greatest importance in pericarditis. Rest is an absolutely essential condition. As soon as the

diagnosis is made a blister is of the greatest value. There is no other disease in which I am so satisfied of the efficiency of a blister. It should be at least three inches (7.5 cm.) square. I am confident that it helps to prevent effusion and also to promote the absorption of effusion. Along with this, measures to relieve pain are indicated. Nothing is so satisfactory as moderate doses of morphine administered hypodermically associated with atropine in the proportion of $\frac{1}{150}$ grain (0.00044 gm.) of the latter to ¼ grain (0.0165 gm.) of the former. Cold applications to the pericardium in the shape of Leiter's coil or the ice-bag are sometimes useful. Sometimes, on the other hand, hot applications are more comforting.

Digitalis in moderate doses is usually indicated to steady the heart and keep it at work without, however, stimulating it too forcibly. For the same reason alcohol and ammonia, especially the aromatic spirit of ammonia, are indicated. Liquid food, including milk and broths, should be adhered to until convalescence is established. Eggs may, however, be early allowed.

If the effusion is very large, tapping the pericardium may be necessary to relieve the patient, although practically the relief which first follows a successful operation is rarely followed by complete recovery. The aid of the surgeon should be secured if possible, but if not, puncture may be made in the 4th interspace an inch (2.5 cm.) to the left of the edge of the sternum. If made in the 5th interspace, the puncture should be made a little further out, say 1½ inches (3.5 cm.). A safe point which may be used in large effusions is the left xiphocostal angle, at which the needle should be pushed upward and backward. Where the pericardial fluid is pus, a simple tapping is insufficient. Free incision should be made and free drainage should be established with aseptic precautions. It is not impossible that if operation were done earlier, better results would follow.

The treatment of chronic adhesive pericarditis is mainly symptomatic, and directed to building up the strength of the patient.

OTHER PERICARDIAL AFFECTIONS.

Other affections of the pericardium are hydropericardium, hæmopericardium, and pneumopericardium.

HYDROPERICARDIUM.—This term is applied to large accumulation of serous fluid in the pericardium. In health, the pericardium is simply lubricated by this fluid. It occurs sometimes as a part of a general dropsy, most frequently cardiac dropsy, more rarely in renal dropsy. The accumulation is seldom large in these cases. It is not common, but is sufficiently so to demand frequent examination of the heart, as it is often overlooked. Its signs are the same as those of the inflammatory effusion.

HÆMOPERICARDIUM, or blood in the pericardium, occurs only as a result of rupture into the pericardial sac of an aneurism in the first part of the aorta; from rupture of the heart itself or a wound of the heart. It is rapidly followed by shock and death. Its physical signs are those of effusion. Cancer of the pericardium may be associated with blood effusion, which may also be tubercular.

Pneumopericardium, or gas in the pericardial sac, is a rare condition analogous to the much more common one of pneumothorax. As in pneumothorax, the presence of air implies also the presence of liquid, and that usually pus. It is produced by similar causes, such as perforation into an air-containing space like the lungs or œsophagus. Such perforation is usually traumatic. Decomposition of pericardial exudate or morbid growth, it is said, may also produce it.

Symptoms.—Its symptoms are pain and pericardial embarrassment, but the physical signs are most distinctive, especially those of auscultation. To inspection there is prominence of the pericardium with indistinctness or obliteration of apex beat, restored by the patient's bending forward. Percussion furnishes dulness over the lower portion of the cardiac area and tympany above it, the position of both being altered by change in position of the body. To auscultation the heart sounds assume a striking metallic character, being audible even at a distance from the body. A similar metallic character is given even to a friction sound if it is present, as it often is.

Diagnosis.—The diagnosis of this condition requires differentiation from the effect of an air-dilated stomach on the heart sounds or rarely of a phthisical cavity or pneumothorax. All doubt in the case of the stomach is removed by filling it with water. The associated symptoms of the other conditions make a mistake unlikely.

Treatment is scarcely available except in case of external injury, where operation may be of service.

DISEASES OF THE ENDOCARDIUM.

ENDOCARDITIS.

Definition.—Endocarditis in both its acute and chronic forms is for the most part an inflammation confined to the valves, whence for such inflammation *valvulitis* is a more correct term. The lining of the cavity of the heart is, however, sometimes affected in acute endocarditis, especially the more severe cases, when it is known as *mural endocarditis*. It is usually in the apex of the left ventricle that such inflammation occurs.

Etiology.—All acute endocarditis in the light of modern studies must be regarded as infectious, that is, as due to a specific poison commonly associated with some disease which is regarded as the cause of the endocarditis. Acute rheumatism is the best recognized and most frequent of these. Upon this follow closely the infectious fevers with their various specific organisms. To these latter must be added the toxic substances now known to be the product of such bacteria, although in the disease which is acknowledged to be the most common cause, acute articular rheumatism, no causing organism has as yet been found.

There is, however, a great difference in the severity of different cases of acute endocarditis, and the disease is easily separable into two classes, from one of which recovery almost always takes place up to a certain

point, leaving, however, a certain degree of valvular defect, known as chronic endocarditis, while the other is invariably fatal. The first or milder of these classes was for a time ascribed to some specific non-organized agency, even after the more severe and fatal form was recognized as infectious, whence arose the terms simple endocarditis on the one hand, and infectious, ulcerative, malignant, or mycotic on the other.

In attempting to explain why now the simple form and now the virulent form of endocarditis arises, it may be stated that the toxins generated by the less virulent bacteria pave the way for the operation of the virulent streptococcus pyogenes, staphylococcus, pneumonococcus, and other organisms which are found in the morbid products of malignant endocarditis; and it is not unreasonable to suppose that the former produce the simple form of endocarditis, while the coöperation of the septic bacteria named is necessary to produce the malignant variety.

The Mild or Simple Form of Acute Endocarditis.

Etiology.—Almost any one of the recognized infectious diseases may become a cause of simple endocarditis. Acute articular rheumatism is, however, the most frequent cause, 20 per cent. of all cases being ascribed to it. After this come diphtheria, scarlet fever, and peliosis rheumatica; less frequently typhoid fever, erysipelas, small-pox, and pneumonia. The frequency with which chorea is followed by acute endocarditis has attracted a good deal of attention lately. William Osler, for example, who has made the subject a special study, says, "There is no disease in which, post mortem, acute endocarditis has been so frequently found." This fact has suggested even a microbic origin of chorea which is sustained by other features in the history of the disease, but not by the essential demonstration. In the absence of such demonstration chorea must be regarded in the light of a predisposing cause. Cachectic states, such as are caused by tuberculosis and cancer, also seem to favor the development of acute endocarditis. Finally, chronic valvulitis is a predisposing condition to simple acute endocarditis as well as to the malignant form, being often complicated by acute attacks, whence the term "recurring" endocarditis.

Morbid Anatomy.—The left side of the heart is most frequently involved, and in this the mitral leaflets first, in at least half of all cases; next the aortic cusps, then the tricuspid valve, and finally the pulmonary. In embryonic life, in which acute endocarditis also occurs, the right side of the heart and the tricuspid valve are most frequently affected, accounting thus for certain congenital valvular defects.

The type of the morbid change on the valves in simple endocarditis is so constantly a product warty or fungous in appearance, that the term *warty* or *verrucose* endocarditis is often applied to this form. On the auricular surface of the mitral and the ventricular surface of the aortic valves, at the line of their contact margin, *i. e.*, $\frac{1}{25}$ to $\frac{1}{12}$ inch (one to two mm.) back of the valve edge, granular and warty excrescences make their appearance, rising $\frac{1}{12}$ to $\frac{1}{8}$ inch (two to three mm.) above the surface and extending a variable extent along the valve. These soon become capped with fibrin, often abundantly, and thus a vegetation is formed.

They arise by a proliferation of the cells of the adventitia and of the connective tissue of the external laminæ of the endocardium. The vegetation thus formed is a friable product, liable to be broken off at any time and carried into the general circulation to a point of lodgment, where it plays the rôle of an embolus. In point of fact, however, this accident does not often happen in the acute endocarditis succeeding febrile diseases. It occurs more frequently in the acute endocarditis engrafted on chronic valvular disease. More frequently the vegetation undergoes organization and contraction and the valve is restored partially to its natural condition, leaving a simple sclerotic thickening which is especially prone to become the starting-point of new processes. But not every aortic murmur in the course of rheumatism implies endocarditis, as the condition of the blood predisposes to a hæmic murmur ; nor every murmur at the apex, because the state of the muscle predisposes to imperfect closure of the auriculo-ventricular orifice. Unless there has been previous valvular disease of the heart there is no enlargement in acute endocarditis, so that neither palpation, inspection, nor percussion gives any information.

Symptoms.—These are often masked by those of the previous disease, and sometimes overlooked, the autopsy first disclosing the lesion. There is frequently noted, however, greater or less *embarrassment of breathing*, orthopnœa being not infrequent, the *pulse* is much more frequent and may be *irregular*, the patient is restless, the countenance dusky, while the *temperature* is a degree or two higher. Altogether, it is plain that he is sicker. Yet there is no actual pain, as in pericarditis.

Physical Signs.—In first attacks of endocarditis there is no notable enlargement of the cardiac areas as determined by percussion. *Auscultation* recognizes a murmur in the majority of instances in the mitral area well conducted into the axilla, usually soft and blowing, at times quite harsh. Very rarely is there a presystolic murmur, though its more frequent occurrence might be expected from the nature and situation of the lesions described. When the lesion is at the aortic orifice the murmur is heard in the aortic area at the 2d interspace at the right edge of the sternum. Basic murmurs also occur in the pulmonary area to the left of the sternum, which are functional in origin.

Diagnosis.—This is based almost entirely upon the physical signs and the etiology, as none of the symptoms mentioned are pathognomonic. The systolic murmur is not always due to regurgitation. The same excrescences which grow on the valve leaflets may also attach to the papillary muscles and chordæ tendineæ as well. Mitral regurgitation may also occur in rheumatism and in other acute febrile diseases from myocardial changes, as the result of which the basal part of the cardiac muscle is enfeebled and unable to do its part of the work of closure of the mitral orifice, and the valve leaflets are insufficient to complete it. Some of the cases of murmur which disappear with recovery may belong to this category.

The distinction of the endocardial from the pericardial murmur was considered in treating of pericarditis. The more superficial situation of the latter over the body of the heart, its to-and-fro rhythm not connected with the heart sounds, its failure to follow the usual laws of conduction, and the fact that it is made more pronounced by pressure all serve to dis-

tinguish it. A. E. Sansom calls attention to a possible source of error in a pericardial roughening at or about the apex, especially in children, which causes a systolic apical murmur. This should be remembered as a possible but rare occurrence.

Prognosis.—The subject of the simple form of acute endocarditis rarely dies, but the chances are many that he gets up with a damaged heart, in other words, that chronic valvular disease results. This is not, however, always the case, for it is not impossible for complete recovery to take place. On the other hand, some of the instances of complete recovery after mitral regurgitant murmur belong doubtless to the category described of insufficiency due to myocardial defect without mitral lesion. It should not be concluded, however, that because a murmur has disappeared the patient has certainly recovered, since the disappearance of a murmur due at first to myocarditis may be succeeded by another true valvular murmur, which is the result of contraction of cicatricial tissue in the valve. Finally, one acute attack from which recovery has taken place is liable to be succeeded by another and another, so that sooner or later chronic valvular defects are produced.

Treatment.—The key-note of a proper treatment of an acute endocarditis is absolute quiet. It is not often that much else is required. A blister is not of the signal service here that it is in pericarditis, while digitalis is not indicated unless there is irregularity, when the dose should be moderate, only enough to steady the heart. Dyspnœa is best treated by sufficient doses of opium or morphine. The diet should be easily assimilable and liquid until convalescence is established.

The Severe or Malignant Form of Acute Endocarditis.

Synonyms.—*Ulcerative, Infectious, Mycotic, or Diphtheritic Endocarditis.*

Definition.—Malignant endocarditis is an acute infectious fever due to inoculation of the blood by a bacillus, and characterized locally by a specific valvulitis.

Historical.—It was recognized as a separate form of disease in 1851 by Senhouse Kirkes, and further studied by Charcot and Velpeau in France, Virchow in Germany, and recently in this country by William Osler, who made it the subject of his Gulstonian lectures before the Royal College of Physicians, England, in 1885. Its mycotic nature was not suspected until after Koch discovered the bacillus of tuberculosis, in 1882.

Etiology.—Malignant endocarditis shares with the simple form an infectious origin. No satisfactory explanation has, however, been furnished of its more malignant nature. Allusion was made when treating of the simple variety to the suggestion sustained by the experiments of Wyssokowitch, Ribbert, Orth, and others, that a state of the blood due to the toxic effect of bacteria may be the cause of the simple form, and that it may afford conditions favorable for the operation of the more virulent bacteria found associated with the malignant form. It is extremely doubtful whether there can be a primary malignant endocarditis without the intervention of some one of the diseases which usually precede it. The presence of chronic valvular defects affords the most important pre-

disposing cause favoring the operation of the causes of the acute malignant form. Goodhart found it in 61 out of 69 cases, and Osler in 54 out of 209. The latter also found it 11 times at 100 autopsies of fatal cases of pneumonia. Of the infectious diseases associated with the malignant form of endocarditis, pneumonia is the most frequent. The disease occurs also in association with gonorrhœa, rheumatism, peliosis rheumatica, pleurisy, puerperal fever, bone-necrosis, and septicæmia from any cause. More rarely it has been found in connection with meningitis, small-pox, diphtheria, scarlet fever, tuberculosis, and dysentery. Most frequently, perhaps, the micro-organism is the lancet-shaped bacillus of pneumonia, after this pus organisms, the streptococcus and staphylococcus.

Morbid Anatomy.—As to the acute cardiac lesions associated, we find, in addition to the old sclerosis, three sets—vegetative, ulcerative, and suppurative. The vegetative are for the most part made up of closely packed spherical micrococci more or less commingled with small fibrin-masses. The vegetations vary in size from that of a pin's head to a pea, and are reddish-yellow in color. The seat of this vegetation becomes rapidly necrotic and breaks down into an ulcer which may perforate the valve, with or without previous protrusion, the so-called valvular aneurism. More rarely minute foci of pus are found in the deeper tissues of the valve leaflets. The invasion is, however, not always confined to the valves, but may extend to the mural endocardium. Of the valves the mitral is most frequently involved; next the aortic; next mitral and aortic jointly; next the lining of the heart wall; next the tricuspid, and last the pulmonary valve. In a few instances the right heart alone is invaded. Other morbid changes include the lesions of the concurrent affection and the phenomena of embolism due to lodgment of fragments of the vegetation. The result of the latter when complete is a metastatic abscess, though the earlier stages of red infarction may also be present.

The spleen, kidney, skin, and even the cerebral cortex may be seats of embolism. In addition to these, we may have also embolism and hemorrhagic infarct occurring in the lungs from emboli starting in the right heart, as contrasted with those originating in the left heart which lodge in the systemic circulation. The number of embolisms varies greatly in these cases. They may be altogether absent, while they may be counted by hundreds, in which event they are, of course, very small.

Symptoms.—Given a pneumonia, pleurisy, the puerperal process, or any one of the diseases named with the supervention of *chills*, followed by *fever* and *sweats*, this form of heart disease should be immediately thought of and the organ carefully examined for the auscultatory signs of endocarditis. In the primary form, should this exist, we have not even the presence of one of the diseases named to suggest the occurrence of ulcerative endocarditis. In this form particularly the resemblance to intermittent fever seems at first close, but a careful study of the temperature chart from day to day, and, above all, utter failure of the antiperiodic remedy to produce any effect, will in a short time show that the malarial disease is not present. Doubtless, often, the malady under consideration has been mistaken for intermittent fever, and not without reason, for many a case of irregular quotidian and tertian fever presents symptoms not more diagnostic; but the regular, almost rhythmical, rise and fall of temperature

as exhibited in the chart of an intermittent fever is wanting. Indeed, I think there is no disease in which the extreme irregularity in temperature reaches that of the one under consideration, as a careful study of the appended temperature chart from a case of my own in the University Hospital will show. Note that the maximum is reached at any time of day or night.

It always greatly aids the diagnosis when to chills and fever are

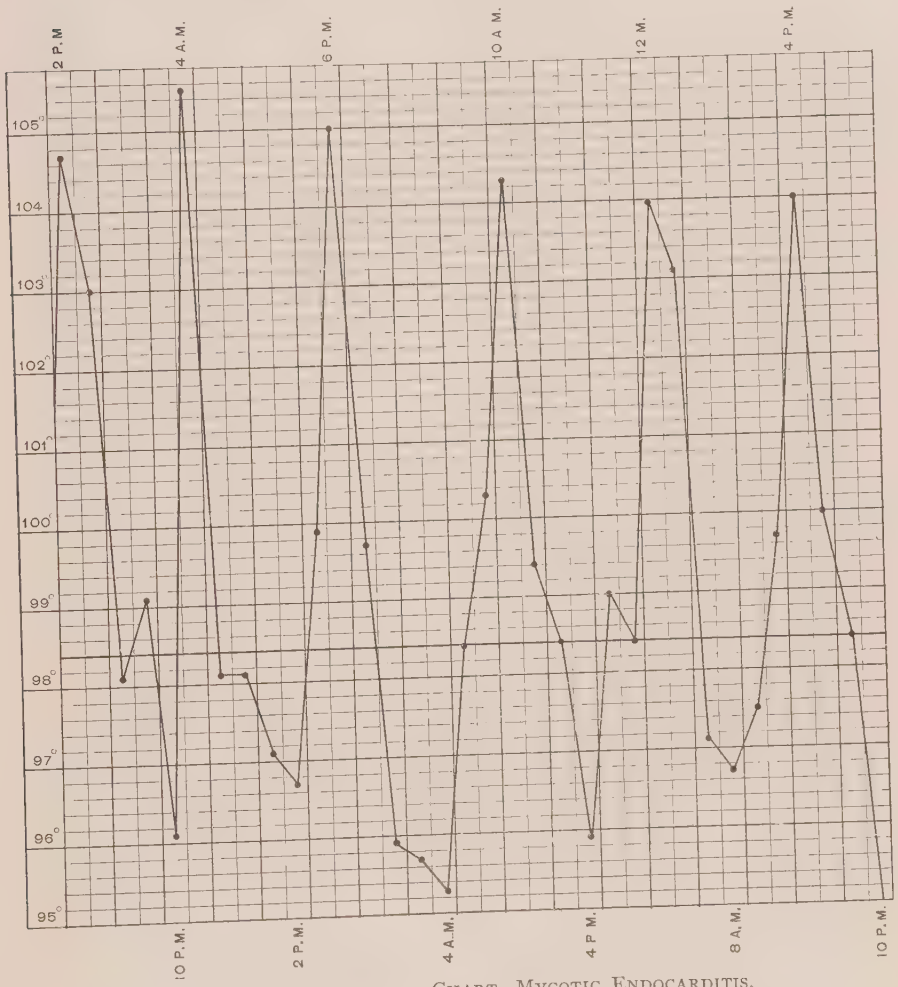


FIG. 42.—TEMPERATURE CHART, MYCOTIC ENDOCARDITIS.

added other symptoms suggesting *embolism*, which so frequently occurs. The occurrence of a hemiplegia, pain in the region of the spleen, with increased dulness on percussion, pain in the region of the kidney, with hæmaturia, or a *sudden blotch in the skin* of the kind described, is of inestimable value. More rare symptoms of similar origin are *impaired vision* from retinal hemorrhage, *parotitis*, and *abscess of the parotid gland*. The symptoms are not always as pointed as detailed, while they may include

others not mentioned. The fever may not be as high, but it is always present; again, it may not be remittent, but continuous. There may be *jaundice*, *præcordial oppression*, *shortness of breath*, while heart symptoms may be altogether absent, when it is almost impossible to distinguish the disease from a septic fever of the ordinary kind. The *pulse* and *respirations* are invariably accelerated. *Albuminuria* and *casts* occur in all forms, either as the result of acute nephritis or of renal embolism.

A further study of the symptoms of malignant endocarditis permits their classification into four groups, known as the *septic*, the *typhoid*, the *cardiac*, and the *cerebral*.

The *septic* type occurs in connection with such septic processes as external wounds, the puerperal process, or acute bone-disease with necrosis. The symptoms added are rigor, irregular fever, sweats, and exhaustion. Yet these are only the symptoms characteristic of pyæmia. In fact, it is a pyæmia; and the term *arterial* pyæmia, suggested by Wilkes, is a good one, because the pyæmic abscesses are the result of emboli, starting in the left heart and lodging in arteries. The endocarditis constitutes the distinctive feature of the disease. The resemblance to intermittent fever here exists also, and a quotidian or double tertian type may be simulated.

The symptoms of the *typhoid* type are even more characteristic. We meet here, too, the same prostration, irregular temperature, and sweating; rigor is less frequent and the onset is more gradual. There are delirium, drowsiness, often diarrhœa, with distention of the abdomen and tenderness in the right iliac region, to which a rash may also be added, which, though not identical with that of typhoid fever, is nevertheless similar to it. The tongue is dry and brown, and sordes collects about the teeth. The temperature is remittent, like that of typhoid, reaching 103° and 104° F., and even higher. Here again the heart-symptoms may be overlooked.

In the *cardiac* type are included those cases in which there has been chronic valvular disease, on which has supervened fever, with rigors and sweats, and the symptoms of embolism described.

Still another group is the *cerebral*, in which the symptoms simulate meningitis, basilar or cerebro-spinal, with acute delirium as the distinctive symptom.

Physical Signs.—If there is anything peculiar about the physical signs, it is their want of definiteness. When murmurs are present it is often difficult to locate or time them precisely. They often vary from day to day. They may occur at both base and apex, and with reason, for both sets of valves may be and often are involved. The super-addition of pericarditis adds a further source of confusion in the friction-sound superadded. If chronic valvular disease exists, its signs are also present, including those of hypertrophy.

Complications.—As to complications, these are mainly the original cardiac disease or the diseases that most frequently cause the mycotic inflammation. Pericarditis and pleurisy are frequent complications in the strict sense of the term. So is meningitis. Acute nephritis, the result of the sepsis and quite independent of embolism, may be present, with its characteristic symptoms, albuminuria, blood-casts, and free blood-cor-

puscles. Gastro-intestinal derangements, not of embolic origin, are sometimes conspicuous. Diarrhœa may be especially troublesome.

Diagnosis.—The diagnosis is easiest in the cardiac group. A few days' study of the temperature, with its extreme fluctuations, the rigors and supervening sweats, should at once lead to suspicion, and these, if continued, mean only one thing. The other forms are not so easily recognized; but if one would always remember the possibility of the occurrence of malignant endocarditis in connection with the diseases named, it would be less frequently overlooked. The presence of concurrent disease should guard us against the conclusion that there is typhoid or rheumatic fever. Septic fever is essentially the same in all cases, the heart-symptoms adding the peculiarity. In true typhoid fever there is always splenic enlargement and often parotiditis, so that the presence of these symptoms naturally suggests that disease, and an erroneous diagnosis is not inexcusable. It is said that splenic enlargement is not so marked as in typhoid fever, and that there is commonly more tenderness in mycotic endocarditis. This may be true in some cases, not in others. Rheumatic fever often more closely resembles malignant endocarditis, with its high, irregular fever and copious sweats, while confusion is further contributed to by the fact that endocarditis is one of rheumatism's most frequent complications, the malignant form being, however, more infrequent than the simple. But recurring rigors are not usual in rheumatism. The joint-symptoms of rheumatism are conspicuous at an early stage of the disease; there is no enlargement of the spleen nor symptom ascribable to embolism, unless secondary to endocarditis. The essential identity of ordinary pyæmia and malignant endocarditis has been mentioned, and only the endocarditis and its consequences distinguish the disease from ordinary septic fever.

Prognosis.—The prognosis is always unfavorable, though ulcerative endocarditis may be prolonged for many weeks and even months. Usually, however, five or six weeks measure its course, while some cases are of shorter duration. Eberth reports one fatal in two days.

Treatment.—Treatment, unfortunately, avails little. The patient should be kept at rest. Remedies should be restorative and supporting —quinine, stimulants, digitalis. Nourishing food is indicated. The high temperature may be treated by sponging or by an ice-cap or by Leiter's coils applied to the thorax or abdomen; but high temperature is seldom of so long duration as to require special treatment.

Chronic Valvular Defects.

Synonyms.—*Chronic Endocarditis; Chronic Valvular Disease.*

Etiology.—The majority of chronic valvular defects are the consequence of endocarditis, acute or chronic. It may be that the very first attack of acute inflammation has left the valve leaflets in such a sclerotic condition that they readily become the seat of the subsequent changes which constitute the chronic disease, or that several attacks may be necessary before a permanent effect is produced. On the other hand, we must acknowledge, too, a chronic valvulitis, in which valvular defect is brought about gradually without the intervention of acute inflammation.

This process is analogous to chronic endarteritis, consisting in hyperplasia with fatty (atheromatous) and calcareous degeneration of the new tissue. In fact, a chronic endarteritis may spread from the aorta to the aortic valves. These slowly induced inflammations are variously caused. The rheumatic poison may cause them, as it does the acute forms. Alcoholic indulgence and over-abundant eating, whether by the direct irritation of the substances taken into the blood or through the poison of gout engendered by their use are an efficient cause. Another is prolonged muscular strain, producing over-tension of the valve leaflets. This operates in laborers who do much heavy lifting, and sometimes in athletes. Especially potent is it when, as is often the case, hard muscular work and over-eating and drinking are associated. To these syphilis, also, often contributes a factor in some unknown way. Under all of these circumstances it is the aortic cusps which suffer most.

In addition to these causes, however, incompetency of the cardiac valves is often brought about by dilatation of the ventricles and the great vessels leading from the heart, the valve-leaflets themselves remaining intact. Such relative insufficiency affects most frequently the auriculo-ventricular valves, and as a consequence the latter are not "sufficient" to stretch across their respective orifices and close them. Less commonly the semilunar valves are similarly deficient, more frequently the aortic in dilatation of the aorta, and more rarely also the pulmonary when that vessel is dilated. It should be said of the auriculo-ventricular insufficiency that it is found more frequently in the autopsy-room than recognized clinically, for it does not necessarily cause a murmur.

Morbid Anatomy.—The anatomical condition of the defective valves is made up of five separate factors, each of which may enter more or less into the lesion. This is true both of the auriculo-ventricular and similunar valves. These conditions are :—

(1) Thickening.
(2) Retraction.
(3) Adhesion.
(4) Calcification, and
(5) Atheroma, either alone or associated with calcification.

(1) Thickening is the immediate result of an overgrowth of connective tissue. The slighter degrees are seen along the bases of the aortic cusps and the line of contact in closure of the mitral leaflets. Such degrees do not necessarily impair the function of the valves. More advanced stages produce a distinct thickening and sclerosis of the whole of each aortic cusp and mitral leaflet.

(2) Retraction or curling is the result of shrinkage of this hyperplastic tissue. The three aortic cusps are often reefed back and fixed, though the very edge of the valve may still remain movable. In the case of the mitral valve the tendinous attachments of the papillary muscles often contract and draw the valves into the left ventricle, producing a permanent funnel-like extension analogous to that which takes place in physiological closure of the mitral orifice.

(3) Adhesions. These unite the valve leaflets, increasing their fixedness and rigidity, interfering with complete opening and closure. The right and posterior aortic cusps are most frequently united. Most serious

is the effect of union of the mitral leaflets, which sometimes results in a reduction of the orifice to a mere slit or buttonhole-like opening—the buttonhole mitral orifice.

(4) Calcification or limy infiltration of the valves thus united may succeed in various degrees, producing in extreme cases firm, bony rings which further diminish the mobility of the valves. In less degrees there are formed splinter-like projections into the substance of the valve which also interfere with complete closure and opening; at other times there may be simple marginal deposits which impede in a less degree the function of the valves, or, indeed, may not interfere at all.

(5) Atheroma is also often found at spots on the surface of the valves and at the marginal attachments of the aortic cusp without producing insufficiency.

Still another form of lesion found at necropsy is *rupture of a leaflet* the result of strain. This is perhaps not possible with a sound valve, while one weakened by the morbid states described may give way. The physiological result is insufficiency, while the lumen of the orifice during systole is not encroached upon. Such an accident is not infrequent in acute ulcerative endocarditis, in consequence of erosion and partial destruction of the valve.

Insufficiency of the aortic orifice at the time of life at which it is most common, say middle age, is favored by a gradual widening of this orifice from $\frac{4}{5}$ inch (20 mm.) at birth to $2\frac{4}{5}$ inches (70 mm.) at eighty years.

Congenital defects are relatively common in the right side of the heart, which is the subject also of inflammations during intra-uterine life. The changes resulting from the latter are of the nature of fusions. Such defects also occur rarely on the left side, most rarely in the mitral valve.

Mitral Insufficiency.

Occurrence and Mechanism.—This is the most frequent of the uncombined forms of valvular disease. The valve leaks. The blood flows backward during systole from the left ventricle to the left auricle. The distended auricle, first attempting to resist the backward flow, hypertrophies, but eventually dilates, and the blood is crowded backward into the lungs, which become engorged. The right ventricle, in its efforts to push the blood through the engorged lungs, hypertrophies, and the pulmonary factor of the second becomes louder and sharply accentuated. The compensating effect of the hypertrophied right ventricle for a time arrests the mischief. At this stage, perhaps, begins the hypertrophy of the left ventricle, which in all cases of mitral insufficiency presents itself sooner or later, although at first the double outlet for the blood from the ventricle would seem to demand less strength of the left ventricle. The right ventricle, however, in its hypertrophied state, delivers more blood to the left ventricle, which demands more power to drive it on, hypertrophy results, and thus compensation is a while longer maintained. Sooner or later the right ventricle dilates, the tricuspid valve becomes insufficient, the blood regurgitates into the right auricle and thence into the great veins of the

neck. The valves of these ultimately yield, the jugular pulse appears, and the general venous system is engorged.

Symptoms.—Often there are no symptoms, because for a considerable length of time compensation keeps pace with the development of the disease unless the latter be sudden, as by rupture of a valve leaflet. The first thing noticeable is usually *shortness of breath* on exertion, the so-called *cardiac asthma*. With this is soon associated *palpitation* or "beating" of the heart, which increases and abates *pari passu* with the dyspnœa. Next is *irregularity of heart's action*. This is the beginning of waning compensation, of which the immediate result is congestion of the lungs. Dyspnœa is now permanent. Thence the engorgement extends to the right ventricle and venous side of the circulation, the pressure in the arteries being proportionately less. The lung engorgement invites frequent attacks of bronchitis, which excites *cough* and increases dyspnœa. *Orthopnœa* is frequent at this stage and the patient can only rest sitting in a chair. There is sometimes *blood-stained expectoration* in which may be found alveolar epithelium dotted with pigment granules.

Along with this, or before it, the *liver becomes congested*, enlarged, and tender; the mucous membrane of the stomach also becomes congested, causing *nausea* and *indigestion*. The hepatic enlargement is sometimes very great, and I have known it to be taken for cancer of the organ. The liver is often the seat of pulsation and as often a jugular pulse is seen. Both signs are pathognomonic of mitral regurgitation. In advanced stages the kidneys also become passively congested, the urine scanty, and the specific gravity high, while there are copious deposits of urates. It contains a small quantity of albumin and there may be hyaline tube casts, rarely even a few blood discs. As a secondary result of hepatic engorgement only, there may also be enlargement of the spleen.

Physical Signs.—*Inspection* discovers the apex beat to the left of its normal position in the 5th interspace or perhaps a little lower down. It may be in the line of the nipple or even beyond it, more forcible and diffuse than in health. The outward dislocation of the apex beat is due to the enlargement of the two ventricles. An auricular impulse may be present to the left of the pulmonic area in the 2d interspace, and may be presystolic and active for the auricle that is produced when the auricle contracts; or systolic and passive for the auricle that is caused by a filling of the auricle by regurgitation from the ventricle during the latter's systole. A bulging præcordium may be looked for in young persons in the 2d and 3d interspace, to the left of the sternum; also to the left of the lower sternum from hypertrophy of the right ventricle. In advanced stages there is a jugular pulse, which is pathognomonic.

On *palpation* the apex beat is found more forcible than normal, at least while compensation is maintained, and there may be a pulsation near the ensiform cartilage caused by the systole of the enlarged right ventricle. As compensation wanes the apex beat becomes weaker and irregular. Sometimes an intermittent *systolic thrill* is felt in the 4th interspace in the left mammillary line.

The radial pulse in the early stages is comparatively unaltered.

Later it becomes frequent and irregular in volume. Appended, Fig. 43, is a sphygmogram of the pulse in advanced mitral insufficiency. It is of the type of the *pulsus parvus irregularis.*

Percussion discovers enlargement of both the relative and absolute areas of dulness, upward in the direction of the left auricle, downward to the left and also to the right, the right border of the heart reaching at times beyond the right border of the sternum.

Auscultation recognizes a systolic murmur in the mitral area, conducted with various degrees of loudness into the left axilla and under the angle of the scapula. This direction of its conduction is the distinc-

FIG. 43.—TRACING OF PULSE OF MITRAL INSUFFICIENCY.

tive feature of this murmur. It is usually soft, but occasionally rough, more rarely musical. It is also sometimes well heard to the left of the pulmonary area, and rarely over the entire præcordium. Not always loud enough to be easily heard, it may be brought out by exertion on the part of the patient.

The second sound of the heart is sharply accentuated at the pulmonary interspace until the tricuspid valve fails, when the accentuation vanishes. The aortic second sound is less strong, corresponding with the less degree of hypertrophy of the left ventricle.

MITRAL STENOSIS.

Mechanism and Occurrence.—This lesion occurs as an uncombined or simple form of valvular disease in young persons, especially women, but is very much more commonly combined with mitral insufficiency. The orifice is stenosed and the blood is restrained from passing freely into the left ventricle. The same backward effect is produced upon the left auricle, on the lungs, the right ventricle, and general venous circulation as in mitral regurgitation, but the left ventricle is not hypertrophied in simple mitral obstruction because no extra muscular demand is made on it, while hypertrophy of the left auricle is one of the most characteristic signs of mitral stenosis. Theoretically, the left ventricle should even atrophy, while the absence of the enlargement is of great diagnostic value. Excellent compensation is often maintained in mitral stenosis for many years.

Pure stenosis without regurgitation is possible if the mitral valve-leaflets are fused without retraction, so as to form the funnel-shaped opening already described. In these cases a post-mortem demonstration of insufficiency by means of the hydrostatic test is scarcely possible. More frequently the mitral orifice viewed from above is a mere slit—Corrigan's button-hole contraction, straight or slightly crescentic, in a smooth septum formed by fusion and contraction of the valve leaflets

and tendinous cords. In some cases calcareous infiltration is added and in a few rare instances uratic deposits. The ratio of button-hole mitral to the funnel-shaped orifice varies with different observers—one to ten by A. E. Sansom, one to 13 by Hayden, one to 46 by Hilton Fagge.

Most frequently mitral stenosis is the result of endocarditis, acute or chronic, but it may in rare cases be congenital. In these cases, of which a number have been collected by Bedford Fenwick, the stenosis is secondary to narrowing of the tricuspid orifice, thus explained: A small quantity only of blood being allowed to pass into the right ventricle and lungs, a diminished supply is sent to the left heart, whence both its cavities and orifices are reduced in size. *No functional disorder can cause mitral stenosis.*

Symptoms.—These, often delayed, as in mitral insufficiency by compensation, are the same as in that lesion when they occur. In consequence of this commonness of symptoms the diagnosis of mitral stenosis is based largely on the physical signs.

Physical Signs.—Inspection, consistently with what would be expected in absence of hypertrophy of the left ventricle, recognizes little or no displacement of the apex. If there is any it is due to the hypertrophy of the right ventricle, which pushes the apex toward the left rather than downward and to the left. Nor is the true apex beat increased in force, though there may be strong epigastric pulsation because of hypertrophy of the right ventricle, and in thin-chested persons there may be an impulse in the 3d and 4th interspaces to the left of the sternum while compensation is maintained. A left auricular impulse, presystolic, may be noted for the same reason as in mitral regurgitation. A jugular pulse may also be present if there is tricuspid regurgitation. The bulging præcordium occurs under the same conditions as in mitral regurgitation, but is not often seen. In children the lower sternum and 5th and 6th left costal cartilages may be prominent from this cause—enlargement of the right ventricle.

Palpation discerns that the apex beat is without undue force, but it may be diffuse, and an impulse may be felt in the epigastrium, the situation of the apex of the right ventricle. The most marked feature of palpation when present is the *presystolic thrill* at the apex, differing in this respect from the rare systolic thrill of mitral insufficiency. It is usually best felt in the 4th or 5th interspace within the nipple line. It is similar in rhythm to the presystolic murmur, but may be present without it, and is not always present. It is pathognomonic of mitral stenosis.

In moderate degrees of stenosis the pulse is not altered; in high degrees it is small, from want of blood and left ventricular power. Irregularity, like that of mitral regurgitation, is characteristic of advanced stages. Two tracings from cases of mitral stenosis are introduced in the text.

Percussion recognizes cardiac enlargement in the direction of the left auricle and right ventricle, but not of the left ventricle.

Auscultation does not discover a murmur in every case of mitral stenosis, because of the feebleness of the auricular contraction, especially toward the end of life when compensation has failed and there is not the

force of contraction sufficient to throw the blood stream into audible vibration. Most characteristic is the abruptly terminating *presystolic* murmur, confined for the most part to the mitral area to the inner side of the apex beat, though it may be conveyed upward, and it is even heard posteriorly, though rarely. It is true that the presystolic murmur is heard in atypical situations, especially in the axilla and below the angle of the scapula, more frequently than has commonly been supposed.*

The *presystolic murmur* is a diastolic murmur occurring at the end of diastole of the ventricle, because it is at this time that the auricular systole takes place, giving the propulsive force necessary to produce the audible vibration. It is a loud, rough, vibratory murmur terminating suddenly with the first sound, sharp and ringing and coincident with the presystolic thrill. The murmur terminates with the impulse, and as the two are not always easily separable, the former is commonly more readily distinguished by its qualities than by its time. It may occupy the entire period of diastole. In such cases there is sometimes a short pause between the beginning or diastolic part and the terminal or presystolic part of the murmur.

The murmur of mitral stenosis is sometimes difficult to distinguish

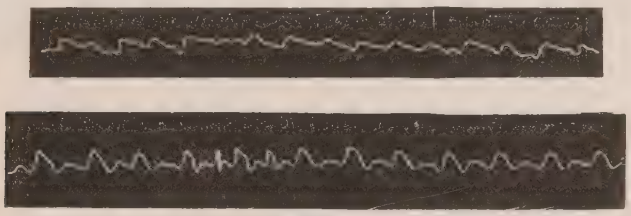


FIG. 44.—TRACINGS OF PULSE IN MITRAL STENOSIS.

from that of aortic regurgitation, but in the latter there is enormous hypertrophy of the left ventricle, which is wanting in mitral stenosis. The time of tricuspid stenosis is identical with that of aortic regurgitation, but it is heard in a different part of the præcordium,—in the epigastrium. Much more closely does the murmur of mitral stenosis resemble the so-called *Flint's murmur*, and the infrequency of the latter alone prevents more mistakes. This murmur is heard at the apex, the same site as the presystolic, and may be similar in quality. It occurs in high degrees of dilatation of the ventricle and is due to the fact, according to the late Austin Flint, Sr., that in such dilatation the mitral leaflets cannot, during diastole, be kept back against the ventricular wall, but remain in the blood current, throwing the latter into audible vibration.

Not infrequently the presystolic murmur is associated with a mitral systolic murmur, usually soft and not very loud, though sometimes it is distinct and well transmitted into the axilla. The pulse is small, as would be expected from the small volume of blood ejected from the ventricle, but

* See an excellent paper on this subject, well illustrated, by J. P. C. Griffith, in the Transactions of the Association of American Physicians for 1895.

may be quite regular, as seen in the sphygmograms. On account of these difficulties, while the presystolic murmur is a valuable sign of mitral stenosis, it should not be alone relied upon for diagnosis, but should be taken in connection with other signs. Tricuspid stenosis may be associated with mitral stenosis, or insufficiency, or both. With the loss of compensation the presystolic murmur disappears, together with the thrill, and there remains only the sharp, ringing normal first sound.

Accentuation of the second sound is marked, but confined to the pulmonary area, because there is no hypertrophy of the left ventricle. The second sound may also be duplicated, because of the want of synchronousness in the closure of the aorta and the pulmonary valves. Dr. Sansom regards this reduplication as a seeming one only of the second sound. He regards it rather as the normal second sound followed by another sound due to a sudden tension of the mitral valve itself. He also says it occurs in at least a third of all cases of mitral stenosis, and is rare in other cardiac conditions. The accentuation of the pulmonary second sound also disappears with the enfeebling of the contraction of the right ventricle.

Dr. Sansom lays great stress on the evidence of the cardiograph in the diagnosis of mitral stenosis, which enables one to judge of the relative length of systole and diastole. In stenosis the interval between the systoles may be greatly prolonged or the diastolic intervals vary greatly in duration. In mitral regurgitation, on the other hand, a short interval only separates the systoles.

Patients with mitral stenosis are subject to attacks of recurring valvulitis with consequent embolism in different parts of the body.

Mitral Insufficiency and Stenosis.

Occurrence.—More frequently stenosis is associated with insufficiency, when we have the double mitral murmur, sometimes with difficulty divisible into its two parts. Extreme irregularity of rhythm and pulse, with frequency and smallness of the latter, conspicuous thrill, marked right-sided hypertrophy, and sharply accentuated pulmonic sound are characteristic of advanced stages.

Aortic Insufficiency or Incompetency.

Mechanism and Occurrence.—This is the most serious and irremediable of the valvular diseases of the heart commonly met with. Next in frequency to mitral incompetency, more frequent than aortic stenosis, with which it often coexists, it is a disease of men rather than women, commonly adults at or before middle life. The width of the aortic orifice increases from birth to old age, while the valve cusps tend to shrivel, so that conditions favorable to incompetency coexist. It includes 30 to 50 per cent. of all cases of chronic valvular disease. It is the lesion most frequently followed by sudden death. When it exists the aortic valves are incompetent to close the aortic orifice,

either on account of the large size of the latter or disease of the valve segments themselves, and the blood flows backward into the left ventricle during diastole. The ventricle, seeking to restore the balance, redoubles its energy, hypertrophies. The blood is thus driven into the aorta with great force, swelling the arteries to extreme fulness, which, however, falls away promptly, because of the backward flow into the ventricle at the same time with the forward movement into arteries and capillaries. This sudden falling away of the pulse, from extreme distention to collapse, is very characteristic of this form of valvular disease, and is called the "trip-hammer" or "water-hammer pulse," also Corrigan pulse. It may even be visible to the casual observer in the exposed arteries, such as the carotid, temporal, and radial, while the aortic beat, ordinarily beyond reach in the supra-sternal notch, may be felt in this situation.

The abrupt jerking impulse with sudden recoil is easily recognized by the finger on the pulse, which, however, fails to find the pulse as strong and hard as would be expected from its appearance. On the

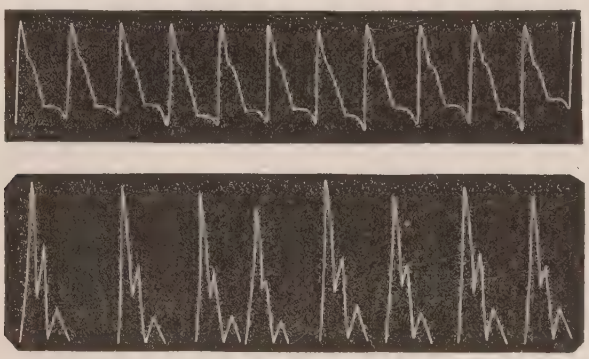


FIG. 45.—TRACINGS OF PULSE OF AORTIC REGURGITATION.

other hand, it is soft and receding. A tracing of this pulse is seen in Fig. 45. It is the typical *pulsus celer et altus*. Sclerotic changes in the arterial walls are not uncommon in aortic incompetency.

The product of this defect is the largest heart met in morbid anatomy, the left auricle and right ventricle often sharing in the enlargement. From its size the heart is often called the *bovine heart*. It often weighs 35 ounces (1050 gm.), and has attained a weight of 50 ounces (1500 gm.) or more. The cavities are enlarged and the walls thickened, so that it furnishes an instance of eccentric hypertrophy. There may be ultimate dilatation of the arch of the aorta from the constant pounding of the blood against it in systole, while the carotids may be seen throbbing in the throat. Degeneration of distal blood-vessels also sooner or later occurs.

The tremendous systole of the ventricle may ultimately force the mitral valve to yield. Compensation is still maintained for a time by hypertrophy of the left auricle, which also after a while yields, becoming

dilated and allowing the blood to engorge the lung. Hypertrophy of the right ventricle comes then for a time to the rescue. Sooner or later it, too, yields, dilates, the tricuspid valve weakens, and finally gives way, allowing the blood to flow back into the venous side of the circulation, producing engorgement of the liver, stomach, kidneys, general dropsy—the train of symptoms described under mitral regurgitation. Where aortic insufficiency is primary it is less apt to be followed by stenosis. On the other hand, where stenosis is primary there is very apt to be insufficiency as well. Causes of insufficiency in addition to those considered under the general etiology of valvular disease are congenital malformations, including fusion of two leaflets, commonly those behind which the coronary arteries are given off. Such fused leaflets are especially prone to valvulitis and its consequences. Aortic insufficiency is quite often caused by dilatation and aneurism of the ascending aorta, causing *relative* insufficiency.

Symptoms.—Like all other forms of valvular heart disease, aortic incompetency may be compensated for a long time. Indeed, full compensation is said by some to be most common in this form of valvular disease. Both *dropsy* and *dyspnœa* are characteristically *absent* until compensation ceases, which is never until the mitral valve begins to yield. Then, however, both appear and may be very distressing. An especially frequent symptom is *dizziness* with *faintness*, particularly on rising quickly. *Palpitation* ensues on slight exertion, and this effect is in marked contrast to the comfort of the patient when quiet, when the pulse may be slow and breathing quiet. The patient is very apt to be *troubled in his sleep* and to dream, probably because of disturbed circulation in the brain. Even permanent mental symptoms may result from this cause, including *insanity* and *suicidal tendency*. Lesser degrees are *irritability* and *peevishness*, though these are not confined to this form of heart disease. Præcordial *pain*, present also in stenosis, is frequent in this form of valvular disease. It may be a dull ache with a sense of constriction of the chest or sharp and radiating down the arms, particularly the left, as in angina pectoris, which condition itself is also common. With the yielding of the mitral valve and loss of compensation come the symptoms of mitral disease already described.

As already stated, this is the form of valvular disease in which sudden death is most frequent. It has overtaken many a victim in the course of his daily vocation and without warning, though it is most apt to be invited by some slight over-exertion or mental excitement. The cause of such sudden death is probably interruption of the circulation in the coronary arteries. This may be brought about in one of two ways. These arteries in common with others are especially disposed to endarteritis and resulting sclerosis and atheroma, a condition which constantly invites thrombosis and obstruction to the circulation. Or it may be due to embarrassed circulation in these vessels caused by the aortic regurgitation. For even if the blood enters the coronary arteries during systole it must still receive in health some further supply in the recoil of the blood on the closed semilunar valves, which cannot take place when the valves are incompetent. On this variety, too, supervenes not infre-

quently the cardiac form of acute infectious endocarditis of the grave type with the train of symptoms and the sequelæ described. Embolism in various organs is also a complication independent of the acute involvement.

Physical Signs.—Inspection often discerns the left præcordium prominent, with the apex beat lowered and to the left, and the visible pulsation far beyond the normal situation of the apex, all confirmed by *palpation.* Palpation also recognizes at times a diastolic thrill over the base, in the carotids and subclavians, and sometimes in the aorta at the suprasternal notch. This is, however, much rarer in aortic regurgitation than the systolic thrill in stenosis. The Corrigan pulse may also be felt, but is much more strikingly manifested in the sphygmogram. A *capillary pulse* is also sometimes demonstrable in the skin and mucous membrane. This may be brought out by drawing a pencil lightly across the skin of the cheek or forehead; and on the mucous membrane of the everted lower lip by pressing a glass microscope slide against it, as suggested by F. C. Shattuck. Pulsation in the retinal arteries may be recognized by the ophthalmoscope.

Percussion discloses increased dulness to the left and downward, and also, sometimes, in advanced cases, upward to the left of the sternum, owing to hypertrophy of the left auricle, as well as to enlargement of the ventricle upward.

Auscultation recognizes a diastolic murmur, long, loud, and blowing in quality, usually harsher than the aortic obstructive murmur. Its area of maximum intensity is commonly to the left of the second interspace near the midsternum, sometimes as low as the fourth left costal cartilage, and even at the ensiform cartilage. Hence it may be mistaken for the mitral obstructive murmur and for the murmur of tricuspid disease, but each of these, be it remembered, is unaccompanied by hypertrophy of the left ventricle. The murmur is naturally transmitted downward toward the ensiform cartilage or along the left edge of the sternum and toward the apex in the direction of the regurgitating column, but it is not conducted in the direction of the great vessels of the neck, at least with any loudness. In this condition also occurs the Flint murmur, described under mitral stenosis.

Auscultation of the vessels furnishes interesting results in aortic insufficiency. The second arterial sound, well known to be the aortic second sound, is absent in aortic incompetency, since the valve remains open. The aortic diastolic murmur is sometimes faintly transmitted into the carotid, while a short, rough, systolic murmur is sometimes heard in the same vessel. A valvular sound may also be heard in the arteries of medium size, such as the femoral, the brachial, and often the radial, the ulnar, and even the palmar arch and dorsalis pedis, by pressing lightly with the stethoscope, rendered more intense by strong pressure. Then there is Traube's double sound, in the femoral and popliteal quite often heard. The sounds are such that the two sounds follow each other shortly, so that the first seems preparatory to the second, or they are separated by a longer interval, like the two sounds of the heart. The explanation of these sounds is not undisputed. The simple femoral

sound is ascribed to a sudden tension of the vessel wall, and Traube explained the first of his sounds in the same way, while he explained the second by sudden relaxation of this tension. Friedreich pointed out that a similar double sound could be heard in the femoral *vein* in tricuspid insufficiency, which he ascribed to tension of the valves of the vein. It is claimed that the double sound is heard in other diseases of the heart, especially mitral stenosis, and even in aneurism, but it is acknowledged that it is most frequent in aortic incompetency. Finally is Duroziez's double *murmur* in the femoral, which, though rare, is sometimes heard in aortic insufficiency. It consists of two murmurs plainly separated, produced by pressing with the stethoscope. Of these murmurs the first comes with the passage of the systolic blood wave, and the second from the passage of the abnormal backward wave from the periphery through the compressed vessel.

Aortic Stenosis.

Mechanism and Occurrence.—By aortic stenosis is meant a narrowing of the aortic orifice. This is usually due to fusion and stiffening of the aortic leaflets in a position of partial opening. The further changes of thickening, calcification, and atheroma described under morbid anatomy may be present in various degrees. Stenosis is said to be relative when there is a normal orifice and the aorta is dilated beyond it.

This form of valvular disease, though much less common than insufficiency of the aortic valves, is not rare. It occurs in older persons, and the older the person the more likely are there to be calcareous deposits causing it. It may be congenital. When uncombined with insufficiency it is the least dangerous of the various forms of valvular disease. The narrowed orifice prevents the free discharge of blood from the left ventricle into the aorta. The ventricle attempts to overcome this, and its walls hypertrophy in proportion to the degree of resistance, and often for a long time compensate for the obstruction—until dilatation occurs, when the danger really begins. The hypertrophy thus induced, usually of the simple form, is only second to that of incompetency.

Symptoms.—The symptoms of aortic stenosis may be long delayed, so long as compensation is maintained, and when they do occur they are usually those of a deficient supply of blood to the brain and heart itself, viz., *dizziness* and *fainting*. Succeeding exertion there is apt to be a *sense of constriction* or *oppression* and even *pain* in the præcordium, which may develop into the symptoms of a true angina pectoris.

Physical Signs.—*Inspection* and *palpation* recognize usually a forcible apex beat beyond the normal situation and at varying distances in accordance with the degree of hypertrophy, while palpation adds often a thrill of great intensity with each beat of the heart when dilated or eccentric hypertrophy is established. A bulging of the præcordium may also be present, though less often than in incompetency.

The pulse is the *pulsus tardus*, slow in reaching its maximum volume, which is small. It is frequent but regular, contrasting in the latter respect with the pulse of mitral disease. Fig. 46 is a sphygmogram.

Percussion elicits dulness downward and laterally toward the left, since, as a rule, the enlargement is confined to the left ventricle. There may, however, be slight enlargement upward to the left of the sternum if hypertrophy of the left auricle is added.

Auscultation discloses a systolic basic murmur, loudest at the aortic area—2d interspace at the right of the sternum—conducted distinctly into the carotids, and even sometimes along the course of the aorta, behind and to the left of the vertebral column, into the popliteals and dorsal arteries of the feet. It is not, however, confined to the aortic area, but may be heard over the entire præcordium. It is usually rough, but may be soft and musical. It is made louder by exercise. The aortic factor of the second sound is very feeble or not at all heard if the constriction be at all decided, because of the feeble recoil, the result of the small amount of blood in the aorta. The first sound is normal and somewhat louder and more prolonged than natural, because of the powerful contraction of the left ventricle.

Roughness of the *aorta, dilatation,* and *narrowing* of the vessel, however caused, may also produce a systolic murmur; and so may roughness within the ventricle in the course of the outgoing column of blood. But these causes have a less positive effect upon the substance of the

FIG. 46.—PULSE TRACING OF AORTIC STENOSIS.

heart, that is, do not produce as marked hypertrophy of the left ventricle. Nor do they interfere with the production of a normal second sound, except perhaps dilatation, which in that event is accompanied by an aortic regurgitant murmur. From this it follows that the important point to remember in diagnosis is that an aortic systolic murmur by no means always indicates aortic stenosis. So, also, anæmic or hæmic murmurs, which are always systolic and for the most part basic, may simulate aortic systolic murmurs, but these occur in young, delicate persons, of both sexes, and are often intermittent and without other effect on the muscular heart, while they are also unaccompanied by thrill. There may be roughness, too, in the pulmonary artery which can be localized to the left of the sternum.

Stenosis of the aortic orifice is very apt to be associated with insufficiency, the same rigidity and adhesion which prevents complete patulousness of the orifice preventing also complete closure.

AORTIC STENOSIS AND REGURGITATION.

Occurrence.—This double lesion is a comparatively frequent one, indeed, commonly regarded as the next in frequency after mitral insufficiency, and therefore more frequent than either aortic insufficiency or aortic stenosis alone. It occasions a double basic murmur, systolic

and diastolic, and is also a grave condition, giving rise to the same dangers as aortic regurgitation and the same enormous hypertrophy of the left ventricle.

Diagnosis.—The diagnosis of this condition requires special mention because it not infrequently happens that it is mistaken for aneurism of the arch of the aorta, which is associated with a similar double murmur of which the systolic element is due to the roughness of the aorta and aneurismal walls, and of which the diastolic is a sign of relative insufficiency due to dilatation of the aorta. The distinctive differences between the two conditions will be given in treating of aneurism of the arch of the aorta.

Tricuspid Regurgitation.

Occurrence and Mechanism.—Tricuspid regurgitation as a primary condition is extremely rare, and when present is probably the result of an endocarditis during fœtal life, endocarditis at this period being more prone to attack the right than the left side. Endocarditis involving the tricuspid valve may, however, occur in children also, according to Byrom Bramwell * more commonly than has been supposed. In primary tricuspid regurgitation hypertrophy of the right ventricle occurs just as that of the left follows mitral insufficiency. Infectious or mycotic endocarditis also affects the tricuspid valve, according to Osler, in 19 out of 238 cases. More frequently tricuspid regurgitation is the result of a relative insufficiency, one of the terminal events of mitral disease, the tricuspid orifice yielding with the dilatation of the right ventricle, which takes place sooner or later, consequent upon the resistance to the movement of the blood through the engorged lungs. It is also one of the possible sequelæ of emphysema of the lungs and long-standing fibroid phthisis, succeeding also a primary hypertrophy of the right ventricle, due to these causes. Its effects, depending upon the venous obstruction, have already been detailed, p. 525.

Physical Signs.—In primary tricuspid disease with regurgitation, *inspection*, and *palpation* note an apex beat diffused toward the ensiform cartilage and the epigastrium. *Percussion* detects enlargement toward the right edge of the sternum.

To *auscultation* the systolic murmur thus engendered is invariably feeble and is heard almost solely in the tricuspid area, just above and to the left of the ensiform cartilage. Occasionally only is the second pulmonic sound accentuated. There should be no confounding of this murmur with that of aortic regurgitation conducted toward the same situation nor with that of mitral regurgitation heard at no great distance, for the reasons already given. To these must be added a difference in quality and pitch between the tricuspid and the mitral murmur not always, however, manifest.

The jugular pulse is also more or less constantly associated with tricuspid regurgitation. It is often more forcible in the right than left

* Amer. Jour. Med. Sci., April, 1886, p. 419.

jugular. The presence of the pulsating liver is also characteristic and regarded as pathognomonic.

The jugular pulse is systolic in time and does not appear until the valves situated at the opening of the internal jugulars into the innominate veins yield. These give way first on the right side because the course of the right innominate is straighter and communication is more direct. So long as the valve above the bulbus jugularis is closed, the pulse is confined to the bulb, but with the yielding of this valve the pulse becomes general throughout the vein. It is sometimes not easy to distinguish a true jugular pulse from the "physiological" or "false" jugular pulse, which may sometimes be seen in health and whenever the venous system is over-full. Pressure on the vein above the valves will cause the false pulse to disappear while the true pulse, coming from the right ventricle, will remain. The physiological pulse alternates with the ventricular systolic and corresponds with the auricular systole, while the true jugular coincides with the systole of the ventricles.

Tricuspid Stenosis.

Occurrence.—Tricuspid stenosis is a more rare condition, but it may be an acquired condition in association with left-sided heart disease as the result of rheumatic endocarditis and unknown causes. It is much more frequent in women, fully 80 per cent. of all cases being in women. As in endocarditis of the left side, there is thickening, adhesion, narrowing.

Physical Signs.—The diagnosis in such cases is seldom made, because the murmur is masked by the coincident mitral systolic murmur. In a very few cases only is it confined to this valve. In such an event a presystolic murmur and thrill may be found in the tricuspid area at the right edge of the sternum from the 4th costal cartilage to the 6th cartilage at the junction of the sternum. Frequently there is no murmur. Percussion shows dulness to the right of the sternum.

A presystolic tricuspid murmur pointing to such a condition in a case observed by Gardner was found due to a growth from the endocardium of the right auricle, so placed as to fall over the tricuspid orifice in the manner of a ball valve. Fred. C. Shattuck has met one instance of tricuspid stenosis with mitral stenosis and regurgitation, along with adherent pericardium, hepatic cirrhosis, and slightly granular kidney, as determined by autopsy. In this case there was a presystolic tricuspid murmur observed for three years before death.

Congenital stenosis of the tricuspid orifice occurs, but is usually associated with defects of other valves which early cause death.

Other symptoms are cyanosis of the face and lips and in the later stages extreme and obstinate dropsy.

Pulmonary Stenosis.

Occurrence.—The great majority of systolic murmurs heard at the pulmonary orifice are functional. Pulmonary stenosis, though very rare, may, however, exist, in which case it is far more likely to be congenital from arrested development, although intra-uterine endocarditis may also

cause it. So, also, may infectious endocarditis, and in rare instances atheroma. The valve-leaflets are apt to be fused. When the lesion is congenital, it is commonly associated with patency of the foramen of Botal or foramen ovale, together with imperfect ventricular septum and tricuspid stenosis.

Physical Signs.—Pulmonary stenosis should furnish a systolic murmur in the pulmonary area, to the left of the sternum. The murmur may even be heard behind, between the shoulders, and it may be rough. It is accompanied by hypertrophy of the right ventricle. There may be a basic thrill, as in aortic obstruction, but the pulse is uninfluenced. Compensation may be set up by means of the patulous foramen ovale, an open ductus arteriosus, or interventricular communication. The invariable presence of cyanosis due to venous obstruction and of attacks of dyspnœa complete the picture and aid greatly in the diagnosis. Anæmic murmurs at the same time and place are unaccompanied by cyanosis.

Walshe has described a case of death from thrombosis of the pulmonary artery in which he heard a pulmonary systolic murmur before the end came.

Pulmonary Regurgitation.

Occurrence.—Simple pulmonary regurgitation is scarcely known. It may, however, exist as a congenital defect (fusion of two segments), and the pulmonary valve has been found involved in ulcerative valvulitis.

Physical Signs.—It is easy from what has gone before to deduce the physical signs which are to be expected—a diastolic murmur heard in the pulmonic area, difficult, however, to distinguish from an aortic diastolic murmur, hypertrophy of the right ventricle, later jugular pulse, venous congestion, and cyanosis. A few cases are related in which a diastolic murmur has been found associated with defects in the pulmonary valves, in one warty, which might have been the result of infectious endocarditis. All others are congenital. Among them is aneurismal dilatation. Such was a case reported to the Pathological Society of Philadelphia by Edward T. Bruen. (See Transactions for 1883.)

Congenital Defects.

Congenital defects in the cardiac valves and orifices deserve a passing notice. They may be the result of endocarditis during fœtal life or of arrest of development. Their most frequent seat is the right heart and the most frequent form is stenosis of the pulmonary orifice, the effects and signs of which have already been considered. Another is a permanently patulous foramen ovale; or there may be a defect of the septum of the ventricles, or a communication between the aorta and pulmonary artery—a persistent ductus arteriosus—or between the aorta and the vena cava or aorta and right auricle. All of these intercommunications produce murmurs difficult to separate, and it is, after all, by attention to the general condition that the defect is recognized. The patient, a child of arrested development, more or less permanently cyanosed, with continued embarrassed breathing, all of these are conditions which point to the congenital defect. If there be added to these a persistent loud murmur at

the base of the heart without other signs or symptoms of valvular disease, this may be due to congenital defect.

In addition to these, there are a large number of defects of development which are rather pathological curiosities than of clinical interest. Among these may be mentioned *acardia*, or absence of heart, met in the monstrosity thus named; *double heart*, sometimes present in high degrees of fœtal defect; *dextro-cordia*, in which the heart is on the right side, alone or with other viscera. In *ectopia-cordis*, or dislocation, which is associated with fission of the chest-wall and of the abdomen, the heart may be in the cervical, pectoral, or abdominal regions. Then there are anomalies of the cardiac septa, of which the patulous foramen ovale is the most frequent, various in degree. Next is a small defect in the upper part of the septum, between the ventricles, in what is known as the "undefended" space, or just anterior to it. A *bi*cuspid state of the semilunar valves, from fusion of cusps, is often met, most frequently of the aorta. The combined valve is more liable to sclerotic change. Finally, there is fenestration of the semilunar cusps.

Relative Frequency of Valvular Defects.

The order of frequency of the various valvular defects is not entirely agreed upon. As to one, however, there seems to be universal concurrence, and that is that mitral regurgitation is the most frequent. After this, however, statistics differ. Thus of the older authors W. H. Walshe presents the following order of frequency for the single or simple murmurs:—

(1) Mitral incompetency.
(2) Aortic stenosis.
(3) Aortic incompetency.
(4) Mitral stenosis.
(5) Tricuspid incompetency.
(6) Pulmonary stenosis.
(7) Tricuspid stenosis.
(8) Pulmonary incompetency.

This, in the light of modern studies, has to be corrected except as to mitral incompetency. Frederick J. Smith, analyzing the registers and post-mortem records of the London hospitals for eleven years—1877–1887—and taking the fatal cases only, arrived at the following order:—

(1) Mitral incompetency.
(2) Mitral stenosis. } Of practical equal frequency.
(3) Aortic incompetency. }
(4) Aortic stenosis.
(5) Tricuspid stenosis.

Out of the 705 cases Smith found 26, or 3.38 per cent., of mitral stenosis, and 25, or 3.25 per cent., of aortic regurgitation. So it cannot be said there is any practical difference in the relative frequency of these two lesions. Von Leube says of aortic incompetency that after mitral incompetency it is the most frequent, and this is my experience. Smith's

statistics, being recent and based, as they are, upon the examination of registers and autopsy records, are probably nearly correct, but these two lesions approximate in frequency.

Of "double" murmurs heard at one orifice those of mitral stenosis and insufficiency are more numerous than aortic stenosis and insufficiency, women being the most frequent subjects. It should be mentioned, however, that George S. Middleton, in a clinical study of 150 cases of chronic valvular disease, at the out-patient department of the Royal Glasgow Infirmary, found a much larger number, 22 per cent., of the double aortic lesions as against Dr. Smith's four per cent.; also that Walshe makes the double aortic lesion the second in frequency of all valvular diseases.

Associated or Combined Valvular Lesions.

These terms are applied when two valves are diseased at the same time, a very frequent occurrence.* The valves which are most frequently jointly affected are, of course, the aortic and mitral, then mitral and tricuspid, then aortic, mitral, and tricuspid. Aortic disease of either kind is more frequently associated with mitral incompetency than mitral stenosis, because the former is sooner or later a result of the aortic disease, while mitral stenosis arises by a separate process. The very careful analysis by Frederick J. Smith referred to furnishes the following order, which is not far from correct:—

(1) Aortic incompetency and stenosis; mitral incompetency.

(2) Aortic stenosis and mitral incompetency.

(3) Aortic incompetency and mitral incompetency.

There is less than one per cent. difference in the frequency of (2) and (3).

(4) Aortic incompetency and stenosis; mitral stenosis and incompetency.

(5) Mitral incompetency and tricuspid incompetency.

(6) Aortic incompetency and stenosis; mitral incompetency, tricuspid incompetency. In children, it is said the most frequent combination is aortic incompetency and mitral incompetency.

The diagnosis of these combinations is based upon the quality and situation of the murmurs and their conduction.

Prognosis of Chronic Valvular Disease.—Possible positive statements as to the prognosis in chronic valvular disease are few, so uncertain is it, and so many circumstances influence it. Undoubtedly valvular disease often exists where the subject is totally free from symptoms and therefore quite unconscious of it. Yet such subject is not free from danger. On the other hand, fifteen, twenty, thirty, and even forty years pass over such cases without inconvenience, compensation being easily maintained. Such cases are usually of mitral incompetency or stenosis or both. Much depends upon the life led by the patient, whether one of

* Dr. Sansom has suggested that the term "combined" be retained for two murmurs at one orifice, commonly known as "double" murmurs,—an unfortunate suggestion, as it will be sure to give rise to confusion, while the term double is easily understood.

ease and quietude associated with proper food and clothing without dissipation. Even where such disease occasions symptoms the same measures may hold them in abeyance for a long time, and occasional judicious medication may raise the patient from a serious condition to one of comfort. It is astonishing with what little disturbance women with these affections sometimes bear children. Of the lesions at the mitral orifice incompetency is usually most easily compensated, then combined stenosis and incompetency, and finally stenosis only; but even the latter exists at times without subjective symptoms in persons who have worked hard. After all, the prospect of life must be judged from the symptoms in each case. The compensation which is obtained by extreme hypertrophy and apex displacement is tottering. An added danger in mitral disease, especially mitral stenosis, is that of embolism. Recurring attacks of rheumatism not only increase the latter danger but augment the valvular defect. The supervention of dropsy and dyspnœa indicate failing compensation, and though they may be overcome it is with increasing difficulty at each recurrence.

Aortic incompetency is a much graver condition. It is this valvular disease in which sudden death overtakes the patient. Yet it, too, may be compensated for years. Much here depends upon the state of the arteries, the dangers being increased when associated with sclerosis or atheroma. For these conditions are likely to affect the root of the aorta and the valves and especially the coronary arteries. Any obstruction in these, as already stated, may be the cause of sudden death. Angina pectoris indicates a diseased condition of the coronary arteries which may at any time be followed by complete obstruction and sudden death. Overdistention, such as takes place during exertion, may be too much for a fatty heart already dilated and become also a cause of sudden death.

The most unfavorable of all forms of cardiac valvular disease is tricuspid regurgitation, which occasions obstinate dropsy and dyspnœa.

Chronic valvular disease is regarded as much more serious in young children, say under ten years of age. This in spite of the fact that many conditions favorable to compensation are present, such as integrity of heart muscle and vascular supply. Notwithstanding this, the valve lesion is apt to grow. On the other hand, there is a popular notion, which physicians are disposed to encourage, that a child may outgrow a heart disease under favorable circumstances, such as abundance of good food and protection against exposure and overwork. This may be true, but it is more likely that compensation is established with the growth and development of the organ. That the apparent event does sometimes occur I can attest. Congenital defects in the heart are apt to destroy the lives of children the first few years of their existence.

Finally, almost any serious illness, especially when involving the lung, increases the danger to the life of the subject of cardiac disease, while mitral disease, and especially mitral stenosis, invite pulmonary congestion and inflammations.

Treatment of Chronic Valvular Disease of the Heart.

Since there are certain points in the treatment of disease of the cardiac valves which are the same for the different orifices, I shall consider first such measures as are thus common, referring more especially to mitral and aortic disease.

In the first place, it is well known that there exist chronic valvular defects at either of these orifices which give rise to no symptoms whatever and are often accidentally discovered. From the standpoint generally conceded, that such defects themselves are irremediable, it is clear that, in the absence of symptoms, medicinal treatment is quite unnecessary. On the other hand, it is a happy circumstance when the subjects of such lesions are made aware of their presence, because they are enabled so to regulate their mode of life as to prevent harmful consequences, either symptomatically or organically. Such persons should avoid over-exercise and excitement. Running, or even walking rapidly, hurriedly ascending stairs, extremes of passion of all kinds, and especially of anger, should be avoided, as should also exposure and irregular living. In a higher grade of involvement of either orifice the same treatment is demanded in a more imperative manner, since its omission results in a loss of compensation, manifested by dyspnœa, palpitation, and præcordial distress.

In a still more advanced degree of interference with normal function, the treatment becomes different with the seat of the lesion. Let us first consider lesions of the mitral valve, and first the most common of all forms, —*mitral regurgitation.* We have seen that the blood flows back into the left auricle during systole of the ventricle, at a time when all communication between these cavities should be cut off and the movement of the blood should be forward only. Averted for a time by hypertrophy of the left auricle, engorgement of the lungs ultimately results, with defective aëration of blood and consequent shortness of breath. This effect is at first counteracted by the increased effort of the right ventricle, whence its hypertrophy with sharp accentuation of the pulmonary second sound.

So long as compensation is thus maintained there is probably no sign of embarrassed breathing or irregularity or præcordial oppression or digestive derangement ; but as soon as this begins to fail, through a suspension of the conditions which coöperate to help it, or a slight yielding of the heart-muscle to the resistance to be overcome, assistance is demanded. The heart-tonics, of which digitalis is the type, are the agents preëminent for this purpose. That they operate by directly increasing the force of the right ventricle and left auricle, and thus contribute to the compensation, can scarcely be doubted ; but that they help also to make the closure of the mitral orifice more complete by forcibly increasing the contraction of the left ventricle seems also reasonably sure, since the experiments of Ludwig and Hesse have made it so plain how this can occur. They have shown that the mechanism for closing the mitral orifice does not reside in the valve alone, but that the surrounding muscles of the ventricle have an active share, not only in floating up the valve curtains, but in reducing also the size of the opening which these valve curtains

have to close. This is, of course, less applicable in chronic valvular conditions, where there is stiffness from calcareous change, than where regurgitation results from simple feebleness of muscle in anæmia and after the infectious fevers.

The effect required of this class of drugs varies with the degree of obstruction to be overcome, and the doses vary accordingly. Very often the heart requires but little steadying to enable it to accomplish the desired end, and moderate doses—such as five minims (0.3 c. c.) of the tincture of digitalis once in eight hours—suffice. On the other hand, it is a mistake to give too small a dose, and too great timidity often results in failure. Doses of ten to 15 minims (0.6 to one c. c.) of the tincture of digitalis every three hours, and corresponding doses of the other preparations, are often necessary and sometimes produce magical effects. The irregular and halting pulse becomes regular, the dropped beat is again taken up, the dusky lips become pink, the scanty urine is increased, the shortness of breath disappears, and calmness and quiet succeed distress and restlessness. As soon, however, as the desired effect is produced the dose should be lowered. The same principles apply to the management of the still more serious engorgements of the venous system which succeed upon tricuspid insufficiency, and produce dropsies and serous effusions. It is in the effort to drive the blood through the engorged veins that the left ventricle finally hypertrophies in this form of disease. Its failure to accomplish this through its inherent power demands the same remedies which the right ventricle needed at an earlier stage to overcome the lung engorgement, and here it is that large doses of cardiac stimulants are demanded.

This engorgement is also relieved by the use of purgatives, and as the portal area, including the liver itself and the stomach, is especially involved, mercurial purgatives are especially indicated. Five to ten grains (0.32 to 0.65 gm.) of blue mass at bedtime, followed by a saline in the morning, relieve the congestion, and with it the nausea and indisposition to take food which attend it. Such remedies may be resorted to semi-occasionally. Sometimes the continued use of small doses for a long time—say ½ to one grain (0.03 to 0.065 gm.) of blue mass three times a day—are more efficient. The old-fashioned combination of calomel, squills, and digitalis—in doses of ½ grain (0.03 gm.) of the first and one grain (0.065 gm.) of the second and third three times a day, or smaller doses more frequently repeated—is sometimes most happy in its effect. Digitalis is a remedy always better intermitted to obtain its best effects, and a remedy, too, which, having once excited nausea, is thereafter badly borne. It does, however, sometimes happen that digitalis may be given continuously in moderate doses—say five minims (0.3 c. c.) three times a day—with great advantage, while its omission is followed by signs of failing compensation. It is generally recognized that digitalis produces also contraction of the arterioles, and that through this, in connection with the forcible systole, the arterial pressure is increased. This effect is desirable and useful in the early stages of mitral regurgitation, before tricuspid regurgitation and dropsy have set in. Later in the disease, however, when dropsy has set in, this effect militates against the

diuretic action which is so much needed. How this may be overcome will be mentioned later.

As to the relative value of the different preparations of digitalis. While testimony is generally favorable to the infusion as the most efficient remedy, yet, on account of convenience and accessibility, the tincture is mostly used. I am inclined to believe that the greater apparent efficiency of the infusion is partly due to the fact that it is generally given in larger doses. Thus, a tablespoonful, or ½ ounce (15 c. c.), is not an infrequent dose of the infusion, while ten minims (0.6 c. c.), or 20 drops, of the tincture and one grain (0.064 c. c.) of the powder are not often exceeded. When it is remembered that ½ ounce (15 c. c.) of the infusion, as made by the U. S. Pharmacopœia, represents nearly three grains (0.19 gm.) of the powder, or 20 minims (1.2 c. c.) of the tincture, one may understand why it is more efficient. It is true, however, that the infusion is sometimes better borne by the stomach than equivalent doses of the tincture. It may be that the cinnamon-water with which it is made has this effect.

Of remedies which may be substituted for digitalis, strophanthus should be first mentioned; not that it is always the best. It was at one time thought that strophanthus might have all the effects of digitalis on the left ventricle without the contracting effect on the arterioles. This expectation was not, however, realized by clinicians, but it is a fair substitute for digitalis in about the same dose. It is better borne by the stomach, as a rule, than digitalis.

Caffeine is an admirable heart-tonic in mitral regurgitation. I do not give less than three grains (0.2 gm.) at a dose, but seldom give more, every three hours. When caffeine has been given in full doses for some time it produces mental symptoms quite characteristic, consisting in hallucinations not unlike those of delirium tremens, the patient imagining there are persons, animals, and other objects about him, and he is sometimes difficult to control. They, however, cease immediately when the drug is discontinued. Another effect of caffeine, which sometimes interferes with its usefulness, is that of insomnia.

Sparteine sulphate is another heart-tonic which I have come to value very highly, especially where a diuretic effect is desired. The dose should never be less than ¼ grain (0.016 gm.), increased to ½ grain (0.032 gm.), four, five, and six times a day. I am sure many have been disappointed in sparteine because they have given too small a dose.

In the much rarer disease of simple *mitral stenosis*, compensation is even easier and longer maintained by nature's own resources than in mitral regurgitation. Here, for evident reasons, there is no tendency to dilatation or hypertrophy of the left ventricle. On the other hand, hypertrophy of the left auricle becomes a conspicuous condition, succeeded by hypertrophy of the right ventricle, for the same reason as in mitral regurgitation. Especially easy is it to maintain compensation if the narrowing is not too great and if there is a well-preserved left auricle and a strong right ventricle. If, however, the mitral narrowing is extreme, it is plain that the pulmonary engorgement will become greater if we increase the force of the right ventricle. Much more cautious must we be, therefore,

in the use of digitalis. Much more needed under these circumstances is relief to the pulmonary congestion, which in turn will relieve the right heart-tension. For the same purpose aconite is sometimes of advantage in these cases in small doses, say one minim or 1½ minims (0.06 to 0.09 c.c.) every two hours or every hour, watching its effect. It is possible that it is through a somewhat similar action that convallaria majalis —a remedy in which most observers have been disappointed—has been found useful by Dr. Sansom* in mitral stenosis and also by the French school. By these observers it has been found diuretic, increasing the twenty-four hours' urine to 85 and even 115 ounces (2550 to 3450 c. c.), reducing the pulse-rate, regulating irregularity, and improving the breathing, even when accompanied by tricuspid regurgitation. The doses given are ten to 20 minims (0.6 to 1.2 c.c.) of the tincture three times a day, and it may with advantage be associated with caffeine. The French physicians give the extract in doses of one gram to 1½ grams a day, *i. e.*, 15 to 21 grains. More effectual than either of these remedies to relieve pulmonary congestion is purging, sometimes bloodletting, and repeated small bleedings are often of great advantage in this form of chronic valvular disease.

The principles governing the treatment of combined mitral regurgitation and stenosis are rather those of mitral regurgitation than of mitral stenosis.

And what shall be the treatment of *pure aortic disease?* It will be remembered that both aortic obstruction and regurgitation give rise to hypertrophy of the left ventricle, and that this is compensatory in purpose, for a time quite sufficient to ward off any unpleasant symptoms, and for a still longer time competent to do this when associated with a quiet life, the absence of excitement, of exposure, and of privation. Its well-marked degrees are accompanied with a powerful systolic impulse, a symptom which is of itself at times a source of great discomfort. Shall we, then, give heart-tonics, which increase the force of this thumping blow? Certainly not. Shall we give aconite or veratrum viride, which slow the heart and diminish the force of its stroke? Yes, at times these remedies are very useful. Whenever, as the result of over-exertion or undue excitement or gastric derangement, the heart is turbulently overactive, and even irregular in its rhythm, then often I have seen aconite in small doses—say one minim (0.06 c. c.), or two drops, repeated every half-hour or hour under close observation—act happily, especially when combined with bromide of potassium, say 15 grains (one gm.). The tincture of veratrum viride may be given in slightly larger doses. As soon, however, as this period is past, the aconite should be omitted. Even in mitral regurgitation I have seen aconite act most happily under these circumstances.

We want rather in this condition to find remedies which will help to maintain the integrity of the heart-muscle. Such are strychnine, iron in small doses, arsenic, and nutritious, easily assimilable food. Especially

* "The Treatment of Some of the Forms of Valvular Disease of the Heart" (Lettsomian Lectures. 2d ed., with corrections, London, 1886).

useful are well-ventilated living- and sleeping-rooms, wholesome out-door life, with moderate, deliberate muscular exercise. On the other hand, the mountain-climbing advocated by Oertel seems irrational and dangerous. Every one who has had experience knows that the high altitudes are not well borne by cardiac cases of any kind.

Such measures as these tend to ward off the next stage, for sooner or later the integrity of the muscle of the ventricle yields, dilatation is added to hypertrophy, the auriculo-ventricular orifice enlarges, and we have mitral regurgitation. Then the treatment becomes that for mitral disease.

The treatment of aortic regurgitation and of aortic stenosis with regurgitation is similar to that of aortic stenosis.

Treatment of Dyspnœa.—As the dyspnœa is primarily the result of deficient blood-aëration in the congested lungs, the same remedies which force the blood through these organs, and thus relieve the congestion, tend also to relieve the dyspnœa, and often do so. When the dyspnœa persists, it is frequently caused by effusions into the pleural cavity, which are most promptly and successfully removed by tapping, although a blister may also answer the purpose. Repeated tapping may be necessary. Dyspnœa not thus relieved demands an opiate, and of opiates, under these circumstances, morphine is the best. One-quarter grain at bedtime, by the mouth or hypodermically, gives unspeakable comfort. Hoffmann's anodyne will sometimes relieve the milder degrees, given in fluid-drachm doses (3.5 c. c.), and should perhaps be tried first, as it is always desirable to put off the use of morphine as long as possible. Paraldehyde may be substituted for Hoffmann's anodyne in the same doses. Chloralamide is even a better remedy in 30-grain (two gm.) doses. Sulphonal may be tried in full doses of 15 to 30 grains (one to two gm.). Trional in the same doses is a similar drug. None of them are anodynes. They are simple hypnotics and cannot be expected to take the place of morphine, though they may be tried at first. All the coal-tar products are more soluble in hot liquids, of which milk is a typical form.

Treatment of Dropsy.—In like manner the measures that relieve the congestion and dyspnœa tend also to relieve the dropsy, but special means are also necessary. Here it is that full doses of digitalis are especially indicated, and at close intervals, every three hours and even every two hours. But these measures are often insufficient. They may be materially aided by restricting the ingestion of liquids. With the tissues waterlogged and secretion insufficient, it is plain that copious liquid ingestion only increases the difficulty. I am speaking now of cases where there is general dropsy which resists the ordinary treatment.

The principle of the Matthew Hay method is correct, but in practice it is limited, because, with an already congested stomach, solids cannot be digested without an admixture of liquid, and further embarrassment results from the effort to dissolve them and from the presence of undigested residue. Therefore it is better to omit solid food altogether and reduce the liquid to a minimum that will sustain life,—not more than two ounces every two hours, and that only during the waking hours. To this may be added the use of purgatives. While diuretics sometimes fail

us, we can always secure an effect from purgatives. A daily morning dose of Epsom salts or Rochelle salts is given. Then, when action of the bowels begins, full doses of digitalis, or caffeine, or sparteine, associated with nitro-glycerin, are almost sure to be followed by copious diuresis, and when diuresis starts up in these cases it is astonishing what quantities of urine are passed. The association of nitro-glycerin with digitalis at this stage is important. A $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.00065 to 0.0013 gm.) may be given as often as the digitalis and simultaneously. One need not be afraid of this drug. I have given this dose every two hours for twenty-four hours or more at a time. Elimination by the bowels and kidneys being simultaneously stimulated, the sucking up of the interstitial fluid is greatly favored and often rapidly brought about. If these measures be associated with chest-tapping, which may be required, the diuresis set up is often enormous while the swelling rapidly declines. As diuresis is established or hunger sets in the quantity of milk allowed may be increased, and when the dropsy has entirely disappeared a cautious return to solid food may be permitted.

For *irregularity* of heart-action and *palpitation*, which are more common in mitral disease, belladonna is also a useful remedy. It may be combined with digitalis. A good belladonna plaster placed over the palpitating heart is one of the most efficient agents in subduing it. Nitro-glycerin is often very useful to the same end. One $\frac{1}{100}$ grain (0.00065 gm.), rapidly increased to $\frac{1}{50}$ grain (0.0013 gm.), three times a day or oftener, is the proper dose. It may also be combined with digitalis, as above directed. Cardiac pain is also sometimes relieved by the same remedy.

DISEASES OF THE MYOCARDIUM.

The heart is subject to alterations in its muscular substance independent of valvular defect. Simple hypertrophy, dilatation, fatty infiltration, and fatty metamorphosis, or true fatty degeneration, are the most important. Myositis, abscess, and aneurism of the walls of the heart are such rare conditions that they need only to be mentioned in passing, especially as there is no way to recognize them before death.

HYPERTROPHY AND DILATATION.—ATROPHY.

Definition.—Some vagueness exists in the application of these terms. The word *enlargement* may be applied with its literal meaning to any increase of size, whether hypertrophy or dilatation. *Hypertrophy* should be limited to indicate such enlargement as is associated with increased thickness of the walls. Such increased thickness may be associated with a cavity of normal size, when it is known as *simple hypertrophy*. More frequently the cavity is also enlarged, producing *eccentric* hypertrophy, also known as hypertrophy with dilatation. The term concentric hypertrophy

was applied to a condition in which thickening is associated with reduction in the size of the cavity, but it is now conceded that such condition is a post-mortem product and does not exist ante-mortem. Even simple hypertrophy is more infrequent in the post-mortem room than is supposed. Where the left or right ventricle alone is affected the hypertrophy may be simple or eccentric, where there is general hypertrophy it is always eccentric. All true hypertrophies are numerical, that is, there is an actual increase in the number of muscular fasciculi due partly to a fission of previously existing fibres and partly to a new formation of fibres.

The word *dilatation* is applied to conditions in which the cavities are enlarged with or without attenuation of the walls. The former is the typical condition. The latter is rare—indeed, its existence is denied by some. Dilatation implies fatty degeneration, for it is through intermediate degeneration that the muscular fasciculi waste and ultimately disappear, producing thinning.

Hypertrophy more frequently affects the left ventricle, dilatation the left auricle and right ventricle, but the whole heart may be involved. As a matter of course, the left ventricle is most frequently affected by hypertrophy.

Hypertrophy of the Heart.

Etiology.—Hypertrophy implies an overgrowth of muscular tissue and is naturally the result of extra work, increased effort to overcome increased resistance, whatever its cause. The term *idiopathic* hypertrophy is applied to such hypertrophy associated with no abnormality in the valves. Hypertrophy without valvular disease involves the left ventricle more frequently, but may involve both cavities. The resistance needed to excite it may be from within or from without, or due to nervous influence. Resistance from within is occasioned by obstruction to the outflow of blood or to increased intravascular pressure.

Such obstruction is offered in the case of the *left ventricle* by aortic stenosis, congenital narrowing, by aortic insufficiency, and mitral insufficiency. Increased intravascular pressure is caused by endarteritis and resulting sclerotic changes in the vessel walls and by aneurism; by contraction stimulated by the irritation of toxic substances in the blood, such as accumulate in Bright's disease, or as the result of over-eating or drinking, especially of large quantities of beer; finally by excessive physical exertion. All these are, therefore, causes of hypertrophy of the left ventricle.

External obstruction to the contraction of the left ventricle is found in pericardial adhesions and myocarditis. Such hypertrophy is always eccentric. Hypertrophy of the left ventricle from nervous influence is seen in exophthalmic goitre and allied conditions and in long-continued palpitation. Constant mental excitement is a possible cause.

In the case of the *right ventricle*, internal resistance is produced by pulmonary congestion due to mitral regurgitation or mitral stenosis, to narrowing of the same vessel or branches, such as occurs in pulmonary emphysema. Valvular lesions of the right side of the heart produce hypertrophy of the right ventricle, just as those of the left cause it to

hypertrophy. The greater infrequency of these lesions and their frequent development *in utero* are to be remembered. Pericardial adhesions also constitute a cause of external resistance of contraction of the right ventricle.

Auricular hypertrophy is always eccentric; that is, while the walls are thickened the cavities are also dilated. Hypertrophy of the left auricle is usually caused by stenosis of the mitral orifice, and to a less degree is a result also of regurgitation of the blood in incompetency of the mitral valve. Hypertrophy of the right auricle might also be expected as a consequence of regurgitation of blood from the right ventricle to the right auricle, but the resistance to the further backward flow into the veins is so much less than on the left side of the heart that hypertrophy is correspondingly less. In like manner, even if stenosis of the tricuspid orifice is present, the same conditions prevent any marked degree of hypertrophy of the right auricle.

Morbid Anatomy.—The hypertrophied heart is altered in its weight, dimensions, and shape. The adult heart weighs in health, in the male fifty to sixty years old, about 335 grams (11.8 ounces); in the female, 295 grams (10.44 ounces). The average thickness of the wall of the left ventricle in health is $\frac{5}{8}$ to $\frac{2}{3}$ inch (1.6 to 1.7 cm.); of the right ventricle, $\frac{1}{6}$ to $\frac{1}{4}$ inch (0.4 to 0.6 cm.); of the left auricle, $\frac{1}{8}$ inch (three mm.); the right auricle, $\frac{1}{12}$ inch (two mm.).

Hearts, therefore, exceeding these weights and measurements are hypertrophied. Measurements should be made before rigor mortis sets in or after it has passed away. The latter may be favored by soaking the heart in water. Commonly the hypertrophied heart does not exceed 25 ounces (750 gm.), though hearts weighing 48 and 53 ounces (1440 to 1590 gm.) have been noted.

The shape of the heart varies. In left ventricular hypertrophy it is elongated to the left and lies more horizontally, while the conical shape is less marked; when both ventricles are hypertrophied the heart is round. In mitral stenosis with hypertrophy of the left auricle and right ventricle it is also quadrate, the right ventricle occupying the chief bulk of the organ, while the left ventricle recedes behind it.

Symptoms.—Hypertrophy, being a process of compensation, is not at first attended by any symptoms. It is the result of a generous conservative effort of nature by means of which symptoms are averted. But unlike the hypertrophy of the muscles of the blacksmith's arm, it tends ultimately to degeneration, and thus becomes the initial link in a chain of evil which is well stated by J. G. Adami:* "In the first place, it leads to an increased nutrition of the walls of the arteries; increased nutrition leads to increased connective-tissue growth of the walls; increased fibrous tissue of the walls leads to contraction and increased rigidity of those walls; the increased rigidity leads to increased resistance to the passage of the blood current. The increased resistance requires increased propulsive power on the part of the ventricular muscle, that is to say, increased work; the increased work of the heart leads to overgrowth and

* "Notes upon Cardiac Hypertrophy," Montreal Medical Journal, May, 1895.

hypertrophy (myocarditis), and with this heightened blood pressure and further increased nutrition of the walls. And now, at last, the stage is reached, this vicious circle continuing, in which either the (vessel) walls give way or the heart." From this standpoint, increased blood pressure alone is sufficient to explain the anatomical changes, *i. e.*, the arterial sclerosis, atheroma, and fibroid thickening, so constantly seen in valves and heart walls without calling in chronic inflammation or specific agency. Certain it is that the two conditions react on each other, and it is more than likely that the former (increased blood pressure) may produce the latter (chronic inflammation) *de novo*, and many otherwise unexplained facts are rendered clear.

When reactive effect sets in we begin to have symptoms which are at first intermittent, brought about only by some temporary cause which excites the heart, such as exercise, mental emotion, fatigue, mental or physical, tobacco or alcohol. They are a feeling of vague discomfort about the heart amounting seldom to pain, sometimes increased when the patient lies on the left side, palpitation, a consciousness of the beating of the arteries in the head, dizziness, headache, ringing in the ears, flushes or flashes of light, tendency to hemorrhage of the nose.

Physical Signs.—While symptoms other than physical signs may be wanting, the latter are present from the beginning, increasing with the duration of the hypertrophy.

In hypertrophy of the left ventricle inspection and palpation furnish much the same information as in hypertrophy of the left ventricle from valvular disease, an apex lower and to the left, strong and diffuse. The radial pulse is strong and tense, the carotids pulsate visibly, and the ausculted vessel sounds are loud and distinct. Percussion shows enlargement to the left and downward. To auscultation there is no murmur, but a distinctive intensification of the aortic second sound is noted, sometimes ringing, quite characteristic, and itself of great diagnostic value. It may be reduplicated. The first sound, while louder, is also duller, more prolonged and diffuse, clicking in quality, sometimes suggesting a systolic murmur not present. It may also be reduplicated.

In hypertrophy of the right ventricle the signs of enlargement are toward the right edge of the sternum and beyond, also without murmur, but with sharp accentuation of the second sound in the pulmonary area to the left of the sternum. The apex beat is also displaced outward but not downward, pulsation may be distinct in the lower sternal or epigastric region or between the ensiform cartilage and the 7th rib. In thin-walled persons there may be an impulse in the 3d and 4th right interspaces. The pulse at the wrist is small unless there be associated hypertrophy of the left ventricle.

Diagnosis.—In view of the fact that hypertrophy is a part of the morbid anatomy of chronic valvular defect we need concern ourselves only with the so-called idiopathic hypertrophy. The resemblance to hypertrophy may arise from pericardial effusion, circumscribed pleuritic effusion, aneurism, or mediastinal tumor in the neighborhood of a normal heart, the latter especially if it push the heart forward. A normal heart may appear enlarged to percussion when it is uncovered by a lung

retracted from any cause, as cirrhosis of that organ. Simple palpitation of the heart may be mistaken for hypertrophy.

In all cases the situation of the apex beat is a valuable criterion because, although its position may be changed in pleuritic effusion and pericardial effusion, it is in the opposite direction from that in hypertrophy, while the impulse is feeble instead of being strong. Aneurism and mediastinal tumor will certainly furnish some of the signs peculiar to them and thus permit a distinction. Simple palpitation is without the percussion signs of enlargement of the heart. Under none of the circumstances named will its situation be altered.

Hypertrophy may be obscured if the heart is overlapped by an emphysematous lung. This is partly the case in hypertrophy of the right ventricle, which is often associated with emphysema of the lungs. In such cases the pulse does not help, but rather helps to mislead, because it is small in hypertrophy of the right ventricle.

Prognosis.—When associated with valvular disease the prognosis is that of the disease itself, against which it is for a time a protection, counterbalancing the growing defect of the valve until the nutrition of the heart begins to be impaired and dilatation replaces hypertrophy with loss of compensation. The latter may be sudden, though it may be delayed for a time by treatment. While such malnutrition may be of local origin, that is, resident in the heart muscle itself, it may be due to general causes also, as general illness, hardship and exposure, over-exertion, fatiguing occupation, insufficient food, and the like. When it is the result of endarteritis and aneurism, the termination comes with the apopletic rupture of the vessel or of the aneurism.

In the so-called idiopathic forms due to increased intravascular pressure excited by toxic substances in the blood, it is not until the sclerotic changes established in the blood-vessels which react upon the heart produce the last links in the vicious train described that danger begins to threaten.

Treatment.—This embraces that of the causal condition.

Dilatation of the Heart.

Synonym.—*Fatty Degeneration of the Heart.*

Definition.—This has already been defined on page 546, so far as the state of the chambers is concerned, but a dilated heart is also a fatty heart in the sense that the muscular fasciculi are in a state of fatty metamorphosis.

Etiology.—Dilatation of the heart demands separate consideration from the etiological rather than the anatomical standpoint. It occurs as the last stage in a valvular disease as the result of failing nutrition, or it may occur acutely from sudden over-distention of the cavities. The latter is possible as a rare event with a heart previously normal. More frequently the two causes act jointly. The conditions under which the former manifests itself have been described. This joint action occurs when a hypertrophied heart associated with valvular disease becomes over-distended. Moderate degrees of distention occur with

any decided muscular effort. The more marked degrees capable of mischievous consequences are the result of prolonged severe muscular exertion. The effect of moderate, well-regulated exercise on the heart, known as training, by which endurance is developed, is to produce eccentric hypertrophy, or hypertrophy with dilatation, which is not dilatation in the sense under consideration—enlargement of the cavity with thinning of the walls. The right heart is the seat of such dilatation. In overexertion the harmful effect of excessive acute strain is averted for a time by the safety-valve action of the tricuspid valve. While moderate degrees of dilatation may be recovered from, either rapidly or slowly, dilatation may be carried to degrees at which recovery is impossible and death results. Such results have followed rowing and mountain-climbing.

The so-called *irritable heart*, to which attention was first called by J. M. Da Costa in a graphic description based on the cases of soldiers in the American War of the Rebellion, is an example of dilated heart. Another example of the idiopathic hypertrophy already referred to is seen in those persons who through hard work acquire muscular strength and at the same time through alcoholic indulgence become obese. Such are the drivers of beer wagons and workers in breweries where an unlimited amount of beer is allowed, as much as 20 liters (as many quarts) a day. In a few instances in malignant forms of the infectious diseases, such as scarlet fever and diphtheria, the nutrition of the heart may be so rapidly impaired by the toxic agency which causes the disease that dilatation occurs with very little or no undue intravascular pressure.

Symptoms.—The symptoms of "heart strain" are sudden *pain* in the region of the heart or epigastrium, *shortness of breath*, and *rapid, feeble action of the heart*. If it be not immediately fatal the symptoms may pass off, but are renewed on the slightest exertion. In the acute cases described as due to the toxic causes of infectious disease, sudden death may be the only symptom. In some cases it may be preceded or not by very brief præcordial distress. Less serious degrees may be associated with *faintness* or *palpitation* on exertion, *extreme feebleness* of the heart's action, and *dyspnœa*. It is rather characteristic for these symptoms to pass away when the patient is at rest, to be renewed on the slightest exertion.

Symptoms growing out of dilatation of the heart, going also to make up the sum of those constituting chronic valvular disease with failure of compensation, are *general venous congestion, dropsy, feeble*, frequent, and *irregular radial pulse*, rarely, on the other hand, a *slow pulse*. The former is probably due to defective pneumogastric inhibition the result of anæmia of the brain, the latter to scanty nutrition and a loss of irritability of heart muscle. To anæmia especially affecting the medulla oblongata may be ascribed, too, Cheyne-Stokes breathing, also a symptom of the terminal stage of the disease. To it may be ascribed symptoms simulating apoplexy, which characterize the slower dying in some of these cases. Palpitation, angina pectoris, and dyspnœa—cardiac asthma, with syncopal attacks, coldness, and slow pulse, 30 to 40—are all symptoms more or less associated with dilatation of the heart. It is fur-

ther characteristic of these symptoms of dilatation that they are not transient or amenable to treatment by the usual heart tonics, of which digitalis is the type.

In some instances, especially in the dilated heart of pernicious anæmia, there may be a full, strong, and regular pulse.

Physical Signs.—When the termination is not immediate, physical signs may be recognized. To *inspection* the impulse is diffused over a wide area, but is feeble and fluttering, a point of greatest intensity or an apex beat being often wanting. If the right heart is chiefly involved, the beat, as far as caused by the left apex, is completely wanting, while an impulse may be felt below or to the right of the ensiform cartilage, as well as a wavy impulse in the 4th, 5th, and 6th interspaces to the left of the sternum. A pulsation may be seen in the 2d left interspace, which, while sometimes presystolic, is commonly systolic. In the latter event it may be a further expansion of an already dilated auricle by blood regurgitating during systole of the left ventricle; or if presystolic it may be the pulse of auricular systole. Such at least are possible explanations. The fact that at autopsies, even in extreme dilatation, the left auricle is found so far back from the thoracic wall as to be scarcely able to beat against the 2d interspace does not preclude the possibility of this during life. In dilatation of the *right* auricle, on the other hand, there is sometimes seen an impulse in the 3d interspace on the right side which is clearly systolic and due to regurgitation from the right ventricle during its systole.

To *percussion* there should be increased dulness to the right and downward toward the epigastrium or left beyond the normal line, though this may be obscured by an emphysematous lung. The results of *auscultation* are greatly influenced by complications. If cardiac murmurs are present they may obscure all else. On the other hand, previous murmurs may disappear. The typical sounds are found in the dilatation following idiopathic hypertrophy. The impulse is feebly heard as well as felt; the first sound is feeble but pure, that is, shorter and more like the second, lacking as it does the muscular element. It may be scarcely audible even in the absence of murmurs. It is sometimes reduplicated because of asynchrony in the action in the two halves of the heart. Sometimes there is a loud systolic murmur at the apex due to relative insufficiency of the mitral valve, the true nature of which becomes apparent only in the event of its disappearance. The second pulmonic sound may remain sharp if there be dilatation only of the left ventricle and there is compensatory hypertrophy of the right, feeble if the right ventricle is involved. Finally there is intermittent and irregular action, at times the characteristic gallop rhythm, which is almost pathognomonic of dilatation. The pulse is very frequent and feeble.

Diagnosis.—An acknowledged difficult matter at times is the distinction of pericarditis with effusion from the dilated heart. As to whether inspection furnishes any information depends mainly upon the stoutness or leanness of the patient. In the stout person nothing is recognizable in either condition. In the thin-chested the impulse is visible and wave-like in dilatation; it is not visible, or barely so, in pericardial

effusion. The same is true of palpation except that if the patient leans forward the impulse may be felt in pericarditis.

Percussion affords the most valuable information. If it brings out the well-known triangular shape of dulness with the apex toward the inner end of the left clavicle and the base in the 5th or 6th interspace, especially in the absence of a cardiac impulse, there must be pericardial effusion. To auscultation, while the heart sounds have lost their characteristic sharpness they still contrast with the distant and muffled sounds in pericardial effusion. Especially if there is left any of the original hypertrophy the second sound will retain some of its sharpness, while if there happens to have been valvular disease the murmurs remain to help us.

The encroachment on the lung is very much less in dilatation than in pericarditis with effusion, and while there is shortness of breath in both it is less pronounced in dilatation and more influenced by exertion, being less while the patient is quiet. This encroachment in the case of dilatation does not give rise to Skodaic resonance in the axilla.

Prognosis.—This is ultimately fatal; in fact, the stage of dilatation is the stage in which remedies become unavailing.

Treatment.—This is essentially that of valvular heart disease. Rest is even more important, while the heart tonics are of course indicated. Strychnine is an important remedy and in dangerous stages may be given hypodermically in $\frac{1}{30}$ grain (0.0022 gm.) doses every three to four hours or oftener for a short time. Digitalis or digitalin, $\frac{1}{60}$ grain (0.0011 gm.), may be given under like circumstances. Suitable, nutritious food and, if the patient survives the primary danger, well-regulated exercise. A timely blood-letting may save life if the signs of engorgement of the right ventricle are present—intense dyspnœa, lividity.

Atrophy of the Heart.

Definition.—Atrophy of the heart is the opposite state to hypertrophy, viz., a reduction in the muscular substance with a corresponding reduction in the size of the cavities. It is limited clinically, being confined to the subjects of wasting diseases, like phthisis pulmonalis and carcinoma. Under these circumstances the muscular fasciculi undergo molecular death, the organ wastes and is symmetrically reduced.

The **diagnosis** of such condition can only be based on a diminution in the normal area of cardiac percussion dulness associated with feeble pulse and the long-continued presence of the causal disease. The **treatment** is that of the causal disease.

The term atrophy is also applied to certain conditions, the result of impaired nutrition, which are better regarded as degenerations. Such are *brown* atrophy and *yellow* atrophy, to be again referred to on p. 555.

DEGENERATIONS OF THE CARDIAC MUSCLE.

The heart muscle is subject to parenchymatous degeneration, to fatty degeneration, to fatty infiltration, to amyloid degeneration, to the hyaline transformation of Zenker, to calcareous degeneration, and to the changes known as brown atrophy and yellow atrophy.

Parenchymatous or Albuminoid Degeneration (*Cloudy Swelling*).—This is a change in which the sarcous substance is converted into granular matter of albuminoid composition which produces also more or less indistinctness in the striated appearance of the fasciculi. The albuminoid composition of the product is attested by its solubility in acetic acid and its insolubility in ether. The general effect is one of softening and flaccidity.

It is ascribed to some toxic agency and occurs most frequently in the infectious fevers—typhoid fever, typhus fever, scarlet fever, diphtheria, and the like. It was at one time considered a consequence of high temperature, but this view is no longer held. It is believed also to be at times, at least, the first stage of fatty degeneration or to precede fatty degeneration. It is certainly at times associated with it. Cloudy swelling may disappear and the muscle resume its natural histology.

Fatty Degeneration or Fatty Metamorphosis.—In this change the sarcous substance of the fasciculi is directly converted into globular fat, as contrasted with the condition of fatty infiltration, where the fat is deposited between the fasciculi. The little fat drops—and they are very minute as a rule—are seen in rows parallel to the fibrillæ of the fasciculus and all transverse striation has disappeared. As intimated, the cause of such degeneration is an interference with the proper nutrition of the heart muscle.

It may be *general*, when it has its most frequent expression in the dilated heart which succeeds upon hypertrophy, involving the walls of one or more cavities. It is also a result of the impaired nutrition of old age, of the grave infectious diseases, and of cachectic states generally, such, for example, as pernicious anæmia. In the last two it may be associated with parenchymatous degeneration or succeed upon it. It is also a result of the action of certain poisons, as phosphorus and arsenic, the effects of which extend also to other muscular organs. Under these circumstances the heart is generally enlarged (dilated), flabby, and relaxed, of a light yellow or yellowish-brown color, and very friable, permitting the finger to be easily poked through it. The papillary muscles and the trabeculæ in the left ventricle may be the seat of circumscribed fatty degeneration and dotted and streaked with yellow, fatty matter. Unlike parenchymatous degeneration, fatty degeneration, when once established, is considered irremediable.

Fatty degeneration of the heart may also be *circumscribed*, in small foci variously distributed. Thus it may be confined to the superficial or sub-pericardial layers, when it is especially the result of pericarditis. Or

there may be numerous pin-head foci in the subendocardial layer in cases of extreme dilatation.

Finally there may be a single focus in the substance of the left ventricle or the septum due to total obstruction of one of the branches of the coronary artery, usually the anterior, by a thrombus or embolus. The product is an area of fatty degeneration known also as *anæmic necrosis* or *white infarct*. In the early stage the infarction is brownish-yellow or hemorrhagic. Minutely examined the muscular fasciculi exhibit a loss of nuclei, and later they break up into a cheesy detritus. The infarct is not always thus made of fatty debris, but may present a hyaline appearance. It may be the seat of rupture and thus cause hemorrhage into the pericardium and immediate death.

Diagnosis.—The diagnosis of fatty degeneration so far as recognizable is that of dilatation, slight degrees and circumscribed fatty degeneration being unrecognizable, while considerable areas of partial degeneration may also exist without symptoms. In fact, the presence of some dilatation of the cardiac cavities seems to be necessary to the production of symptoms—the feeble pulse, palpitation, and dyspnœa being symptoms of the dilatation rather than the fatty degeneration.

Prognosis.—This is grave. It is impossible to restore the degenerated muscular substance to its natural structure. With degeneration established, death is liable to occur suddenly, and remedies which avail with an integral organ are useless here.

Treatment.—This embraces that of cardiac dilatation. Acute attacks should be met by stimulants, of which alcohol, aromatic spirit of ammonia, and digitalis are the type. Strychnine is also indicated and may be used hypodermically.

Fatty Infiltration, or Fatty Overgrowth.—Strictly speaking, this condition is not a degeneration of the heart muscle, though it leads ultimately to fatty metamorphosis. It is the *cor adiposum* of the older authors, and differs from fatty metamorphosis in that the fat is infiltrated *between* the muscular fasciculi. In the true cor adiposum the fat extends deep into the substance of the muscle, sometimes as far as the endocardium. It covers also the outside of the heart, at times so completely that the true muscular structure is invisible. This infiltration sooner or later interferes with the proper nutrition of the muscular substance, a true fatty degeneration results with its symptoms, so far as any are manifested, and becomes ultimately also a cause of death.

The fattily infiltrated heart is commonly a part of general obesity, and occurs therefore at a time of life when this is usual, that is, between the ages of forty and seventy years, and is more than twice as frequent in men as in women.

The condition is inferred from the presence of extreme obesity associated with signs of cardiac weakness.

The **treatment** is that of obesity.

Amyloid Degeneration invades the heart as it does other organs, attacking the blood-vessels and intermuscular connective tissue. Zenker's

hyaline transformation attacks, on the other hand, the muscular fasciculi, causing them to appear swollen and transparent and the striæ to be indistinct or absent.

Calcareous Degeneration is a rare condition in which the fasciculi are infiltrated with lime salts.

Brown Atrophy is a pigmented state of the heart muscle, associated with a reduction in volume of the heart substance. It is the special result of senile marasmus, but it is also found in wasting from other causes, especially phthisis and cancer. It is also found in the heart of chronic valvular disease. The general effect is to produce a heart of a dark red-brown color, firmer in consistence than the normal heart. Microscopic examination recognizes a peculiar arrangement of pigment granules about the nuclei and between the primitive fibrillæ. The source of the pigment is not precisely known. It may be the coloring-matter of the muscle or directly derived from the blood.

Yellow Atrophy is only another name for advanced fatty metamorphosis.

MYOCARDITIS.

Fibro-myocarditis.

Synonyms.—*Fibroid Degeneration; Fibroid Heart; Fibrous Myocarditis; Indurated Degeneration; Myodegeneration; Sclerosis of the Coronary Arteries.*

Definition.—Not really an inflammation of the cardiac muscle, but a fibroid change substituting the normal muscular substances in places.

Morbid Anatomy.—In pure, uncomplicated cases the valves are normal, while the muscle on examination is found dotted with white, shining areas variously numerous. Minutely examined these are found made up of pure or part fibroid tissue, the muscular fasciculi being correspondingly destroyed. They are seated for the most part in the left ventricle toward the apex and in the anterior wall, though they may be found elsewhere also. They may often be seen from the endocardial or pericardial surface as cicatricial-like depressions. Sometimes there is a single large patch known as a fibroid patch. The papillary muscles may exhibit the same fibroid change. The condition is, strictly speaking, not inflammatory, the patches representing transformed areas of anæmic necrosis due to obstructive disease of the coronary arteries and branches, lesser degrees of the more complete obstruction causing the white infarct described. The disease in the coronary arteries, which is of an arteriosclerotic nature, is the result of endarteritis. It not only brings about a diminished blood-supply but it causes degeneration of the muscular fasciculi and their conversion into fibrous tissue. Only in the event of such diminished supply do the changes occur. Hence it is that arteriosclerosis of the coronary arteries is not always followed by fibroid change.

The fibroid change may also be associated with valvular disease, the arterio-sclerosis remaining still the cause, or it may be due to an invasion of the muscular tissue by the endarteritis. Or the endocarditis may give rise to embolism of the coronary arteries or branches, thus cutting off the blood supply. From the cardiac thrombosis which sometimes results there may arise cerebral, renal, and pulmonary embolism.

A further result of the fibroid change is dilatation of a part or of the whole of one of the heart cavities, producing in the former instance sometimes what is known as *cardiac aneurism.* The condition may also be associated with hypertrophy without valvular disease, though the recognition of such combination before death must be a matter of inference based on the presence of arterio-sclerosis elsewhere, together with the causes of such hypertrophy, rather than on demonstration.

The causes of arterio-sclerosis of the coronary arteries, which in its turn gives rise to the fibroid change, are those of endarteritis elsewhere. They include the same causes as those which produce idiopathic hypertrophy (p. 549), but in their absence causes are often sought in vain. Certainly the tendency to arterio-sclerosis is often a hereditary one. It is a disease also which seldom occurs prior to middle life. More frequently its results are seen even later, since it is in a degree evolutional.

Symptoms.—Slight degrees occasion no symptoms while autopsies even disclose advanced stages of indurative myocarditis which were not suspected. In consequence of the frequent association, too, of endocarditis and pericarditis the symptoms of these diseases are often combined and mask the distinctive symptoms of the fibroid change. Unmasked, the symptoms are, in a word, those of dilatation of the heart, including *dyspnœa, palpitation, small, frequent,* and *irregular pulse, præcordial oppression* or *attacks of faintness,* and, finally, *venous stasis* with *cyanosis, œdema,* and *congestion of the liver, stomach,* and *kidneys, feeble digestion,* and *scanty urine.* These symptoms may set in gradually or suddenly. On such a heart digitalis and other heart tonics are without effect. A *persistently slow pulse* should be mentioned as an occasional symptom. *Angina pectoris* is also a symptom of indurative myocarditis, though it also occurs in other cardiac diseases, especially aortic stenosis. It will be described when treating of neuroses of the heart.

Physical Signs.—Physical examination recognizes a feeble impulse and on percussion enlargement of the heart—dilatation. The first sound lacks its muscular element and is more like the second, more purely valvular and therefore short. Both maintain for a time considerable distinctness but ultimately grow feeble. Occasionally there may be a mitral murmur which may be functional and transitory or permanent. These murmurs are explained by the experiments of Ludwig and Hesse, already alluded to, and more recently confirmed by Krehl. These go to show that a certain integrity of the muscles about the mitral orifice or of the papillary muscles is necessary to a complete closure of the latter. Such integrity is impaired by myocarditis, and the resulting murmurs increase the difficulty of diagnosis. There is, however, usually absence of accentuation of the pulmonic second sound characteristic of mitral regurgitation, though this may also be relatively present if the right ventricle hap-

pens to be less severely involved than the left. The second sound is also sometimes reduplicated. The mitral murmur in the fibroid heart is more variable, more subject to intermissions, than that of mitral regurgitation. The sudden addition of a mitral systolic murmur in a fibroid heart previously without a murmur may also indicate a lacerated valve.

Diagnosis.—This is often difficult, requiring the opportunity of prolonged study of the case for an accurate diagnosis. For the most part we are compelled to rely on the absence of the symptoms and signs of valvular disease and the presence of the symptoms of dilatation, the evidences of arterio-sclerosis elsewhere, a persistently slow pulse, angina pectoris, the history of syphilis and of other causes, together with the age of the patient. Where the fibroid condition is associated with murmurs the diagnosis is still more difficult and must, indeed, be a matter of probability if even suggested, so much more likely are the signs to be interpreted as those of valvular disease.

Prognosis.—This is grave, or, to say the least, uncertain. Associated as it is with sclerosis and narrowing of the coronary arteries or branches, complete obstruction is liable to occur at any time, producing sudden death. On the other hand, the patient may live for many years with the heart the seat of considerable fibroid change.

Treatment.—This must mainly consist in treating the causes and in a proper hygienic management. Habits of over-eating and drinking should be overcome. The avoidance of over-exertion associated with just sufficient exercise to develop the heart healthfully should be observed. Out-door life, a proper hygiene of the skin by bathing and manipulation, are important.

Drugs which will remove the diseased condition of the coronary arteries and fibroid overgrowth probably do not exist. Still, the reputation of iodide of potassium as a remover of fibroid overgrowth and for the cure of syphilitic disease should be availed of. For the symptoms of stasis and heart weakness, of dyspnœa and of angina pectoris, the treatment is the same as that for these conditions under other circumstances. (See treatment of valvular disease and angina pectoris.)

Acute Suppurative Myocarditis.

Synonym.—*Abscess of the Heart.*

This is a rare condition. It is said to occur as the result of acute myocarditis, but it is most frequently metastatic or pyæmic in origin, in association with puerperal fever, malignant endocarditis, or other septic processes. It may occur in the septum as well as the outer ventricular walls.

As such it is not recognizable before death and is commonly discovered at autopsies. It may, however, rupture into the heart cavities or pericardium, and in the latter situation cause early fatal termination.

RUPTURE OF THE HEART.

Rupture of the normally integral heart muscle does not occur. It is only when weakened by disease that such an event is possible. Fatty metamorphosis furnishes the most frequent predisposing condition. The softening due to obstruction of a branch of the coronary artery, as already described, is the most frequent cause of heart rupture, but the fibroid change, abscess, or ulceration are all conditions which at times precede rupture. Morbid growths in the heart wall, such as gummy tumor and carcinoma, are also possible causes.

These preliminary conditions presupposed, any unusual strain is sufficient to produce rupture, though this is not always necessary, especially in the case of the white infarct where the degeneration is so great as to admit rupture with the ordinary pressure. It is naturally an event of the second half century of life.

The anterior portion of the left ventricular wall near the septum is the favorite seat. Rupture is rarely recognized before death, which usually follows in the course of a few hours. The symptoms are præcordial pain, a sense of oppression, dyspnœa, pulselessness, and collapse. There may be enlargement of the cardiac area of dulness owing to filling up of the pericardial sac.

ANEURISM OF THE HEART.

This is a term given to two conditions :—

(1) A saccular projection from the ventricular surface of a sigmoid or cuspid leaflet, where the valve is weakened by ulceration through one of the lamella, the intravascular or intracardiac pressure furnishing the distending force. It is much more common in the aortic segments. The saccule may ultimately perforate, causing laceration of the valve.

(2) Projection outward of a circumscribed portion of the muscular wall which has been weakened by the fibroid patch or an injury to the wall. Here naturally the left ventricle, too, suffers, and near the apex in more than half the cases. The resulting pullulation varies in size from $\frac{2}{5}$ inch (one cm.) or less to dimensions equal to those of the heart itself. The aneurism may be sacculated or partitioned and even multiple.

There are no symptoms by which the condition may be recognized with any degree of probability. It may also terminate fatally by rupture into the pericardium.

NEUROSES OF THE HEART.

NERVOUS PALPITATION.

Definition.—By this is meant an unnaturally frequent, regular, or irregular beating of the heart, of which the patient is uncomfortably conscious, but which is unattended by any physical evidence of organic disease of the organ. This does not mean that there may not be functional or accidental murmurs, because these are especially prone to be present in the different varieties of anæmia which are commonly associated with palpitation. Such murmurs are always, however, systolic, a diastolic murmur always indicating organic disease. Palpitation or uncomfortable heart-beating also occurs in connection with organic disease of the heart, but this is not nervous palpitation.

Etiology.—There are numerous causes of palpitation. In the first place, it is much more frequent in women than in men. Again, it is prone to occur at the time of puberty in girls, at the menstrual period, and at the climacteric in women. Anæmia is at once a predisposing and an exciting cause; indigestion is a very frequent causal agent. Mental emotion, including fright, anxiety, and grief, diseases of the uterus and stomach, the exhaustion of protracted illness, sexual excesses, over-work, the abuse of alcohol, tobacco, tea, and coffee, are all active etiological elements. The "irritable heart" described by Da Costa, based on observations made on soldiers in the late "War of the Rebellion" in America, has for its most striking symptom palpitation. Over-work and excitement were its chief causes abetted by exhaustion from illness.

Symptoms.—The "*beating*" referred to is, of course, the chief symptom. It varies, however, greatly in degree and duration. At times a mere fluttering lasting for a few minutes, the pulse rate may reach 160 or more and be scarcely countable. When the characters last described are attained the term *tachycardia* is applied. The rapid heart action is sometimes associated with a sense of *weakness* or "*goneness*" in the epigastrium, and sometimes with *nausea*. The face is usually pale but is sometimes flushed. The physical signs usually add nothing to the undue beating noted on auscultation, though, as already mentioned, there may be functional murmurs systolic in time at the base of the heart, more rarely at the apex. The normal heart sounds may be somewhat sharper and clearer or they may be more blurred.

Diagnosis.—The only two conditions with which nervous palpitation may be confounded are myocarditis and fatty degeneration of the heart, whose symptoms, it will be remembered, are similar. The nervous affection is, however, a less serious affection, characterized by intermissions during which the heart is quiet. Its subjects are also of the anæmic nervous type whose history greatly aids the diagnosis.

Treatment.—This is by rest, nervous sedatives, and a suitable moral treatment by encouraging words and a confident manner. A few drops of tincture of digitalis, with a few more grains of sodium bromide, repeated every hour may be useful. When the patient is weak and anæmic he should be built up and strengthened by iron, quinine, and strychnine.

TACHYCARDIA AND BRACHYCARDIA.

Paroxysmal Tachycardia.—This term is applied to conditions of the heart in which, without evident cause, there appears paroxysmally an inordinate increase in the number of heart-beats per minute, of which also the patient is conscious. The number of beats may reach 200 and more. The paroxysm may last for a few minutes only or for hours. The intervals between two attacks vary greatly and their recurrence may extend over many years.

Brachycardia; Bradycardia; Slow Heart.—The term brachycardia is applied to unnatural slowness of pulse. It is to be remembered, however, that some healthy persons have naturally a slow pulse, as others have one whose rate is more rapid than the typical 72. A rate of 50 to 60 is not unusual. It may even be slower, as in the case of a patient of mine whose habitual pulse for many years of my observation was 36 to 40. I do not know, however, what it was in youth. Of abnormally infrequent rates, cases with 20 beats are reported, some even at 12, nine, and seven. These instances of extremely slow rate are apt to be associated with such nervous diseases as epilepsy and catalepsy.

In the study of an apparently slow pulse, care must be taken that the actual count is based on a corresponding heart rate, for it sometimes happens that in consequence of the weakness of an alternate systole the heart-beat does not reach the wrist.

Brachycardia occurs under a variety of conditions which have been carefully collected by Riegel. They include the following:—

(1) Convalescence from acute fevers, such as typhoid, pneumonia, diphtheria, acute rheumatism, and the like.

(2) Diseases of the digestive apparatus, especially dyspepsia, but also ulcer and cancer of the stomach.

(3) Rarely in diseases of the respiratory system.

(4) Diseases of the circulatory system, more frequently those involving the muscular structure of the heart, and associated with deficient nutritive supply, especially conditions succeeding obstruction to the coronary artery.

(5) In nephritis.

(6) From the action of toxic agents, including the uræmic poison, lead, alcohol, coffee, and digitalis.

(7) Certain diseases of the nervous system, including apoplexy, brain tumors, especially those involving the medulla and cervical cord.

(8) Finally, affections of the skin and sexual organs.

Explanation of Tachycardia and Brachycardia.—The rationale of the production of tachycardia and brachycardia has excited much discussion and cannot be said to be settled. The heart's action is accelerated by stimulation of the accelerator branch of the sympathetic or by paralysis of the inhibitory root of the pneumogastric, and slowed by stimulation of the latter, directly or reflexly. Direct stimulation is caused by increased pressure, which may be exerted through the cere-

bro-spinal fluid, which in turn may be excited by brain tumor, by hemorrhage, and meningitis. Increased arterial pressure, such, for example, as occurs in *acute* nephritis, is said also to produce cerebral vagus irritation. On the other hand, such slowing is not maintained if the arterial tension is kept up, as in *chronic* Bright's disease and arterial sclerosis. Under such circumstances the excitability of the pneumogastric centre may be said to be exhausted. Deficient nourishment to the heart substance, such as is caused by atheroma and obstruction of the coronary artery, leads also to a slow pulse. Reflexly the pneumogastric may be stimulated by diseases of the abdominal organs, as, for example, the stomach, bowels, and peritoneum. Again, affections of the muscular substance itself of the heart may affect its rate. Thus poisons and high temperature increase the pulse rate. In this way the infectious diseases may produce acceleration. Other poisons slow the pulse possibly, too, by acting on the nerve endings in the heart. Such are the salts of the biliary acids and the uræmic poison, whatever it may be.

His and Romberg, in their studies on the innervation of the heart, were led to believe that the cardiac ganglia are sensitive in function and that they share the increased sensitiveness of the entire nervous system variously caused, and thence reflexly may excite the violent cardiac action of tachycardia. Not unlike this is the explanation of H. C. Wood, who suggests that the paroxysms of tachycardia are due to "discharging lesions" affecting the centres of the accelerator nerves. In some one of these views, perhaps, must be sought the required explanation. On the other hand, anæsthesia of the cardiac ganglia, however induced, would be expected to have the opposite effect. By recalling these facts many of the cases of bradycardia may be explained.

IRREGULAR PULSE.

SYNONYM.—*Arythmia.*

Description of Different Varieties, Peculiarities, and Explanation.—The simplest form of irregular heart is that in which there is an occasional drop or *intermission* in the beat while the pulse in the intervals is perfectly regular. This may occur once only in 20 or more beats and from this point of infrequency increase until it happens once in six or four beats or it may be every second or third beat. Or the pulse may be altogether irregular, *arythmical.* A striking feature of even the simplest form of intermittent pulse or heart is that the omitted drop is commonly recognized by the patient himself, and often it becomes a matter of intense annoyance to him. Here again it is quite important to decide whether the dropped beat at the wrist is the consequence of an omitted systole recognized by absence of the first sound in the ausculted heart or whether it is a simple weak systole which does not send the pulse to the radial artery at the wrist. It is more apt to be the former if the patient recognizes it. It may be constant or occur at regular intervals or occasionally only.

In the latter event it may be associated with some such disturbing

cause as dyspepsia and especially flatulence. It follows the use of tea, coffee, or tobacco, and is in rather frequent association with chronic gout. It follows also mental shock and is especially prone to occur in nervous, hysterical persons. The intermittent pulse thus occurring is vaguely ascribed to nervous influences, and nothing more definite can at present be confidently suggested for its causation. It may be due to some influences of the causes named on the cardiac ganglia of the sympathetic. It is probably at times directly due to organic changes in the heart muscle, especially in fatty degeneration, in which event it is a more serious symptom.

It is characteristic of the more nervous forms of intermittent pulse that they may be removed by exercise or excitement while it often disappears during pyrexia. On the other hand, the effect of exertion on the intermittent pulse of fatty heart is to increase the intermission or convert it into an irregularity. The irregular pulse is indicated by its name. The highest degree is known as *delirium cordis*. This irregularity varies also, may be habitual or occasional, and produced by the same causes as intermission. It may also be induced by temporary or permanent derangements of the respiratory organs through changes in pressure on the large arteries in the chest. This, be it noted, is not an influence on the heart. Irregularity of the pulse and heart is a distinctive symptom of mitral insufficiency. This is very properly ascribed by W. H. Broadbent not to nervous influence but to *variations in pressure due* to traction of the lungs during the inspiratory act on the cavities of the heart, facilitated by the thin, flabby, and feebly resisting walls of a dilated auricle, which is not capable of resisting variations of external pressure, as is the normal heart, whence result disturbing influences which interfere with the regular action of the organ. In mitral stenosis, though the auricle is dilated, the narrow auriculo-ventricular orifice presents the disturbing effect of the varying pressure of the respiratory movements.

In like manner may be explained the *pulsus bigeminus* and *trigeminus*, also common in mitral disease, more especially mitral stenosis. In these two and three beats follow each other in rapid succession, separated by a longer interval. Also the *pulsus paradoxus* of Kussmaul, in which the beats during inspiration are more frequent but less full than during expiration, may be explained in this way, occurring as it does in weak heart, chronic pericarditis, and other conditions where the normal relation of respiration to the heart's dilatation and contraction are interfered with.

Other modifications of the cardiac rhythm are the *gallop-rhythm* and *embryocardia*. The former is so-called because it resembles the footfall of a horse in canter. It is the result of a reduplication of the second sound, a synchronous closure of the aortic and pulmonary valves. More rarely the first sound is seemingly broken into two parts. It occurs in arterio-sclerosis and fatty dilated heart. *Embryocardia*, first described by Stokes, is a condition in which the first sound is shortened and therefore more like the second, the resultant being a sound similar to that of the fœtal heart. It occurs especially in the latter stages of

dilated heart in which the muscular element of the first sound has become lost because of weakness and the sound is purely valvular.

The *dicrotic* pulse is a double beat and is found in every normal sphygmogram. It is the effect of the elastic recoil of the over-distended aorta on the contained blood immediately succeeding the closure of the aortic valve, and is shown in the catacrotic or descending portion of the sphygmogram appended. Only when exaggerated is it felt by the fingers. It may occur abnormally wherever there is a forcible systole and unobstructed arterioles, especially in aortic regurgitation, and although here the incompetent aortic valves do not furnish the requisite resistance, this is furnished by the full ventricle behind them, so that while the dicrotic factor may be delayed it is marked when it occurs. This is seen in the sphygmogram on p. 529. It occurs also in anæmia after venesection, the necessary emptiness of the arterial system being thus produced; after the administration of amyl nitrite and nitroglycerin; also in uncompensated mitral regurgitation, where the emptiness of the arterial system is produced in a different way, that is, by diminishing the amount of blood which enters the aorta with each systole.

FIG. 47.—SHOWING NORMAL PULSE TRACING.

Another variety of double beat is the *pulsus bisferiens*, most frequently noted in aortic stenosis, but also in senile degeneration of the arteries, and which resembles the dicrotic pulse though quite different in its etiology. In it the second beat is a reinforcement near its close of a prolonged systole. Under these circumstances the dicrotic wave will be absent.

Mention should be made in passing of the *anacrotic* pulse, though this is not appreciable to the finger. An anacrotic pulse wave is one in which a more or less marked notch occurs in the ascending limb, as in Fig. 48. It is the pulse of high arterial tension and occurs where the arteries are rigid and do not expand promptly to receive the contents of the ventricle during systole. The walls yield slowly, the pressure is pro-

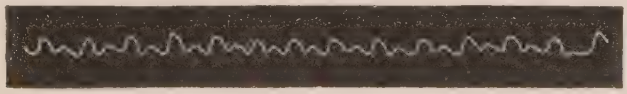


FIG. 48.—PULSE OF HIGH ARTERIAL TENSION.

longed, broadens the top of the sphygmogram, and throws the highest part of the tracing toward the end of the systole and nearer the dicrotic notch. The pulse of prolonged arterial tension is produced by anything which resists the motion of the blood through the capillaries and arterioles, and such causes are numerous. Chronic renal disease, especially interstitial nephritis, is one of them; so are gout, lead poisoning, constipa-

tion, atheroma, or calcification of the arterial walls. The anacrotic pulse is also produced in aortic stenosis, where it has diagnostic value.

Treatment of Palpitation, Tachycardia, Brachycardia, and Arythmia.

It is of the greatest importance that the cause of nervous palpitation should be ascertained, for with its removal recovery may be expected. Indigestion, distant causes of reflex irritation, such as uterine and ovarian disease in women, anæmia, chlorosis, and the like should be carefully sought and eliminated by a proper treatment. During an attack itself the strictest quiet should be enjoined and the patient should be kept in the recumbent posture. It is much more important that this should be done than that heart tonics or heart sedatives should be administered. Yet at this time it may be helpful to give small doses of digitalis, frequently repeated, say three minims (0.18 c. c.), or five drops, every hour, combined with the bromides in ten- to 15-grain (0.660 to one gm.) doses, or aconite and veratrum viride in one-minim (0.06 c. c.) doses as often. When the palpitation is prolonged a belladonna plaster may be applied to the heart, or cold may be applied over the cardiac region.

The treatment of an attack of tachycardia is similar. Every conceivable remedy has been tried by those subject to these attacks and their advisers with results apparently satisfactory at one time and totally disappointing at another. In one remarkable case, a dear medical friend of my own, a glass of ice-water rapidly drunk almost always stopped a paroxysm. I have seen this effect in his case follow in a few minutes. This remedy he arrived at after fifty years' trial of everything he could think of. Brachycardia is perhaps best left alone, unless some evident cause can be found for it and easily removed.

As to treatment between the attacks in addition to the principles of treatment recommended at the onset, the continuous use of strychnine may be expected to be useful. It should be combined with iron and quinine in moderate doses. A very elegant and convenient preparation is the elixir of iron, quinine, and strychnine of the U. S. P. On the other hand, strychnine sometimes causes nervousness. The tincture of nux vomica has been recommended as especially suitable in these cases given in ascending doses until 30 minims (two c. c.) or more are reached. I have been disappointed in it, and to many it is a very disagreeable bitter. Out-door life and exercise, walking, riding, driving, cycling, and mountain-climbing, all contribute to improve the general health and strengthen the heart at the same time. Cold bathing is one of the best measures for the same purpose either at home or at the seaside.

The same principles of treatment apply to intermittent heart and arythmia. When these are the result of organic cardiac disease their treatment is that of the disease. In the treatment of the simple slow pulse any cause of pneumogastric irritation should be sought for and removed, such as dyspeptic states, torpor of the liver, poisoning with retained bile salts, and the like. In the absence, however, of certain knowledge of the presence of such cause the condition is best left alone, as treatment under such circumstances is apt to do more harm than good.

ANGINA PECTORIS, OR STENOCARDIA.

Definition.—An affection of the heart characterized by intense paroxysmal pain, at first usually substernal, extending thence down the arms, especially the left, and up into the neck. It is a symptom rather than a disease, as it is commonly associated with some recognizable organic change in the heart or great vessels, though not always the same change. Often, however, such change cannot be found, and it may be that in rare instances it is a purely functional state.

Etiology.—The commonly assigned cause of angina pectoris, or at least the anatomical condition with which it is most frequently associated, is atheroma of the coronary arteries. It is found, however, also in connection with aortic stenosis, aortic insufficiency, simple hypertrophy of the heart, and other conditions where there is increased arterial tension. The exciting cause of the attack is usually some over-exertion or mental emotion, the stimulus being apparently a distention of the left ventricle, causing an irritation of the sensory nerves of the heart. These are mostly derived from the pneumogastric, but partly from the sympathetic also. The taking of food, even in moderate amount, often excites an attack. Another explanation of the mechanism of the production of the attacks is that it is a spasm of the heart muscle similar to the cramp which occurs in the voluntary muscles in which sensory nerves are compressed with resulting pain. It must be admitted that all explanations are purely speculative, for though total obstruction of the coronary arteries experimentally produced is followed by death, pain has not been found an associated symptom. It is a disease of adults and of men rather than women. Of causes not organic in nature, the excessive use of tobacco seems to be the best substantiated. Other causes include dyspepsia and flatulence, but it is doubtful whether these are alone capable of producing the symptom.

Morbid Anatomy.—It has already been mentioned that atheroma of the coronary arteries is the most constant anatomical change found associated with angina pectoris; also that obstruction of these arteries, experimentally produced, results fatally, though such death is not attended by the pain of angina. On the other hand, many cases of advanced sclerosis of the coronary arteries occur without angina. The other associated conditions named in considering the etiology must also be regarded as a part of the morbid anatomy. A fair estimate of the frequency of such associations may be obtained from W. H. Walshe's statement that in every one of 24 cases he examined during life, distinct signs of changes in the heart, the aorta, or both coexisted. The testimony of G. W. Balfour and P. W. Latham is similar.

Symptoms.—The cardinal symptom is *pain*. Pain in the region of the heart extending up into the region of the neck and down into the arms, especially the left, in the distribution of the ulnar nerve. Associated with this is *shortness of breath*, *præcordial oppression*, and a *sense of impending dissolution*. The pulse is often strikingly natural, though it is also at times unnaturally small, frequent, and at times also irregular. The pain

is often associated with a *numbness* or *tingling in the fingers* or *over the cardiac region.* There is usually *extreme pallor* and an *agony of expression* which is not soon forgotten. The *skin is pale* or ashen grey, and often the *perspiration* stands out in huge beads. The duration of the paroxysm is anything, from a few seconds to a half hour. At the end of this time, or earlier, the patient either passes out of the attack or dies in it. The paroxysms occur at widely different intervals, sometimes once in a few months, sometimes oftener, and sometimes at intervals of a year or more. Neither are they always as painful as described, and it is more than likely that the slighter attacks of cardiac pain, associated with aortic stenosis and sometimes with aneurism, are of the same nature.

Diagnosis.—The only condition with which true angina pectoris is liable to be confounded is the hysterical form known as pseudo-angina. It is not often, however, that practical difficulty occurs in separating the two conditions. The hysterical form is more common in younger women, in nervous and hysterical persons, than the true form, and is associated with other nervous symptoms. In all instances careful examination should be made of the vascular system with a view to detecting alterations in it, such as arterial sclerosis and enlargement of the heart. These will generally be found in some one of their modes of manifestation in true angina. In false angina, as in other manifestations of hysteria, there is something indescribable which will guide the experienced physician aright. In cases of doubt the patient should have the benefit of it.

Prognosis.—True angina is a very grave condition because, although full three-fourths of all persons attacked recover from the first attack, sooner or later a fatal ending may be expected, and no one knows what attack is going to be the last. In some instances attacks recur at intervals of considerable length throughout a lifetime, while in others the first attack proves fatal. G. W. Balfour mentions the case of an old gentleman who had a final fatal attack after an interval of ten years, in which he had enjoyed excellent health.

Treatment.—Morphine is the most efficient remedy for an attack of angina pectoris, and if at hand should be used hypodermically in not less than ¼-grain (0.016 gm.) doses for an adult. An acknowledged remedy has come to be nitrite of amyl, first suggested by Lauder Brunton. A few drops may be placed upon a handkerchief or cotton and inhaled, or, more conveniently, pearls of glass filled with the nitrite are crushed in a handkerchief. The pearls recommend themselves further because they can be conveniently carried, and it is desirable that persons subject to angina pectoris should always have the drug at hand. Chloroform may also be used instead of nitrite of amyl, if more convenient. Counter-irritation should be simultaneously applied. The ordinary mustard-plaster is a most convenient and efficient measure for the purpose.

Prophylaxis is exceedingly important, and best accomplished by avoiding the exciting causes commonly responsible for the paroxysms, over-exertion, over-eating, and mental excitement. The patient subject to angina should never hurry, or get into a passion, or become excited in any way. The use of all indigestible articles of food should be carefully avoided. Nitroglycerin is sometimes efficient as a prophylactic in doses

of $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.00065 to 0.0013 gm.) once in four hours. The alcoholic solution of such strength that one minim represents $\frac{1}{100}$ grain (0.0006 gm.) is the best preparation. Nitrite of sodium may be used in from three- to five-grain (0.1944 to 0.32 gm.) doses. Other remedies recommended with a view to averting the attack are arsenic, nitrate of silver, the bromides, and especially iodide of potassium, the long-continued use of full doses of which has apparently sufficed to prevent the recurrence of attacks previously maintained. Arsenic is an admirable tonic in doses of $\frac{1}{50}$ to $\frac{1}{30}$ grain (0.0012 to 0.002 gm.), though it is doubtful whether it has any prophylactic power over angina. While sudden and over-exertion are to be avoided, carefully graduated exercise is to be recommended, for, like all organs, the heart is strengthened and invigorated by exercise properly regulated.

The treatment of hysterical or pseudo-angina is that of hysteria elsewhere.

DISEASES OF THE BLOOD-VESSELS.

ARTERIO-SCLEROSIS.

SYNONYMS.—*Endarteritis chronica deformans; Atheroma of Blood-vessels; Arterio-capillary Fibrosis.*

Definition.—A disease chiefly of arteries, consisting in inflammation first of the intima and extending also to the media and adventitia.

Etiology.—There is tendency to atheroma in the arteries of the old as an evolution process quite independently of exciting causes. This tendency also varies greatly in different families, being very strong in some and absent in others. Men are decidedly more liable than women. There are, however, many exciting causes, among which are over-eating and drinking, syphilis, the gouty poison, and lead. Chronic Bright's disease is especially frequently succeeded by it, more rarely acute articular rheumatism. In the latter the rheumatic poison is probably the responsible agent, but in Bright's disease it is retained excrementitious matter. Two classes of cases must, however, be associated with Bright's disease, in one of which the arterio-sclerosis is general and primary, causing interstitial nephritis, and in the other it is secondary, the result of the Bright's disease. One set of observers regard all cases of interstitial nephritis as secondary. Among these the late Sir William Gull and Thomas Sutton, in England, and Arthur V. Meigs, of Philadelphia, have been conspicuous by their writings. Still another cause is increased arterial tension due to prolonged muscular exertion.

Morbid Anatomy.—The aorta is the most frequent and conspicuous seat of the changes ascribed to chronic endarteritis, but the carotids, subclavians, brachials, radials, and ulnars, the iliacs, femorals, and especially the arteries of the brain and coronary arteries of the heart, are frequently involved. The arteries to viscera, like the stomach and liver, are

rarely affected, while the pulmonary arteries take an intermediate place. On the other hand, they are sometimes invaded to the exclusion of the aorta. Whatever invites high tension in the lesser circulation tends to produce sclerosis in these vessels. The portal vein may also be invaded. The superficial arteries thus affected are easily recognized. They are tortuous, stand out conspicuously, and feel hard to the finger, under which they may be made to roll. These features are often recognizable in the temporals and less plainly in the radials. The smaller arteries and veins with transparent walls, especially in the brain, exhibit to the naked eye white patches which are the seat of the atheroma. On slitting open the inner surface of these and other arteries involved, it will be found that they have lost their natural smoothness and are rough and uneven, while the lumen is more or less encroached upon.

Minutely examined, the appearances vary with the stage. The first stage is that of cellular infiltration, represented by the translucent yellowish areas of intima thickened to three or four times its natural thickness. Later these young cells are in part converted into connective tissue, causing the primary hardness of the vessel walls. In the second stage the cells of the connective tissue and the surface cells of the intima undergo fatty degeneration, and the intercellular substance liquefies. In the third stage, which is not reached in the smaller arteries, or, indeed, usually in those below the aorta, there occurs a further liquefaction with the formation of the so-called atheromatous abscess, whose contents are not pus, but the well-known atherom-pulp, representing the debris of fattily degenerated cells, including fat-drops and cholesterin crystals. Alongside of the atheromatous patches are also plates or scales of calcareous infiltration of the intima produced by a deposit of lime salts in the intercellular substance of the deeper layers. The atheromatous abscess sometimes undermines the intima, forming sinuous cavities, and after evacuation there results the atheromatous ulcer. Both the limy plates and ulcers furnish inequalities which favor thrombosis. In the later stages of the more diffuse form of arterio-sclerosis, especially studied by Councilman, the media or muscular coat and the adventitia are also invaded, the former mainly by atrophic changes, alongside of which, at times, is also a homogeneous hyaline infiltration. In this form the capillary walls are also thickened, especially those of the glomeruli of the kidneys, in some of which the vessels become obliterated.

A *calcareous* infiltration of the *muscular* coat without previous inflammation is also found in old age in arteries like the radial, crural, and temporal. Still another primary degeneration is the *fatty erosion* of Virchow extending through the intima and media as a transverse fissure thought to be the starting-point at times of dissecting aneurism.

The effect of these changes is to produce rigidity and narrowing of the vessel, a loss of the propulsive power residing in the elastic coat, a slowing of the current, and increased intravascular pressure. These events tax the compensating power of the left ventricle, which therefore hypertrophies. This hypertrophy keeps up so long as its nutrition is maintained.

But another effect of obstructed circulation is an impairment of local

nutrition, some of the consequences of which have already been considered in the study of the fibroid heart. Similar interstitial overgrowth and contraction are also met with in the kidney and have also been referred to. Localized softening of the brain also succeeds upon atheroma, though this event is usually preceded by thrombotic obstruction favored by the sclerosis. A more frequent accident to the brain is rupture of one of these atheromatous vessels succeeded by the symptoms of apoplexy and hemiplegia. Such rupture may be preceded by an aneurismal dilatation. Finally, aneurism of the larger vessels has for its almost indispensable condition, except in traumatic cases, atheroma of the dilated vessel. Both events, the primary atheroma and the subsequent dilatation, are favored by the increased intravascular pressure.

Symptoms.—Superficial vessels in a state of atheroma are easily detected by their *dilated*, *tortuous*, *pulsating appearance* in the temples, while in other situations, as at the wrist, antebrachial and popliteal spaces they may be recognized more or less by the touch. Distinction should be made between simple increase of tension and thickening of vessel walls, though the two are constantly associated. The vessel in both instances is hard and requires some force to compress it, and between beats it is still full and can be rolled under the finger, but the artery with the thickened wall, if firmly enough compressed to obliterate the blood-current, can still be felt beyond the seat of compression. In many instances, on the other hand, the changes escape detection until a fatal apoplexy gives notice of their presence. In most of these instances, however, had attention been directed to the patient, the above-described condition of the arteries would probably have been recognized, while a certain degree of hypertrophy of the left ventricle would also, perhaps, have been detected. It does not follow, however, that an atheroma in one place implies an atheroma in another, since fatal rupture of an artery in the brain has occurred where there has been no sign in the radials, and vice versa. Nor is every hypertrophy of the left ventricle the result of an atheroma of the arteries. For the atheroma may be the result of a prolonged hypertrophy and the increased tension incident to it, or the two may be the result of the same cause as contracted kidney. Cardiac hypertrophy is not always demonstrable to percussion, as the enlarged heart may be covered by an emphysematous lung, which may also be present in the aged, in whom atheroma is most prone to occur. On the other hand, the usual sharp accentuation of the aortic second sound is present if the hypertrophy has not given way to dilatation or fibroid induration.

Cardiac murmurs do not occur unless the atheroma invades the valves to produce insufficiency, stenosis, or roughening of the aortic orifice or aorta near the orifice. This is not so very rare in old persons apart from the relative insufficiency due to aortic dilatation.

For the reasons mentioned, the pulse is prolonged, hard, and tense—the pulsus tardus—while its sphygmogram is very characteristic : a slow, oblique ascent, a broad top ; a slow descent and absence of the dicrotic rise, which in the normal state depends on the elasticity absent in the diseased vessel. Owing to the same slow transmission of the pulse wave

the pulse is sometimes retarded at the wrist, while the rate is also slow. At other times it is frequent and irregular, especially toward the end when the heart begins to fail.

The consequences of atheroma of vessels of the lower extremities include muscular weakness, stiffness, a tottering gait. In extreme cases gangrene of the lower extremities may result from obstruction to their arteries due to thrombosis invited by the atheroma. Such termination I have met in contracted kidney. With the supervention of cardiac dilatation and heart failure there appear paralysis, præcordial oppression

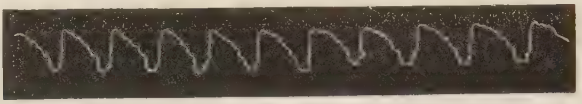


FIG. 49.—SPHYGMOGRAM OF AN ATHEROMATOUS VESSEL.

on slight exertion, dyspnœa, œdema, pulmonary congestion, scanty urine, vertigo, angina pectoris,—in a word, all the symptoms of chronic heart disease. Finally, other symptoms are those of the morbid states it causes, *i. e.*, apoplexy, contracted or senile kidney, atheroma of the coronary artery and its consequences.

Treatment.—Treatment is mainly the removal of conditions causing it, such as too free living, gout, lead poisoning, and syphilis; together with rest and quiet, the avoidance of excitement, also the free use of diluent drinks and aperients to lower the arterial tension, a slight increase of which is often the last straw required to produce an apoplexy. The iodide of potassium reputed as a remedy for fibroid overgrowth may be availed of and there can be no harm in placing these patients on moderate doses of this drug together with corrosive sublimate, five grains (0.32 gm.) of the former to $\frac{1}{50}$ to $\frac{1}{25}$ grain (0.00132 to 0.00234 gm.) of the latter. The latter especially should be kept up for a long time.

ANEURISM.

Definition.—An aneurism is a more or less circumscribed dilatation of a blood-vessel. Aneurism is known as true or false. A *true* aneurism is one in which, at the outset, all three coats of the blood-vessel share in the dilatation, though one or two may disappear later in the course of its growth. A *false* aneurism, on the other hand, starts at the outset with a laceration of one of the coats.

I. True aneurism may be saccular, fusiform or spindle-shaped, cylindrical and cirsoid. The "cirsoid" or varicose aneurism is one in which a blood-vessel, one of medium size, and its branches are irregularly dilated and contorted like a varicose vein, whence the name "varix," a dilated vein. The "invaginating" aneurism is a rare form of cylindrical aneurism, in which the cylindrical sac overlaps at either or both ends the main trunk of the artery involved. Saccular and fusiform aneurisms are for the most part frequent. The "neck" of an aneurism is a con-

stricted portion by which a saccular aneurism is attached to the main trunk.

II. False aneurism includes :—

(1) Traumatic and dissecting aneurism. In traumatic aneurism the initial event is some injury from without to one or more of the coats of the vessels, as the result of which the resistance to intra-vascular pressure is diminished and a protrusion of the intima through the yielding media takes place, the latter being the most passive of all the coats. The simplest illustration of this form of aneurism is the antibrachial aneurism caused by accidental wounding of the brachial artery in venesection of the median vein. The blood pushes out the intima and antibrachial fascia and forms a sac communicating with the artery through the wound.

(2) A second form is the aneurismal varix or anastomotic aneurism, in which the blood from the wounded artery passes directly into the adjacent vein through the wound made at the same time, causing a dilatation of the vein. This is resisted by the valves, which, however, give away to the extent of two, three, and even more pairs before the current is successfully resisted.

The dissecting aneurism, so called, involves the aorta, in which in consequence of a perforation through the intima and media the blood dissects between them and the adventitia. The initial slit is found most frequently in the inner and posterior portion about one inch (2.5 cm.) above the semilunar valves. The blood may dissect from this point around the arch of the aorta, even as low as the diaphragm, before it returns to the lumen of the vessel. Even the visceral pericardium has been thus separated by an aneurism which projects into the pericardium, rupturing finally into the pericardial sac.

Etiology.—The aneurism most frequently encountered by the physician is the true fusiform or saccular form. Its most frequent essential cause is endarteritis and its consequences, including the more acute stage of cellular infiltration as well as atheroma. The coats thus weakened yield to the intra-vascular pressure. The intima is capable of a considerable degree of expansion without rupture, while the media is entirely passive and yields very soon to the distending force. The adventitia alone seeks to guard the sac against rupture by reactive overgrowth. The causes of endarteritis already discussed, such as syphilis, alcohol, and the like, are responsible for the more usual forms of arterio-sclerosis which furnish the initial lesion of aneurism. But weakening of the coats is caused also in the smaller vessels by emboli, after the lodgment of which the proximal side of the vessel often becomes dilated. Such embolus may excite an endarteritis or may occasion direct violence to the vessel walls if it be hard or sharp, as is often the case with a fragment of a calcified valve. Parasites, muscular compression exerted by muscles in certain situations, may also produce it. Such may be the origin of popliteal aneurisms so frequent in footmen who maintain a rigidly erect position. Finally, disturbances of innervation are considered capable of causing dilatation, and to such influence is ascribed the varicose aneurisms of the arteries of the scalp, of the temporal and popliteal.

Aneurism of the Thoracic Aorta.

Thoracic aneurism occurs in the arch of the aorta, in its ascending transverse and descending portions, and in the thoracic aorta below the arch. Such aneurism may but slightly exceed the normal caliber of the vessel or it may be four inches (ten cm.) or more in diameter.

The greater frequency of aneurism in the male sex and during early middle life is recognized. To the preëxisting conditions of atheroma there may be added the effect of extreme exertion in lifting or muscular strain of any kind, the effect of which is to always increase intra-vascular pressure. Partly because they are points of least resistance and partly because they are in the line of successive impingement of the whirling blood stream there are certain points of selection in the aorta which are quite constantly seats for beginning aneurism. These are shown in the appended drawing.

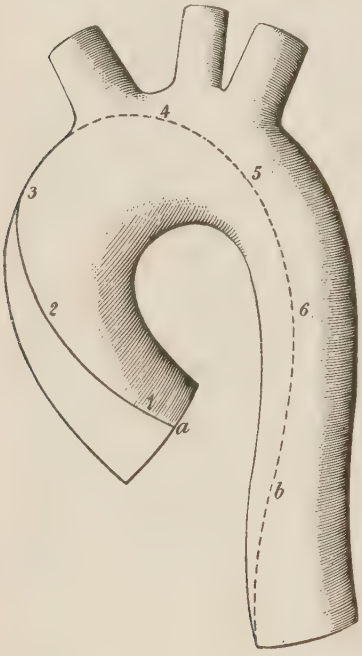


Fig. 50.

The first point (1) of election is the beginning of the aorta directly behind the trunk of the pulmonary artery. Aneurism there originating may produce early hypertrophy of the right ventricle because of the resistance to the outward flow of the blood through this vessel, a basic murmur in the pulmonary area, relative insufficiency of the tricuspid valve and venous pulse, with a possible ultimate perforation into the pericardium or pulmonary artery. The second point (2) is the favorite seat of aneurism of the ascending limb of the arch, behind the sternum at the manubrio-gladiolar junction, at which place it often bores its way through the sternum as a sacculated aneurism which may finally burst through the external integument. The third seat (3) is at the convexity of the arch toward the apex of the right lung. The pleural cavity at this point is soon obliterated by adhesive inflammation, through which the aneurism bores its way, rupturing into the bronchioles of the apex, producing fatal hæmoptysis. The fourth (4) is between the innominate and left carotid at the apex of the arch behind the trachea. It may perforate into the trachea before attaining very large size. The fifth position is posterior in the descending aorta, between the left subclavian and the isthmus of the aorta to the left of the vertebral column. The aneurism here is more commonly a cylindrical dilatation. The remaining aneurisms of the aorta all point more or less toward the vertebræ, but the greater resistance to their formation in that direction favors lateral development. Ultimately they rupture with hemorrhage into the pleural or abdominal cavity. Aneu-

risms of the thoracic aorta lying close upon the diaphragm may bore their way between the trunk muscles behind, attaining often large size without perforation.

Symptoms.—Apart from the physical signs the most important of the symptoms due to thoracic aneurism are the result of pressure of the growing aneurism. Hence they are called pressure symptoms.

The first of these is *pain*, which may be sharp and acute when nerves are directly involved, or dull and boring when the result of pressure on bone. In the latter case, too, it is localized; in the former it may extend all over the chest and down the arms, simulating angina pectoris. It may be unilateral. It may occur in aneurism of any part of the arch, but is more frequent in that of the ascending limb.

Shortness of breath, especially on exertion, is a frequent symptom. It may be due to pressure of the aneurism on the trachea or on a bronchus, especially the left. Dyspnœa may be increased on changing position. *Dysphagia* from pressure of the tumor on the œsophagus is a frequent symptom, especially in aneurism of the descending aorta anywhere in the thorax.

Other signs are *alterations in the voice*, such as hoarseness, aphonia, stridor; also brassy cough. Some of these symptoms may be produced by direct pressure on the trachea itself, others by pressure upon the left recurrent laryngeal nerve. A stridulous voice unaccompanied by dysphagia or aphonia was early pointed out by Thomas Jolliffe Tufnell as indicating that the pressure is on the right side of the trachea and does not affect the œsophagus or recurrent laryngeal nerve. Cough may be caused by tracheal pressure or by a resulting tracheo-bronchitis with copious, thin, or mucous expectoration, sometimes bloody. On the other hand, hoarseness, aphonia, and various degrees of paralysis of the vocal cord are due to involvement of the recurrent laryngeal nerve, commonly the left, which passes around the arch of the aorta and is therefore more likely to be involved than the right. The paralytic phenomena may be present without other laryngeal symptoms. Hence any alteration of voice in a person exhibiting palpitation or dyspnœa calls for a laryngoscopic examination.

Such examination may show little alteration in the position of the vocal cords in *ordinary* breathing, or the left may be a little nearer the median line. In total paralysis of the left vocal cord this cord stands in the so-called cadaveric position, *i. e.*, midway between the position of ordinary respiration and phonation. On *deep inspiration*, however, the right vocal cord is well abducted, the left remaining quiescent. The attempt at *phonation* is more or less abortive. During it the right vocal cord may go to the median line, leaving a small opening between it and the motionless left cord, or it may even cross the line to its paralyzed neighbor.

If only the abductor twigs of the left recurrent laryngeal nerve are involved in the pressure, there ensues gradually a permanent shortening or "paralytic contracture" of the antagonistic adductors, and the affected cord is drawn by this into a position of constant phonation,—that is, into the median line. The result is that breathing may be entirely

unobstructed and the voice natural, the paralyzed cord being in the position of adduction, while its tension is mainly regulated by the external branch of the superior laryngeal nerve, which is uninfluenced in aortic aneurism.*

These phenomena imply, of course, a functional destruction of the nerve, a conduction paralysis which may be the result of simple pressure, leading sooner or later to structural change or a primary neuritis. Such neuritis and resulting irritation of the entire pneumogastric may account for certain attacks of extreme *dyspnœa* sometimes experienced by subjects of aortic aneurism. Associated with the neural degeneration is also found atrophy of the left abductor muscle, the crico-arytenoid, while the adductors remain nearly intact. Constant dyspnœa is more likely to be due to direct compression of the trachea.

Other *nerves* may also be *compressed*, especially the intercostal, vagus, and sympathetic. By compression of the intercostal nerves pain may be caused, of the vagus, vomiting, and of the sympathetic inequality of the pupils and unilateral sweating. I remember well a very stubborn case of intercostal neuralgia in my own practice which turned out to be caused by aneurism of the descending aorta, confirmed by autopsy. Then there is the *tracheal tugging* of aneurism first described by Surgeon-Major Porter, regarded as important by English physicians, but I have not met it once. This is a dragging downward of the larynx with each systole of the heart. The patient sits with his head slightly thrown forward so as to relax the neck and the examiner stands behind him. The trachea is held up by gently inserting the ends of the fingers under the edge of the cricoid cartilage, when with each impulse the larynx is felt to be pulled downward. It is said that it may be the sole sign of aneurism, and a sign, also, that the position of the aneurism is such as to involve the posterior aspect of the arch. It is said never to be present in aneurism of the innominate. Occasionally there is a systolic tracheal blowing caused by forcing the air out of the windpipe by the systole.

Alteration in the *pulse* in distal arteries is also a sign of considerable diagnostic value. It is chiefly when the aneurism involves the origin of blood-vessels leading to those arteries, as the innominate on the right and the carotid or subclavian on the left. If the right radial pulse is enfeebled or delayed, the aneurism will be on the right, involving the origin of the innominate; if on the left, involving the left subclavian. Great care should be taken in the examination, and it should be made from the centre to the periphery, that is, the carotids, the subclavians, the brachials, and the radials should be successively examined, as recommended by Sansom. These effects are variously produced. Thus the aneurism may narrow or distort the orifice of the blood-vessel by traction on it. Or there may be atheromatous change in the branch vessel analogous to that in the aorta itself, which may cause narrowing of the orifice, while the possibility of this, in the absence of aneurism, is also to be remembered. Or the aneurismal sac may act as the elastic air-chamber in a pump, diminishing thus the pulsatile force in the vessel and branches beyond.

* For the muscles involved see Diseases of the Larynx.

It is particularly in the arteries of the lower extremities by aneurism of the descending thoracic and abdominal aorta that this air-chamber effect is seen, though the pulse, even in the abdominal aorta and its branches, has been thus obliterated by a large thoracic aneurism.

Physical Signs.—Inspection does not always discover changes, but if the sac grows outwardly, sooner or later a swelling makes its appearance, to the right of the sternum if in the ascending limb, possibly raising a rib or the end of the clavicle; above and behind the sternum if in the transverse portion, raising the manubrium or boring its way through it; and to the left of the sternum if in the descending limb of the arch. As the tumor protrudes the skin becomes smooth, shining, and tense over it and previous to rupture may become gangrenous. Such a tumor may pulsate or not. The aneurism is, as it were, a rudimental heart, dilating in all directions with every jet of blood that is shot into it so long as the wall is yielding, and contracting on the withdrawal of the intravascular pressure. Should this property be lost, either as the result of calcification or the lining of the sac with successive layers of coagulum, such dilatation becomes impossible, and pulsation does not occur. The pulsation is, however, of great importance in the diagnosis. When present it is synchronous with the systole of the ventricles. The heart itself is sometimes displaced downward, as may be recognized by the lowering of the apex sometimes as low as the 6th interspace and outside the mammillary line. Less frequent is hypertrophy of the left ventricle, and when present not as extreme as in aortic valve disease.

If the aneurismal tumor press upon the great veins of the neck there may be venous engorgement and œdema on one side of the neck or both, according as the innominate of one side only is compressed or the descending cava itself. The aneurism may rarely rupture into the descending cava, producing a form of varicose aneurism, when there occur, in addition to the ordinary signs of aneurism, sudden distention of the veins in the upper half of the body, œdema of the face, hands, and arms, cyanosis, systolic venous pulse, and purring thrill.

Palpation also appreciates the impulse of the aneurism if it is visible, and sometimes when it is not visible. This beating is peculiar, being *expansile*, and by this peculiarity differs from the rising of a tumor over a pulsating blood-vessel. Sometimes there is a double beat, the second and weaker being the usual recoil following closure of the aortic valves. A *thrill* is also often felt, a vibration in the walls of the sac caused by the whirl of the blood in it. It is by no means, however, invariable, and it may come and go. Very great *tenderness* is sometimes present over the seat of the protruding aneurism.

Percussion over the swelling of an aneurism invariably elicits dulness, varying greatly in extent. On the other hand, the adjacent lung may be compressed by an area of dulness thus extended beyond the tumor itself. The dulness is usually in the right upper intercostal spaces, especially if the aneurism is in the ascending limb of the arch. Aneurisms in the transverse portion produce dulness in the middle line under the manubrium and toward the left of the sternum, while aneurisms of the descending part may produce dulness in the left interscapular and scapular

regions. Sometimes the impairment of resonance precedes the pulsation, though such dulness is of uncertain significance.

Auscultation is no exception, as compared with the other modes of physical investigation, in the inconstancy of its information sometimes furnishing the most distinctive signs, while at others it is totally negative. The murmur or bruit heard over an aneurism varies. Sometimes but one murmur is produced, systolic, corresponding with the first sound over the ventricles, but more intense; more rarely it is diastolic only. Not infrequently there is a combined or double murmur, both systolic and diastolic, the first intense and prolonged, the second fainter and shorter. It varies greatly, being sometimes rough, sometimes soft, sometimes musical. The murmur is, however, often absent. The mechanism of these sounds is not settled. The systolic is most easily explained. There can be little doubt that it is produced by the inequalities which meet the entrance of the blood into the sac. When the aneurism is at the beginning of the aorta, the diastolic murmur will probably be an aortic regurgitant murmur, due to relative insufficiency of the aortic valves. When the aneurism is distant from the aortic orifice, the diastolic murmur may be due to the recoil of the distended sac propelling the blood through the outlet with additional force, or the whirling of the blood through the sac. Rarely there is a diastolic murmur only, probably thus caused. A much more constant symptom is a ringing, aortic second sound, which is, in fact, rarely absent in aneurism of the arch not involving the aortic orifice.

But any one or all of these signs may be wanting. Particularly is this the case where the aneurism occurs just after the aorta has left the heart. The most valuable are the pulsations, distinct and separate from those of the heart, or, as graphically put by Da Costa, "two hearts apparently each with its own distinct beat, its own distinct sound." *

Is it possible to determine the portion of the aorta involved by aneurism? Yes, with a certain degree of probability:—

In aneurism of the Ascending Aorta there is more apt to be pain like that of angina pectoris, dyspnœa, dulness to the *right* of the manubrium sterni from 2d intercostal space upward, pulsation in the same region, displacement of the heart downward and to the left, delayed pulse in the peripheral arteries as contrasted with the heart's impulse, compression symptoms involving the sympathetic and the area of the superior cava, pressure upon the pulmonary artery producing a pulmonic systolic murmur, with hypertrophy and dilatation of the left ventricle.

Aneurism of the Transverse Part of Arch furnishes more particularly pulsation in the fossa jugularis, dulness on percussion over the manubrium and to its *left* in the 1st intercostal space, with possible narrowing of the orifices of the innominate, the left carotid, or left subclavian, and resulting inequality of the pulse in the head and arm, pressure on the left innominate vein, with resulting congestion and œdema of the left half of the neck and head. It is when in this situation that aneurism compresses the left recurrent laryngeal nerve and causes paralysis of

* Da Costa, "Medical Diagnoses," 8th ed., 1895, p. 507.

the left vocal cord, presses on the trachea with resulting stridor and cough, and on the left bronchus, producing inspiratory dyspnœa.

In aneurism of the DESCENDING AORTA * we look for the pulsation posteriorly to the left of the vertebral column opposite the angle of the scapula or below. The bruit is faint or wanting. In this situation in consequence of the air-chamber effect we are apt to find smallness of the crural pulse as contrasted with the radial, and symptoms of pressure upon the left lower azygos or hemiazygos vein, *i. e.*, œdema of the upper part of the abdomen and pleuritic effusion; also pressure on and stenosis of the œsophagus and left bronchus. The intercostal nerves may be compressed, producing intense pain in the course of their distribution, the vertebral column may also be eroded, the spinal canal opened, and the cord compressed, with resulting paraplegia. If the aneurism project forward, which is rarely the case, it may press upon and displace the heart, causing palpitation, or it may also compress the œsophagus, causing painful deglutition. It sometimes ulcerates and breaks into the œsophagus.

Aneurism of the ABDOMINAL AORTA furnishes a pulsating tumor to the left of the vertebral column, to the left and above the umbilicus. The bifurcation of the aorta takes place on the 4th lumbar vertebra, which point corresponds to the umbilicus. Sometimes a thrill may be felt and a systolic murmur heard, rarely a double murmur. Here, too, the smallness of the crural pulses as contrasted with the heart's impulse and the radial pulse may be observed, while in some cases the crural pulses disappear altogether. The symptoms vary somewhat, according as the aneurism grows backward or toward the front. In the former case pain is also a striking symptom and may be of two kinds, a fixed and constant pain in the back caused by the pressure of the tumor on the solar plexus and splanchnic nerves, or a sharp lancinating pain radiating along the branches of the lumbar nerves which are pressed on by the tumor, whence pain in the loins, testes, hypogastrium, and in the lower limb, usually of the left side. If the sac grows anteriorly gastro-intestinal symptoms are apt to be present, such as vomiting, gastralgia, diarrhœa, and even symptoms of obstruction. Pain is also present, but is more likely to be fixed in the loins, epigastrium, or some part of the abdomen. Erosion of the spine is much rarer in abdominal aneurism than in thoracic. In emaciated persons the abdominal aorta sometimes pulsates so plainly that one is strongly reminded of aneurism, and I have myself been misled by such pulsation, but under these circumstances there is absence of the systolic murmur and of the alterations in the pulse of the arteries of the lower extremity and none of the pain described. Indeed, abdominal pulsation far more frequently occurs without aneurism than with it.

Aneurism of the INNOMINATE is especially indicated by its murmur,

* The *descending part of the arch of* the aorta is somewhat arbitrarily terminated by anatomists at the lower end of the 5th dorsal vertebra, below which it is called the *descending thoracic aorta*, which terminates at the opening of the diaphragm in front of the last dorsal vertebra, below which it is the abdominal aorta. The symptoms of aneurism of the descending part of the arch and the descending thoracic aorta do not differ widely.

thrill, and impulse in the vicinity of the inner end of the right clavicle, which is sometimes raised by the resulting tumor; also by the comparative absence of signs of pressure on the larynx or œsophagus. The differences in the right radial pulse alluded to are especially here present. Compression of the right subclavian and right carotid diminishes the beat of the tumor, but is without effect in aortic aneurism. Nor are there percussion signs of enlargement of the aorta.

If the SUBCLAVIAN is involved, the signs are further outward, on the outer side of the sterno-cleido-mastoid, while in aneurism of the innominate they are found on the inner or tracheal side. To those named may be added pressure symptoms upon the subclavian vein, producing swelling of the arm and neck; upon the right recurrent laryngeal, producing defective speech and dyspnœa; on the sympathetic, producing contraction of the pupil, and on the brachial plexus of nerves, pain. Especially would these signs point to aneurism of the subclavian if the pulse of the carotids is uninfluenced while the right or left radial pulse is.

The very rare condition of aneurism of the PULMONARY ARTERY may produce a swelling, with the other local symptoms described, to the left of the sternum, in the 2d interspace. A murmur is less constant and is not conducted into the vessels of the neck, while the superficial pressure signs are more conspicuous. There is lividity of the face, with dropsy, and the dyspnœa is naturally very great. There is no cough or voice alteration. It is to be remembered, however, that the swelling of an aneurism of the arch of the aorta *may* extend to the left of the sternum. Such an aneurism may break into the pulmonary artery.

An aneurism of the *heart* is not recognizable, though it may be suspected if there is bulging succeeding the signs of fibroid disease of the organ.

Differential Diagnosis.—Further diagnosis distinguishes aneurism of the aorta mainly from mediastinal tumors. There may be the same percussion signs, and there is often similar pain; there is also pulsation, but instead of the expansile pulsation extending in all directions, there is in mediastinal tumor more upheaving. Percussion dulness is more irregular in mediastinal tumor. Murmurs are not usual in the latter. The ringing second sound is absent if aneurism is absent; it may not be present if aneurism is. Tracheal tugging does not occur, nor do differences in the pulse or changes in the voice. The state of the blood-vessels usually associated with aneurism must be sought for. Fever is often present in mediastinal tumor; very rarely in aneurism. A differential diagnosis is often impossible, and experts have held opposite opinions on the same case. Should the patient develop a cachectic state and secondary glandular enlargements appear, the presumption is in favor of mediastinal disease.

The resemblance of some of the symptoms of aneurism of the ascending aorta to some of those of aortic incompetency is very close. The same pulsating aorta, the same double basic murmur with impaired resonance at the right of the sternum, may be present. I have seen a case diagnosticated aortic regurgitation with stenosis where the

autopsy disclosed perfect semilunar valves with, however, aneurism and relative insufficiency, which caused the diastolic murmur symptoms. In aneurism there is less hypertrophy of the heart than in aortic valvular disease. The age of the patient, if under forty, especially the history of heart disease in early life, the history of rheumatism, and the absence of the causes of atheromatous vessels, point to valvular disease. Though there may be pulsation at the root of the neck in both, in aortic incompetency the same strong pulse-beat extends to the wrists. Traube's double sound in the femorals and popliteals, though possibly otherwise caused, is still more frequently associated with aortic incompetency than any other lesion. Simple dilatation may indeed be present in aortic incompetency, but the pressure signs are wanting.

A pulsating empyema on either side of the upper sternum sometimes closely resembles a pulsating aneurism, and the illusion is more complete because the pulsation is expansile. I well remember a case of my own in which the pulsation to the left of the sternum was so like that of an aneurism that I hesitated to use the exploring needle. Pulsating empyemas are generally to the left of the sternum. Other signs of aneurism are, however, wanting, unless it be tenderness, which may be present. A rare condition is a narrowing of the aorta below the remains of the ductus arteriosus at the junction of the arch with the thoracic aorta, which is also rarely associated with valvular disease. It, too, produces small delayed pulse in the femorals, a thrill and murmur over the upper part of the sternum, but the extraordinary enlargement of the collateral vessels, especially the mammary and epigastric arteries, should set the question at rest.

How shall the symptoms, which are, also, so much like those of a laryngitis, be interpreted as due to aneurism instead of the latter affection in the absence of the physical signs of aneurism?

In acute laryngitis we have often the cause—exposure to cold—to help us, though in chronic we have not. In laryngitis there is more huskiness and less of stridor in the voice, nor is the cough so brassy, or the voice so uniformly changed; it is more likely to alternate with normal voice. In aneurism the voice grows progressively weak until aphonia results. The dyspnœa is more often attended with wheezing, and is sometimes relieved for a time by coughing. Stokes called attention to the fact that in aneurism the stridor of the voice seems to come from the notch of the sternum rather than from the larynx itself. In aneurism the breathing is more apt to differ in the two lungs. Then we have the laryngoscopic picture. There is no swelling of the cords in aneurism and there may be the paralytic phenomena detailed. Finally, in laryngitis there may be fever.

Prognosis.—Aneurism is not infrequently found at necropsy without having been suspected. In other cases the fatal termination is the first notification of its presence. Where an aneurism of the aorta is so developed as to exhibit its usual signs plainly the prognosis is sooner or later fatal in some one of the modes already described. To foretell which of these directions perforation will occur depends upon the accuracy with which diagnosis of its position can be made, and such diagnosis is at best

a matter of probability. Only in cases where aneurism slowly erodes the anterior wall of the chest is there a gradual termination. Then there are sometimes repeated small hemorrhages, which gradually reduce the strength of the patient, who ultimately dies of exhaustion or of an ultimately fatal large hemorrhage. Perforation into the vena cava, pulmonary artery, and right side of the heart is a rare termination. The course of the disease may, however, be prolonged many months, and, if instituted early, treatment may contribute to such prolongation. Where death does not occur from sudden hemorrhage, the symptoms may assume the type of chronic heart disease, for which, indeed, the condition is sometimes taken by the untrained observer. With failing heart come dyspnœa, palpitation, dropsy, and death.

Treatment.—We seek in the treatment of aneurism to diminish intravascular pressure and restore the integrity of the vessel. The former may be accomplished in a degree by placing the patient under conditions which will avert the causes of such increased intravascular pressure, which is constantly coöperating with the disease of the artery to produce further dilatation and ultimate rupture of the blood-vessel. This is, of course, best accomplished by absolute rest. It is plain the less frequently the heart beats and throws the weight of its blood against the weak blood-vessel, the longer will that blood-vessel last, while it is known to every student that the heart beats less frequently in the sitting than in the standing posture, and less in the recumbent than in the sitting position. On the other hand, it is plain that absolute rest is an impossibility. Yet it may be approximated in various degrees. It is impossible also to restore the integrity of the vessel, but to this end also measures are suggested, measures which have for their immediate purpose coagulation of the blood in the vessel and obliteration of the sac. That this sometimes occurs numerous autopsies also attest.

The method which has met most favor is that now known as Tufnell's treatment, though Valsalva originally suggested a restricted diet and practised frequent venesections. Bellingham advised starvation without bleeding. It was, however, revived by the late T. Jolliffe Tufnell and modified by W. G. Balfour. Tufnell's treatment consists in absolute mental and physical rest in the recumbent position, together with a moderate, dry diet. The object of this is to diminish the blood pressure and volume of blood, to increase the proportion of fibrin in the latter, and to promote its coagulation. The method is as follows :—

For breakfast, two ounces of bread and butter and two ounces of milk ; for dinner, two or three ounces of meat and three or four ounces of milk or claret ; for supper, two ounces of bread and two ounces of milk. Thus it is hoped to diminish the blood volume and reduce the pressure within the sac, to render the blood more fibrinous and favor coagulation. The addition of iodide of potassium is Balfour's modification. The proper maximum dose is 15 to 20 grains (one to 1.3 gm.) three times a day. An additional effect of the iodide of potassium is the relief it affords to pain. To its efficacy in this direction I think I may add my testimony. It is supposed also to act by increasing secretions, thus thickening the blood. Dr. Balfour also claims that it lowers the blood

pressure by promoting the flow of blood through the arterioles. It may be expected, also, that cases of syphilitic origin will be those especially benefited, but it is said that experience does not confirm such expectation. Evidences of improvement are reduction in the size of the tumor, diminished force of pulsation, and relief of pain. It should be kept up for several months or as long as the patient will submit to it. It is said to be useful more particularly in saccular aneurisms communicating by a small orifice with the aorta. It is doubtful whether it is worth while to subject a patient with large aneurism communicating with the aorta by a large orifice to the inconvenience of such a treatment, and whether it may not be better to advise him to live a life as quiet as possible and to wait the inevitable, while we relieve symptoms as they arise, and remember especially that iodide of potassium is often one of the best remedies for pain.

Other methods of securing coagulation and contraction of the clot are acupuncture, suggested by Velpeau, and consisting in placing an iron wire or needle into the aneurism, with the hope that the blood will coagulate on it, filling the aneurism with horsehair or fine wire for the same purpose. The wire is introduced through a hypodermic needle. Galvano-puncture, suggested by Loretta, perhaps furnishes the most satisfactory results. Two needles are introduced into the sac, and a mild current of electricity passed through them. In this way a combined electrolytic and mechanical effect is obtained. The introduction of astringent substances, as solution of acetate of lead or persulphate of iron, into the aneurism may be mentioned only to be discouraged, since the danger of producing embolism far exceeds the chance of benefit. Ligation of the carotid, subclavian, or both has also been done for aneurism of the aorta with satisfactory results. It is, however, a formidable operation.

No other internal treatment for aneurism other than that suggested, by iodide of potassium, has ever been of any use. As a part of the medical treatment of thoracic aneurism it should be added that, where there is violent action of the heart, cardiac sedatives are sometimes indicated to allay this, in addition, of course, to the enjoined rest. Among these sedatives we include aconite and veratrum viride in extreme cases, also cold to the seat of the swelling and to the cardiac region.

The treatment of *peripheral aneurisms*, as of the popliteal and femoral, is usually relegated to the surgeon, who will treat it by ligation or compression. The injection of ergotin in the vicinity of the aneurism, as suggested by Langenbeck, may be tried. Two to five grains (0.103 to 0.198 gm.) of the aqueous extract dissolved in water or glycerin are injected every couple of days. Tufnell's method is also applied to peripheral aneurisms, for which, indeed, it was originally recommended.

The treatment of peripheral aneurism by compression has long been an acknowledged method for the purpose, and though looked upon as a surgical procedure is as medical as surgical. The method adopted which has been most successful is digital compression, which is exerted

by relays of students or others available for the purpose. The effect is that in the course of forty-eight hours coagulation has taken place and the aneurism is cured. Failing in these measures in the matter of the smaller arteries ligation is practised, but all details of this operation belong to the province of surgery.

SECTION V.

DISEASES OF THE BLOOD AND BLOOD-MAKING ORGANS.

THE ANÆMIAS.

Broadly defined, anæmia means "bad blood." It is further subdivided into *local* and *general.* The former is known also as *ischæmia.* Its special consideration need not detain us long in a text-book of medicine. It is illustrated, however, in the pallor of the fainting person, and in that interesting disease known as Raynaud's disease, which will be considered among diseases of the nervous system.

By *general anæmia* is meant any state of the blood in which there is a diminution in its total bulk, its red corpuscles, its hæmoglobin, its albumin—any one or all of these. The first is the condition which ensues from a large hemorrhage of any kind, as from the bursting of an aneurism, erosion of a blood-vessel, such as sometimes happens in ulcer of the stomach or in tuberculosis of the lung, or from the wounding of a blood-vessel. In all instances, however, where the hemorrhage is not fatal the original bulk of the blood is rapidly restored by the absorption of water and salts from the tissues, while the hæmoglobin and albumin remain deficient until they can be restored by suitable nourishment. The hæmoglobin in blood may be diminished by a reduction in the total number of red corpuscles or in the proportion of coloring matter in each corpuscle.

Anæmias are further divided into *primary* or *essential* and *secondary* anæmias. The former, strictly speaking, should include only those which are the direct result of a defect in the blood-making apparatus, while secondary anæmias are those resulting from the loss of blood, or some one of its important constituents, or from a defective supply of blood-making material.

Among primary anæmias are commonly included chlorosis, pernicious anæmia, leucocythæmia, lymphatic anæmia or Hodgkin's disease, and splenic anæmia. It is not conceded by all observers that pernicious anæmia is the result of a defect in blood-making. In fact, each year adds more evidence to show that the conclusion of Quincke and William Hunter, that it is a hæmolysis or disintegration of the red blood corpuscles in the circulation or certain parts of it, especially in the liver, is the correct one. Especially noteworthy are the more recent studies of J. P.

C. Griffith and C. W. Burr,* favoring such view. As the chain of evidence is not, however, complete, I shall for the present consider it among the essential anæmias.

The secondary anæmias are numerous, including those due to hemorrhage and other drains of various kinds on the economy, inadequate food, and defects in the digestive apparatus; also those due to the action of poisons on the blood,—the toxanæmias, including lead-poisoning and uræmia.

THE PRIMARY OR ESSENTIAL ANÆMIAS.

These include chlorosis, for the present pernicious anæmia, leucocythæmia, lymphatic leucæmia or Hodgkin's disease, and splenic anæmia.

I. CHLOROSIS.

Synonyms.—*Morbus virgineus; Green Sickness; Chloræmia; Chloranæmia.*

Definition.—An essential anæmia most frequently met in young women, characterized by a very marked relative reduction in the hæmoglobin of the blood.

Etiology.—As stated in the definition, it is a disease of women, and especially of young women. Yet its occurrence is not impossible in men of womanish habit and occupation, among whom Hermann Eichhorst especially instances tailors. Moreover, while it is especially a disease of young women from about the age of puberty to twenty-four years, it is also possible in those who are older and younger. In the former it is known as *chlorosis tarda*, and as such is met in women between thirty and forty. Rather more frequent is its occurrence in children under puberty. Niemeyer held that girls who menstruated at thirteen or fourteen, in whom there was, as yet, no development of pubis or breasts, almost invariably become chlorotic. The disease occurs the world over, and is apt to be recurrent in the same individual. It is more common in blondes than brunettes, in the weak and delicate rather than the strong and vigorous. Yet this general truth is not without exceptions.

Among predisposing causes are overwork, especially in closely confined and illy-ventilated rooms, insufficient nourishment, exhausting drains, such as prolonged lactation and profuse menstruation. Menstrual derangement is, however, also a consequence as well as a cause. Sustained or repeated emotion, especially such as arises from sexual excitement and masturbation, is a cause.

The frequent association of constipation with chlorosis led Sir Andrew Clark to suggest that it might really be a copræmia or poisoned blood due to the absorption from the large bowel of poisons of the nature of ptomaïnes and leucomaïnes. Such poisons may readily interfere with the proper development of the hæmoglobin of the blood disc without in a great degree causing its destruction. This view explains what seems

* "Pathology of Pernicious Anæmia," Transactions of the Association of American Physicians, Vol. VI, 1891.

to me a closer relation between chlorosis and pernicious anæmia than has commonly been admitted, a relation consistent with the newer etiology of pernicious anæmia as well as with features in its clinical course and with the results of treatment, to which attention will be called when considering the latter affection.

Morbid Anatomy.—Other than the changes in the blood, to be considered under symptoms, there is no essential morbid anatomy to chlorosis. Many years ago Virchow pointed out an imperfect development of the circulatory apparatus as more or less characteristic,—that the heart was small, the right ventricle sometimes dilated, the aorta and its larger branches poorly developed and thin-walled. Such a state of affairs, when present, is probably an accidental coincidence. There is no enlargement of the spleen or lymphatic glands. Imperfect development of the uterus and other genitalia has been noted. The rarity of fatal termination in chlorosis may limit our knowledge of the morbid anatomy, uncertain at best.

Symptoms.—Of these the blood changes may be regarded as fundamental, though not absolutely constant. These consist in a decided reduction in the hæmoglobin with a moderate oligocythæmia, or reduction in the number of red corpuscles. Thus the hæmoglobin value of each red disc is diminished. The usual range may be put at from 3,500,000 to little less than normal. Thus Thayer in 63 consecutive cases in Osler's clinic found the average 4,096,544, or over 80 per cent., and Lembeck found the maximum in one of 15 cases to be but 3,600,000. In a few instances, however, in cases of acknowledged chlorosis, there has been found a more decided reduction in the eruthrocytes. One has been reported as low as 1,190,000 per cubic millimeter.

The hæmoglobin, on the other hand, is much reduced, the average of Thayer's cases referred to being 42.3 per cent., which may be regarded as a fair average. This disproportionate fall in the hæmoglobin, while not invariable, remains, however, a tolerably constant feature, producing sometimes a recognizable paleness of color when the blood is seen *en masse*. Along with the lowering of hæmoglobin, as would be expected, since it is a constituent of the hæmoglobin, the iron of the blood falls. An increase of alkalescence, announced by Græber * as a constant symptom, has not been found by Kraus † in his more exact methods of testing.

As to remaining changes, the red corpuscles may be altered in shape to a moderate extent, constituting a small degree of *poikilocytosis*, or they may be larger than in health, when they are known as *megalocytes*. More frequent is an undue reduction in size of the corpuscles—a *microcytosis*. The red discs are sometimes appreciably paler than in health. A very *slight* degree of *leucocytosis* may be rarely present, an average of 8467 in Thayer's counts as contrasted with a mean normal of 6000 per cubic millimeter, while the *blood-plaques* in severe cases may also be increased.

---

* "*Zur klin. Diagnostik d. Blutkrankheiten.*" Leipzic, 1888.

† *Zeitschrift f. Heilkunde*, Bd. ii.

While the blood alterations in chlorosis are scarcely distinctive enough to be considered diagnostic, the other symptoms help greatly to a correct conclusion. The patient is almost invariably a girl, generally between sixteen and twenty, who, although she may have been overworked, does not seem badly nourished; certainly she is not emaciated.

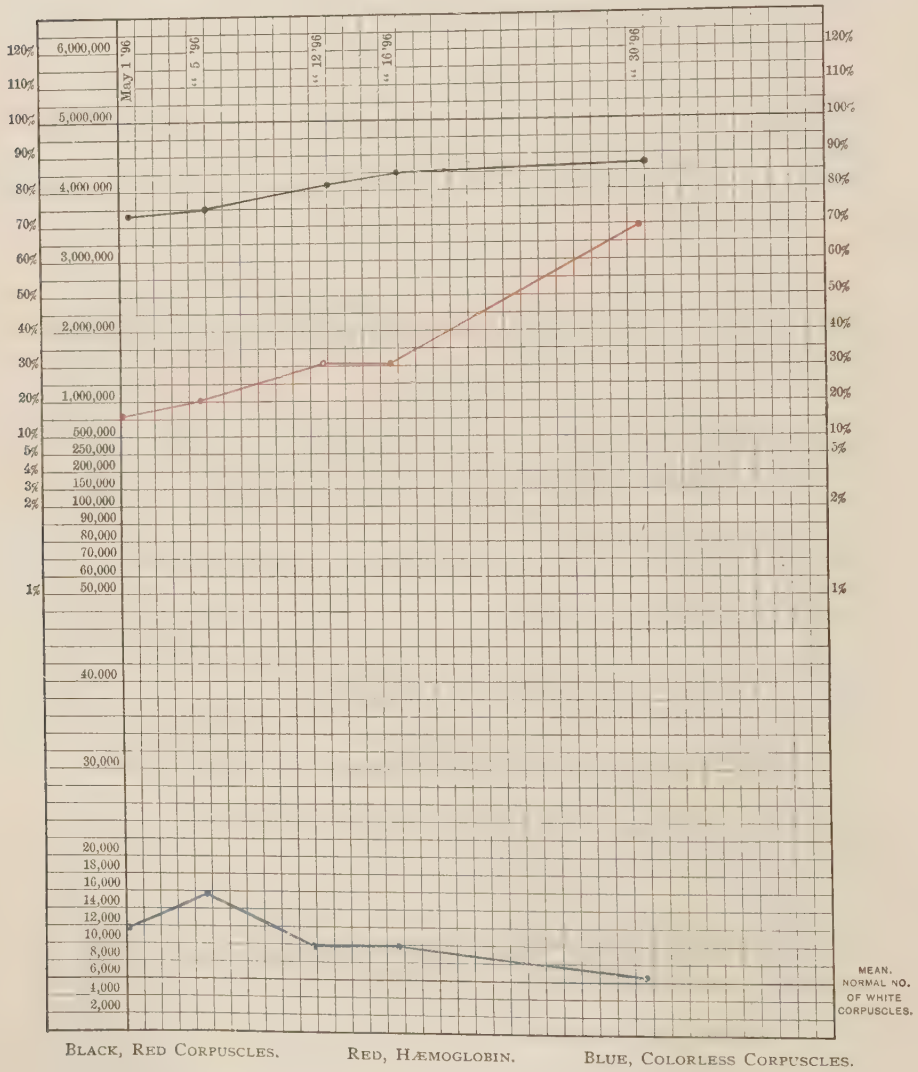


FIG. 51.—BLOOD IN CHLOROSIS.

She is, however, characterized by a peculiar pallor, often exhibiting a yellowish-green tinge, extending to the lips and especially the mucous membranes, and which is responsible for one of the names of the affection, *green sickness*. She is extremely weak, especially on exertion, and

short of breath. She is subject to vertigo, palpitation of the heart, and even irregularity of the heart's action. Physical examination will sometimes discover functional cardiac murmurs; also a systolic murmur at the apex, ascribed by Balfour to a relative insufficiency of the mitral valve due to dilatation of the left ventricle. Rarely a compensatory hypertrophy of the left ventricle has been noted, but never actual valvular disease. Sometimes a *bruit de diable*, or humming-top murmur, may be heard over the right jugular. Epigastric pain is also a symptom at times. It must not be forgotten that a chlorosis late in life, or a *chlorosis tarda*, does sometimes occur.

Diagnosis.—The diagnosis is based chiefly upon the age and sex of the patient, the peculiar greenish-yellow color, the paleness of the lips, and the decidedly diminished hæmoglobin, unaccompanied, as a rule, with a proportionate reduction in the number of eruthrocytes. The same lost normal ratio between the hæmoglobin and the corpuscles is also a characteristic of lead-poisoning, which has, however, superadded its own characteristic symptoms, and is almost restricted to adult males.

The epigastric pain mentioned as occurring in chlorosis resembles that more common in ulcer of the stomach. The anæmia which so constantly attends ulcer of the stomach, often in a high degree, is, however, different from that of chlorosis, there being a proportionate decline in the eruthrocytes and their coloring matter.

A not infrequent error of diagnosis in connection with chlorosis is the mistaking it for a "decline," a pulmonary consumption, which it resembles in the pallor, the feebleness, and shortness of breath of the patient. The absence of cough and of the physical signs of consumption excludes that disease. On the other hand, evidences of tuberculosis should always be sought where the symptoms of chlorosis prevail, while latent cancer is also sometimes responsible for similar symptoms.

Prognosis.—The prognosis is nearly always favorable when the disease is recognized and the proper treatment afforded. There are few results more satisfactory in therapeutics than those of a properly-treated case of chlorosis. Time is, however, required, and too rapid a cure must not be promised, several months and even longer being sometimes required. The question whether a chlorosis will be transformed into that more serious variety of anæmia known as pernicious anæmia has been raised. This seems not impossible. If the view of Sir Andrew Clark be accepted, that chlorosis may result from the absorption of poisonous substances from the larger bowel, and if pernicious anæmia be due to the absorption of more intense poisons from the small intestine, the difference is only one of degree. Both are characterized by defects in the cellular constituents of the blood. In the one, chlorosis, the coloring matter is chiefly wanting, although associated with this is usually found a small degree of morphological defect. In pernicious anæmia both cell shapes and coloring matter are defective. In both diseases the oxygen-carrying office of the blood is interfered with, and thus important vital processes embarrassed, the total suspension of which must be fatal.

Treatment.—The treatment is preëminently by iron, and it matters not very much what preparation is used. The carbonate of iron, in the

shape of Blaud's pill, made by a double decomposition between the carbonate of potassium and the sulphate of iron, is at this day very popular, one to three grains (0.06 to 0.2 gm.) being given at a dose three times a day. But the tincture of the chloride of iron or reduced iron or one of the vegetable salts may be given. I have many times said that iron is given in too large doses in the majority of cases for which it is prescribed. Most of it is unabsorbed, and therefore wasted. Nay, worse, that which is unabsorbed locks up the intestinal secretions by its astringency, produces headache, and makes the patient otherwise uncomfortable. But chlorosis is one of the few diseases in which large doses of iron are well borne. The reason is plain. It is the iron-holding constituent of the blood which is wanting, and the iron is needed to replace it. The blood is, as it were, hungry for it. Next to iron come arsenic. The efficiency of iron is greatly aided by union with arsenic, which should be given in increasing doses, but short of toxic effect.

But to give these drugs is not alone sufficient. Rest in bed, at first continuous, is imperative to secure a rapid result, and this must be associated with an abundance of good food. Daily massage, except during menstruation, is also a useful adjuvant. There is no condition in which the so-called "rest cure" is more efficient than in chlorosis. With a return of color to the lips, or, better, with the growing increase in the hæmoglobin as measured by the hæmoglobinometer, the patient should be permitted to be out of bed at first a half-hour to an hour only, but this should be gradually increased until she is up most of the day. For a long time, however, fatigue should be avoided. To those who can afford it a residence at the sea-side materially aids convalescence. Indeed, I know of no condition so rapidly improved at the proper time by sea-air as chlorosis. To the poor a well-regulated hospital treatment is a boon for which there is scarcely a substitute.

II.—PROGRESSIVE PERNICIOUS ANÆMIA.

SYNONYM.—*Pernicious Anæmia.*

A second variety of essential anæmia is *pernicious* or idiopathic anæmia, originally described by Addison in 1855 in his celebrated paper on "Diseases of the Supra-Renal Capsules." Interest in the subject was revived by Biermer in 1868, and since then it has been thoroughly studied anatomically and clinically. It is, however, still the least understood of all the anæmias. Fortunately, it is a rare disease. It may be briefly defined as an anæmia in which the red corpuscles have been destroyed and reduced in number, associated also with a lowering in the hæmoglobin as usually measured; though the ratio of the latter to the remaining corpuscles is one of excess. The blood also includes red discs which are disintegrating, distorted, large, and many nucleated forms.

Etiology.—The etiology of pernicious anæmia is very obscure. Pregnancy seems to be in some way responsible for a certain number of cases of true pernicious anæmia, the first of which were described by Gusserow. A puerperal form was first described by Walter Channing

in 1842. It has also followed lactation. Other causes are cited, such as atrophy of the stomach, profound and long-continued gastro-intestinal disease, and intestinal parasites, especially the *anchylostomum duodenale* and *bothryocephalus latus*, which undoubtedly produce symptoms clinically indistinguishable from the general anæmia which Addison characterized as "occurring without any discoverable cause whatever,—cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease." I have already expressed my partiality toward the view of Quincke and others who ascribe the state of the blood to a hæmolysis, in proof of which they point to the enormous accumulation of iron in the liver noted by Quincke in 1876, and confirmed by Rosenstein in 1877. To these William Hunter added a pathological increase of the urobilin in the urine. In a recent noteworthy paper Griffith and Burr take the same view.* Such a hæmolysis is most satisfactorily explained on the supposition of absorption from the intestine or elsewhere of poisonous products engendered under any of the circumstances named. Among these may be included the products of imperfect digestion such as may be expected to arise where there is atrophy of the gastric tubules.

The disease affects mostly those past middle age, but children also have it, and Griffith mentions ten cases occurring under twelve. It is also more frequent in males.

Symptoms.—The approach of the symptoms of pernicious anæmia is most insidious, beginning with a gradual progressive weakness. What is first interpreted as a causeless weariness or languor grows slowly into an extreme debility with faintness on the slightest exertion, and thence into a state of thorough muscular weakness, which ultimately prostrates the patient, and he is too weak to rise from bed. To this succeeds a state of mental hebetude and bodily torpor. Yet there is no emaciation. The body-bulk is well preserved. The skin acquires gradually a yellowish hue, and sometimes an actual jaundice, whence the disease has been mistaken for the slower form of yellow atrophy of the liver, a mistake not altogether unsustained by other symptoms. In fact, the jaundice is similarly caused. It is probably a hæmatogenous jaundice, a matter of blood disintegration, although it has also been ascribed to defective cell action on the part of the liver. The mucous membranes, on the other hand, are blanched, as may be noted in the lips, gums, and mouth.

Cardio-vascular symptoms are especially conspicuous in progressive pernicious anæmia. Hæmic murmurs, visibly pulsating and throbbing arteries, even pulsating veins, have been noticed. The large but soft, jerky pulse was mentioned by Addison. The capillary pulse is also frequently seen, and hemorrhages, cutaneous and retinal, occur.

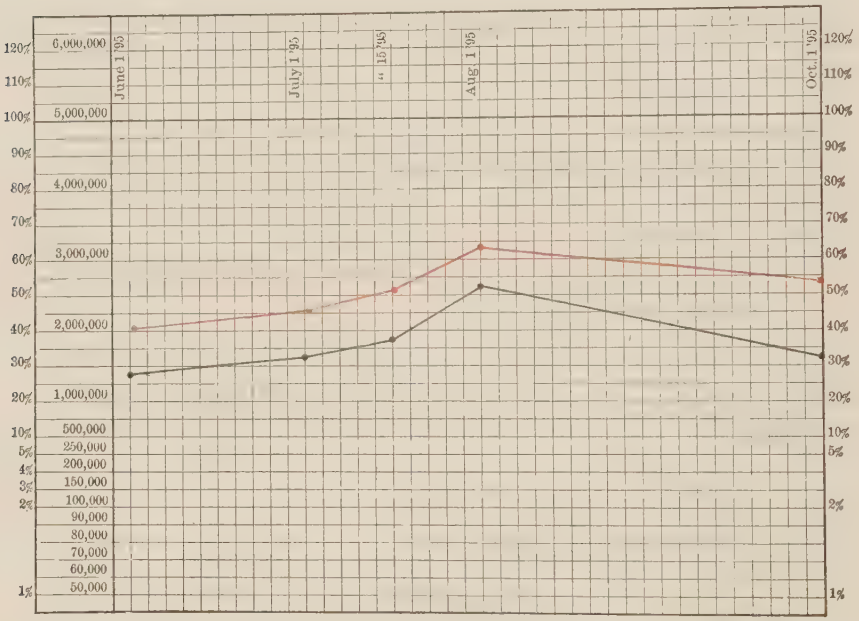
Digestive derangements form an important part of the symptomatology of pernicious anæmia. Indisposition to take food, or rather a disgust for food, nausea, vomiting, and diarrhœa are often troublesome symptoms. Moderate elevation of temperature, irregular and intermittent, is

* *Loc. cit.*

also noted, while nervous symptoms, including numbness, languor, and even paralysis, are sometimes present.

The urine exhibits no constant changes, being sometimes pale and sometimes dark-hued. The dark color is ascribed by Mott and Hunter to an excess of urobilin.

Blood Changes.—The changes in the blood are more distinctive than in chlorosis, although it is true also that there is no single constant characteristic feature. The most constant is, however, a very decided oligocythæmia without a corresponding reduction in the hæmoglobin, although the hæmoglobin, *in toto*, is much reduced. Rarely it has been found increased. Quincke found as few as 143,000 corpuscles in a cubic milli-



BLACK, RED CORPUSCLES. RED, HÆMOGLOBIN.

FIG. 52.—PERNICIOUS ANÆMIA.

meter of blood, while it is not uncommon to find them less than half a million. Frederick P. Henry found 315,000 a few hours before death, and Laache 360,000. The inevitable conclusion from such a state of affairs is that either the hæmoglobin value of each corpuscle must be increased, or there is a hæmoglobinæmia which has its seat in the plasma. This latter view Silbermann has adopted, because he has been able to produce by the administration of blood-corpuscle dissolving substances, as pyrogallol, to animals a complex of symptoms like those of pernicious anæmia. If pernicious anæmia be a hæmolysis, as seems likely, rather than a defective hæmatogenesis, we would expect such a hæmoglobinæmia to result.

A further striking peculiarity in the blood of pernicious anæmia is

an increase in the size of the red corpuscles. They become megalocytes, from ten to 15 micro-millimeters in diameter, as compared with a normal of from 6.5 to 9.4. The majority may be so enlarged. On the other hand, there are also microcytes and poikilocytes—corpuscles characterized by great irregularity in shape, being crescentic, conical, and otherwise distorted. While these irregular shapes were first demonstrated in connection with pernicious anæmia, and although they are more or less characteristic, cases of the disease have been described by Grainger Stewart, Lepine, Hermann Müller, and others, in which poikilocytosis was altogether absent. Megalocytosis is not therefore a pathognomonic feature.

There are also sometimes found in the blood numerous minute highly-colored spherical bodies, called *Eichhorst's corpuscles.* Eichhorst regarded them as pathognomonic, but they, too, are sometimes absent. When present they contribute to the hæmoglobin in the blood, but as they are not included in the blood-count they get no credit for their effect. The relative excess of the hæmoglobin may, in a measure, be thus accounted for. *Nucleated red corpuscles* are a constant constituent of the blood of pernicious anæmia, and have also been regarded by their discoverer, Ehrlich, as almost pathognomonic. Two kinds are found: first, the small, normal-sized corpuscle with its deeply-stained nucleus, and certain large forms with pale nuclei. Blood-plaques are either absent or very scanty. Leucocytes are usually slightly diminished in number, while there is a tendency to an increase of the mononuclear white cells as compared with health. F. P. Henry * has called attention to a fact which condenses the peculiarities of the blood changes, by saying that in this disease the red corpuscles "approach those of the lower animals in many, if not all, of their chief characteristics, namely, in their number, their size, their shape, and the amount of hæmoglobin they carry."

Morbid Anatomy.—Various tissues have been studied in the effort to find a morbid anatomy for pernicious anæmia. In the absence of lymphatic or splenic enlargement, the marrow of bones has claimed close study. H. C. Wood described the red condition of the marrow of long bones in 1871. In October, 1875, William Pepper † described qualities of the medulla of the sternum and radius from a case of pernicious anæmia, consisting, first, in a paleness as compared with health, and, second, in being made up for the most part of pale granular cells with single nuclei. Just a year later Cohnheim ‡ described changes in the medulla, consisting (1) in the absence of fat-cells, (2) the presence of colorless cells closely resembling lymph-cells,—cells of epithelial habitus and various sizes, containing one and two nuclei; (3) a few multinuclear giant-cells, and (4) a very few blood-corpuscles holding marrow cells; (5) an equal proportion of colored elements, among which the biconcave red discs were in a decided minority; more numerous were

* "Anæmia," Philadelphia, 1887.

† "Progressive Pernicious Anæmia," American Journal of the Medical Sciences, October, 1875.

‡ *Virchow's Archiv*, October, 1876.

the spherical non-nucleated corpuscles of different sizes, while most numerous were the *red nucleated cells.*

In 1877 Pepper and the writer published a note in *Virchow's Archiv* * containing the results of the study by the latter of the marrow from the shaft of a femur from a case of pernicious anæmia. This marrow resembled a fresh blood-clot in appearance, but on examination was found to be made up of about equal proportions of red blood discs and of granular cells of epithelioid character, for the most part larger than white blood cells, but otherwise similar. The largest were $\frac{1}{1000}$ inch (0.0254 millimeter) in diameter, the smallest $\frac{1}{3000}$ inch (0.0084 millimeter). Most of these contained a single comparatively large nucleus. A few contained two nuclei. Some contained granular fat particles, while others were completely filled with granular fat (compound granule-cells), but there were no true adipose vesicles. About the same time I also examined the medulla from a clavicle and radius sent from Montreal by William Osler. The mass was dark red and diffluent, slightly decomposed. There was a relatively small number of red corpuscles, but a very large number of granular cells resembling colorless blood corpuscles, but of larger average size, ranging from $\frac{1}{3000}$ inch (0.0084 millimeter) to $\frac{1}{1500}$ inch (0.016 millimeter). While many of them were nucleated and epithelioid, containing one, two, and rarely even more nuclei, in many no nuclei could be brought out even by aid of acetic acid. In addition to these two sets of cells were a number of *nucleated colored* cells, usually $\frac{1}{1500}$ inch (0.016 millimeter) in diameter and oval in shape, containing from two to five nuclei. These were presumably the red nucleated cells described by Cohnheim.† Finally there were found free oil-globules, granular fatty cells (compound granular), fat crystals probably from decomposition, and undetermined granular matter probably from the same source.

These reports do not differ essentially, and although the appearances described are not identical, they are sufficiently constant to justify their association as more than accidental. *Summed* up they amount to this: marrow dark red, consistence less soft, fat-vesicles absent, specific cellular elements (lymphoid cells) increased, numerous nucleated red corpuscles present, especially the larger forms, the gigantoblasts of Ehrlich. These appearances may be interpreted as due to an effort of the blood-making apparatus to reproduce the disintegrated eruthrocytes.

The deposition of iron in the liver-cells has already been alluded to. It is found in the outer and middle zones of the lobules, and may be so distributed as to outline the bile capillaries. It is characteristic. The liver itself is often fatty and is sometimes enlarged. The iron is in like manner sometimes increased in the kidney, but not in the spleen, and these organs are not otherwise essentially changed. The spleen also has its iron pigment increased and has been found reduced in size.

The heart-muscle is fatty, while the other muscles are unusually red.

---

* "*Die Betheiligung des Knochenmarkes bei Perniciöser Anæmia*," *Virchow's Archiv*, 1877, LXXI, 118–126.

† This was before the day of differential staining.

Other morbid changes are described, but they cannot be regarded as essential. Such are changes in the ganglion-cells of the sympathetic * and sclerosis of the posterior columns of the cord, first studied by Lichtheim. While the association of the changes in the posterior columns are so constant that they cannot be regarded as accidental, experimental studies by Burr and Griffith intended to determine this relation to pernicious anæmia resulted in nothing definite. Osler and Henry describe a complete atrophy of the secreting tubules of the stomach in one case studied jointly by them.

Diagnosis.—The diagnosis of pernicious anæmia may be uncertain at first, but the true nature of the disease soon declares itself. The intense anæmia, extreme weakness, digestive derangements, and cardiovascular symptoms, in connection with a blood-count of 2,000,000 or below, with a relative increase, or at least no proportionate diminution, in the hæmoglobin, and an admixture of megalocytes, microcytes, and poikilocytes, point to a condition scarcely mistakable. The large forms of nucleated red corpuscles have been regarded as characteristic, but are also found in leucæmia.

Prognosis.—The prognosis is to-day regarded as less unfavorable than a few years ago, since recent experience has developed the fact that temporary improvement is not uncommon, and it is said that recovery sometimes takes place. Still, Addison's original prognosis, of a termination sooner or later fatal, is not often a wrong one.

Treatment.—Treatment of this form of anæmia is, however, not fruitless. The same measures which are almost a specific for chlorosis are not without effect in pernicious anæmia. Accordingly, arsenic, to a less degree iron, good food, and favorable hygienic surroundings are to be adopted. The arsenic treatment has been followed by results which justify the words "temporary cure," and it is said that permanent cure has followed. Such temporary cures have covered a period of three years. The best preparation appears to be Fowler's solution in gradually increasing doses until 20 and even 30 minims (1.3 to two c.c.) are reached, and this three times a day. It should be continued for a long time, for weeks or months, with intermissions of a few days if unpleasant results appear, to be again resumed. Arsenic is not, as some would regard it, a specific for pernicious anæmia, but the results of its use are often surprisingly gratifying. The relation between chlorosis and pernicious anæmia, already referred to, is sustained by therapeutic results. Certain cases of chlorosis very closely resemble pernicious anæmia, and apparently pass over into pernicious anæmia, especially when not arrested by treatment. The arsenic administered as directed is wonderfully well borne, nausea and vomiting being rare. Rest in bed is indispensable, but should be supplemented with massage if possible. Food should be in easily assimilable shape, such as beef-juice, beef-peptonoids, and peptonized milk.

Transfusion of blood and of milk, which seemed at one time to give promise of favorable results, has been discontinued.

* *Congress für Innere Medicin*, 1887.

III.—LEUCÆMIA.

Definition.—A disease characterized by an enormous increase in the colorless corpuscles of the blood, with hypertrophic changes in the spleen, lymphatic glands, or bone marrow, one or more of each.

It has been called *leucocythæmia* as well as *leucæmia*, the former of these words meaning white-cell blood, the latter simply white blood. From the etymological and histological standpoint, leucocythæmia, suggested by Hughes-Bennett, is the more accurate term, but Virchow's term, leucæmia, has become the one in common use.

Historical.—The history of the development of our knowledge of leucæmia possesses unusual interest. The older observers spoke of a purulent blood, ascribed to an inflammation of this tissue, while Piorry and Rokitansky spoke of a hæmatitis as the cause of a literal pyæmia. Dr. Craigie undoubtedly saw a case of the disease in Edinburgh in 1841. He did not, however, publish the case until John Hughes Bennett published his on October 1, 1845, in the Edinburgh Monthly Journal of Medicine. Both Craigie and Bennett noted enlargement of the spleen. Prof. Rudolph Virchow published his case in Froriep's *Notizen* in the second or third week of November, 1845. The matter of priority between Bennett and Virchow has given rise to much discussion. There can be no doubt that Bennett's case was published first; also that he declared it unconnected with inflammation of any of the tissues, and especially unconnected with phlebitis, and that he attributed the condition to the development of white corpuscles in the blood. Craigie, on the other hand, ascribed it to the absorption of pus from an inflammatory lesion either in the mesenteric veins or the spleen. Virchow confirmed all the observations made by Bennett and published new cases, especially one of great importance, in which there was *enlargement of the lymphatic glands without enlargement of the spleen.* Virchow also said the blood changes consisted essentially in an increase of the colorless cells of the blood, and that these cells originated in the lymphatic glands. Virchow also suggested the name *leucæmia*, while Bennett did not suggest that of *leucocythæmia* until 1851 in a series of papers, and again in 1852 in a separate work. It appears to me that Bennett is clearly entitled to priority, and was the first to interpret the condition as an increase in the colorless corpuscles of the blood, though he speaks of it as *pus*. Virchow's added point was ascribing the formation of the cells to the lymphatic glands.

Vogel first recognized a case during life in 1849. Virchow described two forms of the disease in his "Cellular Pathology," in one of which the smaller forms of leucocytes predominated, and in which there was marked involvement of the lymph glands. In the second the larger white blood cells predominated, and there was marked enlargement of the spleen. It was many years later that Neumann described a case in which the *bone marrow* was markedly altered. Pure splenic and pure myelogenous forms scarcely exist, though a case or two of the latter is described. The pure *lymphatic form* does occur, but even this is rare. Most common is the mixed *lieno-myelogenous.*

The extremely rapid course of certain cases of leucæmia justifies its division into an acute and a chronic form. An instance of the former was a fatal case reported by Ebstein, in which the whole course of the disease, including a prodromal stage, was but six weeks. Similar cases are reported, eleven by Fraenkel.* The duration of the chronic form may extend over years. Leucæmic women have had repeated pregnancies and have borne children at term.

Etiology.—Nothing definite is known of the cause of leucæmia. It occurs in all countries, in both sexes, and at all ages, although more common in middle life and more frequent in males. The eighth week and seventieth year have found cases. It is sometimes hereditary, but leucæmic women have borne non-leucæmic children. Malaria has been assigned as a cause, and certainly its association with this disease has been seemingly more than accidentally frequent. To a less degree this is true of syphilis. Pregnancy is said to favor it. It is said to have followed a blow or injury and to have been found in the lower animals.

The idea of the infectious origin of leucæmia, advanced by Klebs and supported by observations of Osterwold, Roux, Byrom Bramwell, Pawlowsky, Kelsch, Vaillard, and others, seems well founded, but no single micro-organism has been found associated. A case has, however, been reported where an attendant on a case of leucæmia took the disease and died. The frequent association of leucæmia with stomatitis and intestinal ulceration has been pointed out by Hunterberger.

Morbid Anatomy.—Leucæmia has a definite morbid anatomy, consisting in alterations in the blood and in the hæmogenic apparatus, including the spleen, the lymphatic glands, and the marrow of bones, and it is called, accordingly, splenic, lymphatic, myelogenic, while combined or mixed forms are indicated by suitable compound terms, such as lieno-myelogenous. Most leucæmias are mixed.

In the first place, the *spleen* is almost always enlarged. It may be adherent to the abdominal walls, the diaphragm, stomach, or other viscera. The splenic changes exhibit three stages in their development. In the first the spleen is simply hyperæmic, soft, and swollen, sometimes even ruptured. The Malpighian bodies share in the hyperæmia and may be slightly enlarged, but are overshadowed by the swollen pulp. In the second stage hyperplastic changes make their appearance in the Malpighian bodies, and as these grow the pulp is intruded upon. They may reach such size as to be recognized by the naked eye as spherical grey nodules one to three lines in diameter, or they may be elongated or forked, following the course of the blood-vessels. The third stage furnishes the granitic spleen, in which white dots are separated by dark streaks representing the destroyed pulp, pigmented by the disintegrated blood. The spleen is now hard and is cut with resistance. Its size may be enormous and the organ weigh from two to 18 pounds (one to nine kilos).

The *lymphatic enlargement* is a true hyperplasia. Not only do the glands enlarge, but new foci of lymphatic tissue appear in various organs, as the liver and kidneys. These are regarded by some as simple extravasa-

* *Wiener Klin. Wochenschrift*, 1894.

tions of leucæmic blood from the capillaries. All the more prominent groups may share in the enlargement,—the cervical, axillary, inguinal, and perineal glands. The individual glands remain, however, soft. The lymphatic follicles in the tonsils and in the tongue, pharynx, and mouth may enlarge. This is also occasionally the case with the solitary glands of the intestine and the agminated glands of Peyer.

The *marrow-changes* may be described in a word as reversion to the embryonal type of medullary tissue. The fat of the adult marrow has disappeared, and a mass of lymph cells mingled with nucleated red corpuscles in all stages of development takes its place. The marrow is often *pyoid*. The lymph cells include numerous large mononuclear cells, many in the act of division, also multinuclear leucocytes. There are also numerous marrow cells or myelocytes, including both eosinophiles and myelocytes like those found in the blood.

The *liver* is often enlarged, and, according to von Jaksch,* *pari passu* with the spleen, and it has this further peculiarity, that its edges are rounded, while in what he describes as *pseudoleucæmia infantum* the edges are sharp and the enlargement does not go hand in hand with that of the spleen. The liver is also at times infiltrated with leucæmic patches and nodules not unlike miliary tubercles. The same is occasionally true of the kidney.

The thymus gland has been found enlarged in some cases of acute lymphatic leucæmia, and even the skin, stomach, and gastro-splenic omentum have been the seat of growths presumably lymphatic. In fact, there is no situation in which such growths may not make their appearance. The possibility of their being blood extravasations, white in consequence of the large proportion of white cells, is always to be remembered.

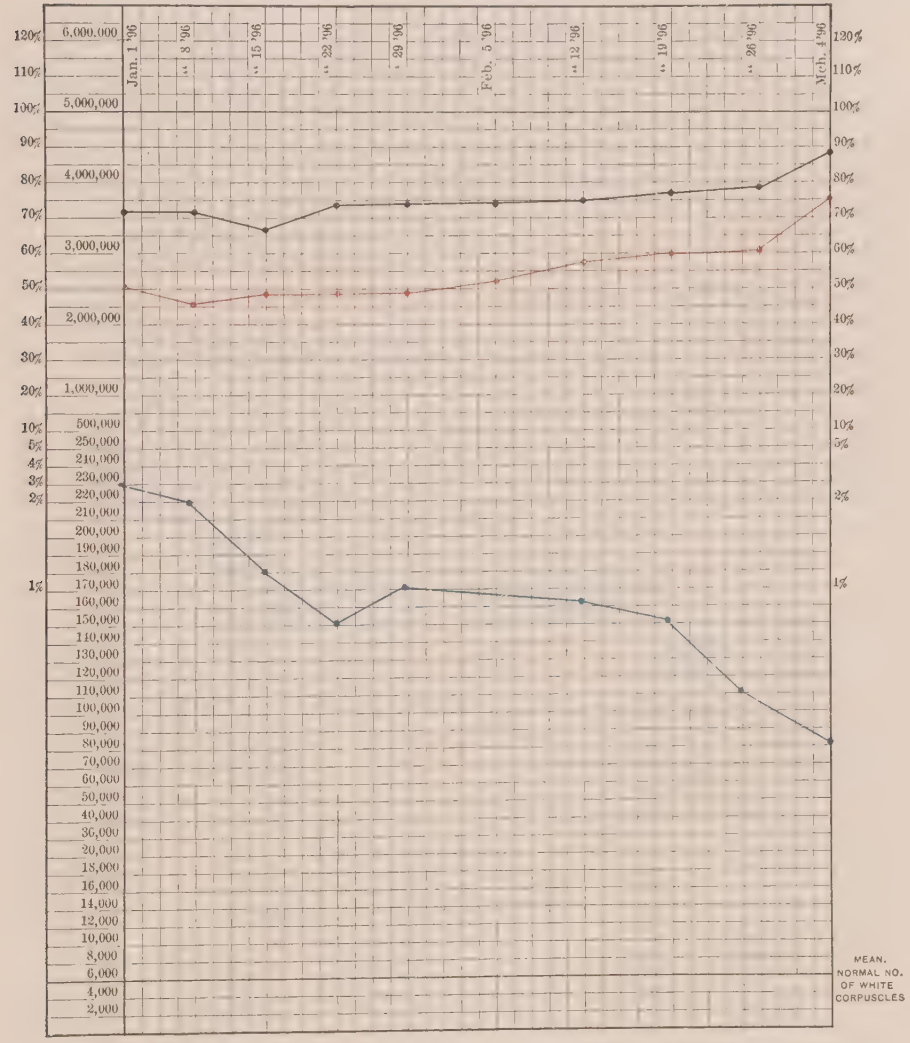
The lungs and heart alone seem free from encroachment by the lymphatic tissue. The heart may, however, be dislocated by a large spleen.

The alterations in the *blood* constitute really a part of the morbid anatomy of leucæmia, but are commonly treated under the head of symptomatology, where I, too, will consider them. An increase in the mass of the blood may, however, here be mentioned. The heart and vessels are commonly found gorged with blood, usually coagulated and sometimes whitish or yellow in color.

Symptoms.—The early symptoms of leucæmia are precisely those of the other anæmias, insidious onset, pallor, rapid breathing amounting to dyspnœa on exertion, weakness and faintness, headache, loss of appetite, and indigestion. The last two symptoms may precede all others. Emaciation is ultimately added. Moderate fever with rapid pulse is also present in the majority of cases, the temperature sometimes reaching 103° F. (39.4° C.). Headache more or less continuous is also a symptom. Swelling of the abdomen from an enlarged spleen may be the first symptom noted, as may also lymphatic gland enlargements. In a case recently under my advice the first intimation of the presence of a large

* See cases reported by J. Chalmers Cameron and Saenger, Sajous' Annual for 1891, E.

spleen was an attack of circumscribed peritonitis, favored, doubtless, by the presence of the splenic tumor and ascribed to exposure to cool air while perspiring. Hemorrhages from the nose and stomach are common, and dropsical swelling toward the close. Hemorrhages from



BLACK, RED CORPUSCLES. RED, HÆMOGLOBIN. BLUE, COLORLESS CORPUSCLES.

FIG. 53.—THE BLOOD IN LEUCÆMIA.

the stomach are sometimes fatal. Recently Thomas Oliver reported a case terminating fatally by sudden post-peritoneal hemorrhage.* Hæmatemesis may be an early, an almost initiatory, fatal symptom.

* Sajous' Annual for 1890, Vol. II, E, p. 12.

Purpura hæmorrhagica sometimes presents itself as a manifestation of the same tendency, as may also cerebral hemorrhage, producing coma. Priapism is an occasional symptom. It is sometimes persistent, and in a case of Edes was the first noted.

The urine often contains a little albumin, is high-colored and scanty, and deposits a copious sediment of uric acid.

Blood Changes.—The blood exhibits a most marked and diagnostic change, consisting in an *enormous leucocytosis.* Normal blood contains about 6000 colorless corpuscles to the cubic millimeter, which, with the red discs at 4,500,000, makes the proportion one to 750; or with these at 5,000,000, one to 833. There is a physiological leucocytosis which, after a full meal, may reach one to 150, and even one to 100. In leucæmia, however, there may be one to 50, to 25, to ten, to three, to two, or the leucocytes may equal and in rare cases even exceed the red discs. The maximum proportion of colorless corpuscles impresses decidedly the color of the blood *en masse*, making it pink, or even the color of chocolate and milk, while aggregations of leucocytes may produce white streaks, and well justify the notion of the older pathologists that there was pus in the blood,—a suppuration of the blood, as they called it.

There is also a reduction in the total number of blood cells, including white and colored, the quantity per cubic millimeter being sometimes reduced to between 2,000,000 and 3,000,000. This reduction may be even more positive, as in a case reported by Suchannek,* in which there were 301,600 red discs and 306,100 colorless corpuscles.

Numerous *nucleated red discs* are sometimes present, and occasionally also poikilocytes. The hæmoglobin falls below the normal proportion, so that the value of each disc is lowered. The blood plaques may be slightly increased.

An early discovery in the study of leucæmia were certain transparent octahedral crystals, known as Charcot's crystals, which form in blood which has been kept for some time on slides. Neumann referred these crystals to the bone-marrow, but von Jaksch says they are the same as those sometimes found in the expectoration and in fæces and in seminal fluid. There is reason to belive they are decomposition products, and that they hold no essential relation to leucæmia. That the alkalinity of the blood is sometimes diminished is true; that it is ever replaced by acidity is not true, as was at one time held. Its specific gravity is lowered to 1030 to 1050.

The *further* minute study of the *blood changes* in leucæmia requires a review of the histological characters of the colorless corpuscles in normal blood. These, through the studies of Ehrlich and others, have assumed a much greater complexity than heretofore. It occurred to Ehrlich that important differences in white corpuscles could be differentiated by staining fluids, and, putting into practice this conception, he was enabled to discover three principal varieties, as follows :—

(1) Lymphogenous elements arising in lymphatic glands. (*a*) Small lymphocytes. (*b*) Large lymphocytes.

* Sajous' Annual for 1891, Vol. II, E.

(2) Myelogenous elements arising in marrow, including eosinophile cells. (More recent studies go to show that myelocytes or marrow cells are rarely eosinophilic, and that the eosinophile granulation has no special relation to the marrow cells.)

(3) Elements undetermined as to source, supposed by Ehrlich to arise in the spleen and marrow or in marrow only, but according to more recent views starting in the lymphocyte and undergoing further development in the spleen. These include (*a*) large mononuclear cells; (*b*) transitional forms; (*c*) polynuclear cells.

(1) (*a*) The small lymphocytes are somewhat smaller than the red blood corpuscle (A), and contain a large, round, deeply-staining nucleus, surrounded by a very scanty and almost invisible layer of homogeneous protoplasm (B). (*b*) The large lymphocytes are a further development of the first, and differ from them only in possessing a larger and easily recognizable rim of protoplasm (C). The lymphocytes are said to make up from 15 to 30 per cent. of the whole number of leucocytes in the blood.

(2) The eosinophile cells, formerly regarded by Ehrlich as originating in the bone-marrow, are only sparsely seen in normal blood. They are characterized by coarse granulation, and contain one, two, and, more rarely, three nuclei. The majority of eosinophile cells are polynuclear. They form ½ to eight per cent. of the normal white corpuscles. They take the α- or eosinophile granulation stain described below. (See Fig. 54, D.)

(3) Undetermined elements originating, according to Ehrlich, in spleen and marrow or marrow only, but probably lymphogenous. (*a*) The *large mononuclear cells* are about three times the size of a red blood corpuscle, and contain a large round or ovoid nucleus and a large ring of protoplasm (E). (*b*) The *mononuclear transition form* differs from these only in that the nucleus is no longer round or ovoid, but indented (F). (*c*) The *polynuclear cells* (G) are somewhat smaller, but still larger than the red corpuscles, and their nuclei exhibit further differentiation, in that they are polymorphous. These are the more usual white blood corpuscles, which have long been recognized, and constitute two-thirds, 65 to 80 per cent., of all the white corpuscles.

Except in the lymphocytes the protoplasm of all these cells is granular, but the granulations possess different properties, which have also been investigated by Ehrlich, who makes five different varieties of granulation, which he distinguishes by letters of the Greek alphabet, from α to ε, as follows:—

The *α-granulation*, or *eosinophile* granulation, is characterized by large granules, highly refracting or oil-globule-like and stainable by all acid aniline staining fluids. As already mentioned, the eosinophiles were supposed by Ehrlich to be myelogenous. Their origin is undetermined, but it has been suggested that they also start as lymphocytes. They are rare in normal blood. They were called *eosinophilic* by Ehrlich because of their strong affinity for eosin. Their increase was also considered characteristic of leucæmia by Ehrlich, but though such increase

may be present, it is not distinctive. In fact, eosinophiles are increased in other conditions. Thus von Jaksch found eosinophile cells in the blood of an adult in health, of a boy with tuberculosis, in a case of pneumonia, and in all forms of anæmia. Dolega and Aldehoff

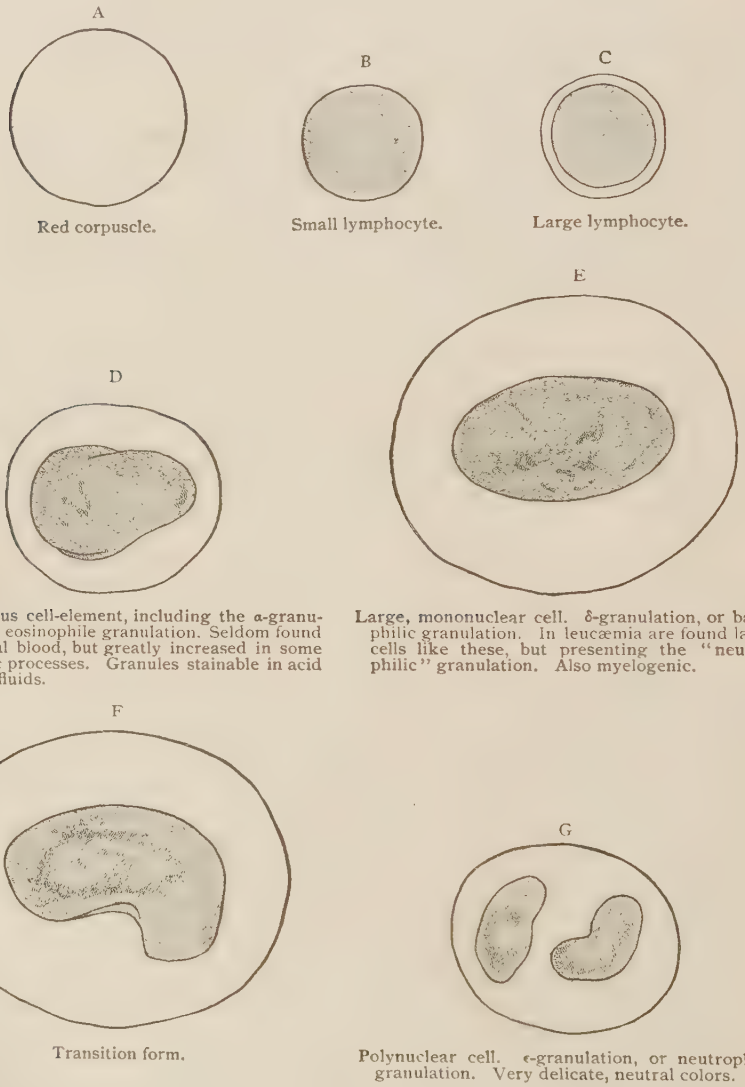
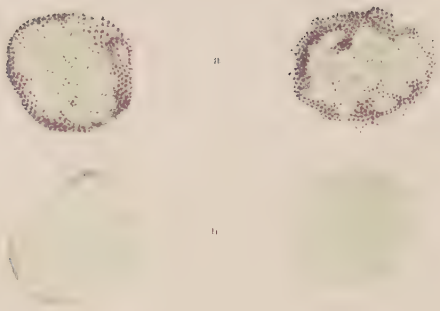

A
Red corpuscle.
B
Small lymphocyte.
C
Large lymphocyte.
D
Myelogenous cell-element, including the α-granulation, or eosinophile granulation. Seldom found in normal blood, but greatly increased in some leucæmic processes. Granules stainable in acid staining fluids.
E
Large, mononuclear cell. δ-granulation, or basophilic granulation. In leucæmia are found large cells like these, but presenting the "neutrophilic" granulation. Also myelogenic.
F
Transition form.
G
Polynuclear cell. ε-granulation, or neutrophilic granulation. Very delicate, neutral colors.

FIG. 54.—DIAGRAM INTENDED TO SHOW THE RELATIVE SIZES OF THE VARIOUS CELLS COMPARED WITH THE RED CORPUSCLE.—(*After a drawing by my colleague, Prof. Guiteras.*)

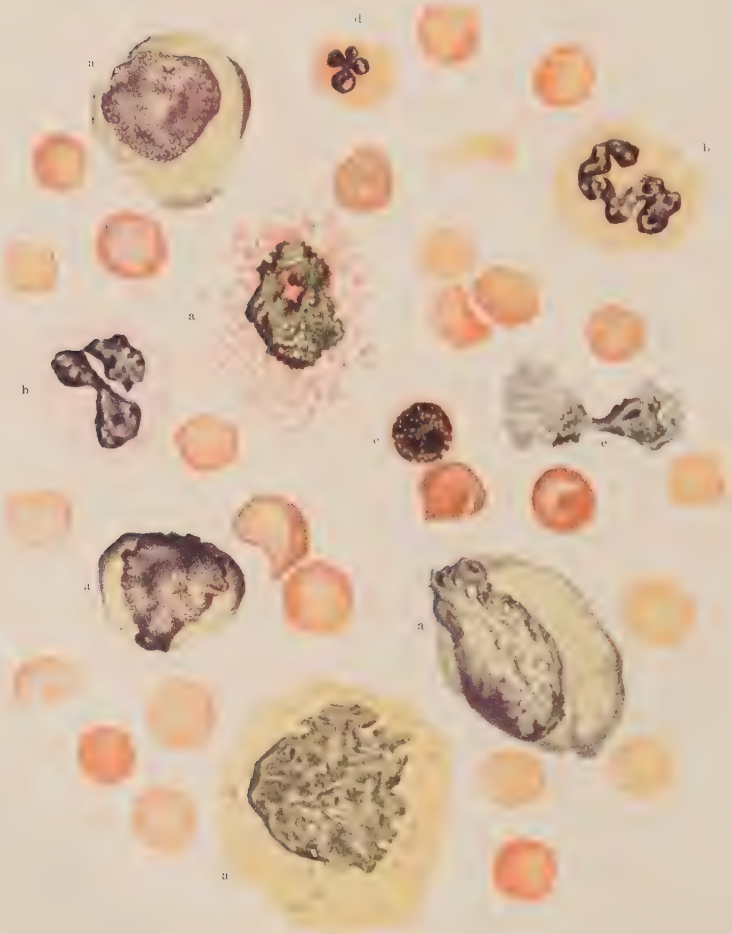
have recently found numerous eosinophiles in the blood of malaria in three cases. Müller and Rieder have made similar observations, and Fink found numerous leucocytes of the eosinophilic-granulation type

a.—MYELOCYTES OF EHRLICH.

b.—NORMAL MONONUCLEAR LEUCOCYTES.



STAINED WITH METHYL-GREEN—ORANGE G—ACID-FUCHSIN. NUCLEI GREEN, NEUTROPHILIC GRANULATIONS PURPLE.



STAINED WITH EOSIN-HAEMOTOXYLON. a.—MYELOCYTES OF EHRLICH. b.—POLYNUCLEAR LEUCOCYTES. c.—LYMPHOCYTE. d.—NUCLEATED RED CORPUSCLE. e.—DEGENERATED NUCLEUS.

in the blood of asthmatics. So also von Jaksch found the karyokinetic forms described by Müller (see below) in the blood of a case of sarcomatosis, as did also Weiss.*

The *β-granulation*, or *amphophilic* granulation, is stainable by acid and basic or similar fluids, hence called *amphophilic*. It is found especially in the cells of the medulla of bones, but also in blood, often in the leucocytes of rabbits and guinea pigs. It is frequently met in leucæmic blood.

The *γ-granulation*, or *basophilic* or "mastzellen granulation," † is stainable by basic aniline colors, as are bacteria. The granules are coarse and refract light but slightly. This granulation is almost altogether wanting in healthy human blood, but is increased in leucæmic processes. These granules are also found in the blood of some animals in health, as the white rat.

The *δ-granulation*, also basophilic, is finer, stainable in basic aniline colors like bacteria, and is found in the large mononuclear leucocytes, but is rare.

The *ε-granulation*, or *neutrophilic granulation*, is very delicate, and pervades the polynuclear elements of normal human blood very densely, is found sparingly in the transition forms of cells, and very seldom in the mononuclear elements. It is also characteristic of the *myelocyte* found abundantly in leucæmic blood. It is stainable by neutral coloring matters, and therefore called *neutrophilic*. *Such is the granulation of the common polynuclear leucocytes.*

All these varieties of granules may become free in the blood by the disintegration of leucocytes, and are among the sources of error in recognizing bacteria in the blood.

In normal blood the polynuclear cells form 65 to 80 per cent. of the colorless corpuscles, the lymphocytes 15 to 30 per cent., the mononuclear and transition forms about six per cent., and the eosinophiles ½ to eight per cent.

In addition to these elements of normal blood, all of which are found in leucæmia also, there are present the large neutrophilic cells also called by Ehrlich myelocytes, because he supposed that they originated in the marrow of bone. They closely resemble the large mononuclear leucocytes but also differ from them. They are as large and larger, mononucleated, but are neutrophilic instead of basophilic, containing fine, closely placed neutrophilic granulations. Ehrlich regards these myelocytes as marrow cells which have escaped into the blood as a consequence of the leucæmia. Löwit, on the other hand, regards it as a large mononuclear lymphocyte altered by disease. Myelocytes are rarely eosinophilic.

In addition to these, H. F. Müller ‡ has added another large mononuclear white cell, varying in size, but usually ½ to ⅓ larger than

---

* For references to all the above, see von Jaksch, "*Klinische Diagnostik*," 3d ed., 1892, p. 31. All are within the dates 1890–91.

† This word is retained because there seems to be no corresponding word in English.

‡ *Archiv für Klin. Med.*, 48, 51, 1891.

the ordinary white cell. The nucleus is large, plump, and oval, usually eccentrically placed and possessed of an intranuclear network rather more delicate than that of the ordinary leucocyte. The cell protoplasm is described as often surrounded by a slightly thickened layer of cell substance. Mitoses are numerous in these cells, and Müller contends that similar cells and karyokinetic figures are found in the bone marrow. They are similar to the cells originally described by Cornil as *cellules medullaires.* They may be the same as the myelocytes of Ehrlich. These cells are regarded, on the other hand, by Löwit as degenerate forms of mononucleated cells.

Diagnosis.—The diagnosis of leucæmia requires the microscope, but with it it becomes easy. (See Blood Changes.) A refinement of diagnosis, thus aided, attempts with indifferent success to separate the different varieties, which are known as simple or complex, according as one or more of the cytogenous organs are implicated. Thus, of the simple forms there would be the splenic, lymphatic, and myelogenous. Most leucæmias are, however, mixed. In his recent monograph, Hermann Rieder says that, so far as he knows, *pure splenic* and *pure myelogenous* forms have never been observed, while the pure lymphatic form is not infrequent.* Even the latter is, however, rare. A case of the pure myelogenous variety reported by Fraenkel, in a girl of fourteen,† is not now admitted to be a true instance. Another is reported by v. Leube and Fleischer.‡ The forms commonly met are the spleno-medullary and the lymphatic.

I. The most frequent of all the varieties is the combined form of *spleno-medullary* or lieno-myelogenous. In it the *lymphocytes* are relatively diminished, that is, instead of representing 15 to 30 per cent. of all the leucocytes, they represent a smaller proportion. *Eosinophiles* are present, but very variable in numbers and not distinctive. When increased they are conspicuously handsome in a stained preparation by their bright red granules. The *polynuclear neutrophiles*—the ordinary leucocytes—are either normal, 65 to 80 per cent., or relatively diminished, particularly toward the close. But the most characteristic feature of this variety is the presence of the large *neutrophilic myelocytes* described.

If relatively large leucocytes prevail, we may infer, as originally held by Virchow, the presence of the *splenic* form with a relatively small involvement of the lymphatic glands and of the bone-marrow. An excess of myelocytes is still held by Ehrlich as characteristic of involvement of the marrow,—in a word, that *myelogenous* leucæmia predominates.

II. Pure *lymphatic leucæmia* is less frequently met, probably less than 15 per cent. of all cases. In it the colorless cells are never so numerous, scarcely ever exceeding one in ten, while the lymphocytes only are increased, all the other forms being greatly diminished, and myelocytes are altogether absent. In a case reported by Uthemann, 93 per cent. of

---

* *Beiträge zur Kenntniss der Leukocytosis*, von Dr. Hermann Rieder, 1892, S. 36.

† *Deutsche medizinische Zeitung*. Berlin, 1890. Sajous' Ann., Vol. II, E, p. 19.

‡ Recall Virchow's announcement in "Cellular Pathology" in 1849, alluded to on p. 594, last paragraph.

all the leucocytes were lymphocytes, as compared with 15 to 30 per cent. in normal blood; in one of Osler's, 98 per cent. Even in the combined form lymphatic involvement is found. The superficial groups are usually involved, but never to the same degree as in Hodgkin's disease.

Combinations of splenic, lymphatic, and medullary changes are rare. Caution should be observed, too, in basing the presence of leucæmia solely on a leucocytosis, as some remarkable instances of this condition have been reported wherein leucæmia did not supervene. Thus von Jaksch, in his studies of the anæmia of children, found such proportions as one white to 12 red corpuscles, one to 17, and one to 20; and in the case of an adult one to eight, and still no leucæmia followed.* Eighty thousand white corpuscles per cubic millimeter is not unusual in leucocytosis, while 170,000 have been produced experimentally in the dog. The association of lymphatic or splenic or myelogenous change with the leucocytosis is therefore essential to a diagnosis. Rieder is also inclined to regard the cases of so-called acute leucæmia as really acute inflammatory leucocytosis rather than leucæmia.

Allusion has already been made to an anæmia described by von Jaksch as *anæmia infantum pseudoleucæmica*, further studied by Loos and Luzet, which is not to be confounded with leucæmia. Its essential feature is an enormous falling off in the cellular elements of the blood, the red cells being as low as 820,000, and the white 54,666. The proportion of leucocytes is always increased, but never to the same degree as in leucæmia, nor is it so rapidly brought about. On the other hand, the leucocytes are characterized by their varied shape and unusual size. The red cells display a high degree of poikilocytosis, while white cells enclosing red cells and fragments of red cells are also found, together with occasional eosinophilic leucocytes and large multinuclear neutrophilic leucocytes and nucleated red cells. All of these modifications of the blood corpuscles may occur in leucæmia, but in the latter disease there is not as marked a reduction either in the hæmoglobin or in the number of cellular elements. The difference in the form of the liver and splenic enlargement in the two conditions has been referred to on p. 595. It has been suggested that this is a form of disease intermediate between pseudoleucæmia, or Hodgkin's disease, and true leucæmia.

Prognosis.—The prognosis of leucæmia is unfavorable, the best that can be expected from treatment being the deferring of the fatal end. Some rather remarkable fluctuations are noted, and cases of cure are even reported, especially of late, by inhalations of oxygen. The lymphatic leucæmias are the more acute and more intractable.

Treatment.—The treatment has heretofore been mainly with iron, quinine, and arsenic, fresh air, and good food. Large doses of arsenic, as much as 30 drops of Fowler's solution, reached by gradual increment, have been especially recommended, and certainly should be tried.

Inhalations of oxygen, suggested in 1887, were used by Sticker and Pletzer with temporary benefit, and Da Costa and Hershey report

* *Op. cit.*, p. 29.

the apparent cure of one case, a boy of thirteen, and such marked improvement in a man of thirty-five that he was thought for a time to be cured. This treatment deserves, therefore, to be tried along with that first named. Thirty to 100 liters (about four to 12 gallons) of oxygen are to be inhaled daily.

IV.—LYMPHATIC ANÆMIA.—HODGKIN'S DISEASE.

Synonyms.—*Hodgkin's Disease; Pseudoleucæmia; Lymphadenosis; Lymphadenoma; Malignant Lymphoma* (Billroth); *Adénie and Lymph-adénie.*

Definition.—The disease consists essentially in an anæmia accompanied by a fibro-adenic enlargement of the lymphatic glands and the formation of lymphatic foci in the spleen and occasionally in other glandular organs, but unassociated with an increase in the colorless corpuscles of the blood.

Historical.—Hodgkin's paper, to which we are indebted for our first definite knowledge of the disease, appeared in 1832.

Etiology.—Its etiology is as undetermined as that of leucæmia. Depressing influences of all kinds are believed to favor it. Scrofulosis has sometimes preceded it. The presence of an irritating substance in the blood has been suggested, and the necessity of local irritation, associated with a lymphatic diathesis, has been insisted upon by Trousseau.

Morbid Anatomy.—Its morbid anatomy is, however, definite. There is both lymphatic and splenic involvement, the latter secondary to the former. The tonsils, intestinal lymphatic structures, and even the liver and kidneys may be invaded. There is, moreover, a deposition of new foci of lymphatic tissue decidedly more marked than in leucæmia. The lymphatic enlargement usually begins first in the more superficial groups, as those of the submaxillary region, neck, axilla, and groin, but the entire lymphatic system may be involved, including the retroperitoneal glands, resulting sometimes in marked abdominal enlargement. Occasionally the overgrowth is limited to the deep-seated glands. Of the abdominal, the retroperitoneal are most frequently involved, producing tumors which have been mistaken for myomata of the uterus. The bronchial glands may also be involved, and by their pressure produce dyspnœa.

The lymphatic enlargement is a hyperplastic one, shared by the cellular and trabecular tissue in varying degrees. When the former predominates, the product is soft and exudes a milky juice on section; when the latter, it is firm and resisting. The individual glands are not disposed to fuse nor to become adherent to adjacent tissue, differing in this respect greatly from glands enlarged by the tubercular process. The enlargement exceeds also that in leucæmia, while the splenic involvement is more limited, the organ rarely exceeding ten inches (25 cm.) in length, as contrasted with the colossal size of the leucæmic spleen.

The alteration in the spleen is hyperplastic, involving the pulp and Malpighian bodies jointly or either alone. The enlarged Malpighian bodies furnish the most characteristic feature. They form greyish-white

masses, varying in size from that of a lentil to a walnut, a couple of millimeters to as many centimeters in diameter, and contrast strongly with the dark red parenchyma. The process in the Malpighian bodies is a true hypertrophy of the adenoid tissue, while in the pulp it is rather the trabecular tissue which is overgrown. The spleen, however, is not always involved. Thus, Gowers found splenic enlargement in 75 per cent. of the cases collected; the organ contained lymphoid growths in 56 of these.

There are, likewise, at times changes in the marrow of bones, which is converted into lymphoid tissue, sometimes pyoid in consistence.

The liver and kidney, and even the thymus gland and lungs, are sometimes the seat of lymphoid deposits; in fact, all organs and tissues may be invaded, including the nervous; and paraplegia has resulted from pressure on the cord by growths in the spinal canal. The posterior nares may be occluded by invasion of the tonsils and the numerous lymphoid follicles in the pharynx. In like manner the intestinal walls may be invaded, producing thickening, while even serous surfaces do not escape.

Symptoms.—The symptoms of Hodgkin's disease are, again, the *pallor*, *weakness*, *dyspnœa*, *palpitation*, and other signs of anæmia, concurrent with or even sometimes in advance of the glandular enlargement. There is quite often *fever*, very irregular and variable in degree, and cases have been observed by Murchison and De Renzi in which there was *paroxysmal glandular enlargement* coinciding with fever, the enlargement subsiding with the decline of the fever, but not reaching the degree present prior to the enlargement. In a case of Laache's the glands diminished in size during the fever. In a case under my care in the wards of the Hospital of the University of Pennsylvania, in which the glandular enlargement was not conspicuous, there occurred an intermittent rise of temperature ascribed to a concurrent peritonitis, but the autopsy discovered this to be so limited that it is perhaps more reasonable to ascribe it to the feverish tendency characteristic of the disease.

The glandular enlargements themselves contribute further to the symptoms by their effects. Thus, in the case of the bronchial glands, dyspnœa from pressure on bronchi or trachea may occur, and is apt also to be intermittent. Pressure elsewhere may lead to pleuritic or abdominal effusions, while the entanglement of nerves in the growth may cause pain. Bronzing of the skin has been found associated with enlargement of the abdominal glands.

The external glandular growths are variously conspicuous; occasionally, however, they are wholly absent. There is no fixed order of involvement, although the submaxillary commonly enlarge first, and with the acme of their growth produce a striking picture. Extreme dyspnœa may arise from encroachment upon the trachea. The enlargement is not uniform, but at times remits and even ceases. It is said it may even disappear altogether for a time. The glands are usually soft, sometimes even to a sense of fluctuation. On the other hand, erosion of bone may result from pressure.

Macroscopically the blood appears thin and pale, and generally the red corpuscles are diminished in number, although not always. Minimum

counts make 960,000 * to the cubic millimeter, while in a case reported by Henry,† that of a boy of five, with enormous enlargement of the right cervical glands, there were 5,462,000 to the cubic millimeter. Thus the diminution is less than in pernicious anæmia. The hæmoglobin is, however, reduced to at least 60 per cent., furnishing thus one of the conditions essential to anæmia.

There are few nucleated red corpuscles and poikilocytes, and especially microcytes. The leucocytes may be slightly increased, occasionally decidedly so, but this is rare, and there is no approximation to the leucæmic state of the blood, and the two states are distinct and separate. A combination of the two may be possible.

Diagnosis.—The diagnosis requires some care, as more than one condition is attended by similar glandular outgrowths. Chronic and even acute adenitis has been mistaken for the early manifestation of Hodgkin's disease, while the converse has obtained perhaps more frequently. Time is the arbiter of such uncertainty.

A group of tubercular glands resembles more closely the disease under consideration, but it is not usually difficult to distinguish between the two. Tubercular glands are adherent to each other and adjacent tissues, while the lymphadenoid growths are loose and easily movable. Tuberculosis rarely involves more than one group of glands, is characterized by caseation and suppuration, while the lymphadenoid growths almost never suppurate. Yet the tubercular process is the slower. Tuberculosis is commonly found in young persons under twenty. Hodgkin's disease may occur at any age, but the average age is greater. It is more common in males. In Gowers' 100 cases, 75 were males and 25 females ; 30 were under twenty, 34 between twenty and forty, and 36 above forty.

Simple lymphoma, affecting as it does a group of glands, resembles most closely lymphadenoma. But it is harder and slower-growing than the lymphatic growth of Hodgkin's disease, while it does not affect the system.

Sarcoma also involves groups of glands, and in the beginning the consistence of the glands is similar to that in Hodgkin's disease. But this disease rapidly invades surrounding tissues, fusing with them, and destructive ulceration soon makes its appearance.

Carcinoma of lymphatic glands should also be mentioned as producing a somewhat similar growth, associated also with cachexia, but it is for the most part secondary to cancer somewhere else.

Finally, all the conditions named as possible to be mistaken for Hodgkin's disease are limited to single groups, while the latter always extends, and the fact of such limitation is of itself sufficient to preclude the disease.

From leucocythæmia the disease is easily distinguished by the leucocytosis characteristic of the former.

Prognosis.—While the prognosis is ultimately fatal, the course of the disease varies greatly, and death seldom results in less than a year.

---

* Case reported by Richard Geigel, quoted by P. Henry ("Anæmia," Philadelphia, 1887), from *Deutches Archiv für Klinische Med.*, 1885, Bd. 37, p. 59. † *Op. cit.*, p. 67.

F. P. Henry puts the average duration of life at two years, but admits it is greatly modified by such circumstances as age and previous health of the patient.

Treatment.—Treatment, too, may avert the fatal termination for a long time. Extraordinary results in this respect have followed the administration of arsenic, and even recoveries have been reported. Large doses, arrived at by gradual increment, should be attained and kept up until some physiological effects are observed. Such doses are 15 to 20 minims (one to 1.3 c. c.) of Fowler's solution. Particularly happy results are claimed for the injection of arsenic into the lymphoid masses. Especially is this recommended when arsenic is not well borne by the stomach. From eight to 30 minims (0.5 to two c. c.) of Fowler's solution have been injected daily in divided doses. Inunctions of iodine and iodide of potassium are also recommended. Supporting treatment of all kinds, including quinine, cod-liver oil, and the best of food, is necessary.

Operative interference is sometimes necessary to avert danger to life threatened by the encroachment of enlarged glands on vital organs and functions, such as respiration. It has even been claimed that the removal of a group of primarily enlarged glands has cut short the spread of the disease, but such an apparent result is rather an evidence of error in diagnosis. In view of the fact that at an early stage a diagnosis is impossible, the removal of a local group of glands should be recommended.

V.—SPLENIC ANÆMIA, OR SPLENIC PSEUDOLEUCÆMIA.

This term is applied to a condition in all respects analogous to Hodgkin's disease, where there is splenic enlargement only and no involvement of the lymphatic glands. Attention was first called to it as a separate variety of pseudoleucæmia by Horatio C. Wood * in 1871, although reports of cases corresponding to it had been previously made by others. It has been more recently studied by Strümpell and Banti. It occurs alike in old and young without assignable cause.

Morbid Anatomy.—Its morbid anatomy consists in the splenic changes. The organ is greatly enlarged, approaching that of the leucæmic spleen rather than the spleen of Hodgkin's disease. It is three to four times its normal size, but retains its normal shape. It is indurated, and its incisures are deep. Its capsule is thickened and opaque in spots and sometimes adherent to adjacent tissues, as is often true of any large spleen. The histology of the organ differs from that of the leucæmic enlargement and that of the enlarged spleen of Hodgkin's disease. There is no true over-growth of the lymphatic tissue in the Malpighian body, but rather a destruction, it being replaced by an over-growth of the reticulum, producing a white body as large as a pea. In addition to this the organ is often traversed by bands of thickened reticulum, visible to the

* "Relations of Leucocythæmia and Pseudoleucæmia," Amer. Jour. of the Med. Sci., October, 1871.

naked eye. The change corresponds rather with that in the harder lymphatic glands of Hodgkin's disease than that in the spleen. In a word, as well stated by Banti, the histological alterations of the spleen consist of an atrophy and sclerosis of the Malpighian corpuscles. Marrow changes, like those described under pernicious anæmia, are also sometimes present.

The blood exhibits the changes one would expect where there is destruction of the tissue devoted to its reproduction. It is anæmic. The red discs are notably diminished, from 5,000,000 to as low as even 1,000,000. There are found also the other changes of the eruthrocytes characteristic of pernicious anæmia. There are poikilocytosis, megalocytosis, and microcytosis. Nucleated red blood cells are also sometimes present. The leucocytes are sometimes slightly more numerous, at others they are in normal proportion, and are said to be generally mononuclear.

Symptoms.—As the blood-changes are analogous to those of pernicious anæmia, so are also the symptoms, which include *pallor*, *weakness*, *dyspnœa*, *palpitation* associated with the signs of *enlarged spleen*, evidence of which is sometimes shown by its weight and the pressure it exerts before other symptoms show themselves. Finally, there results the cachectic state characterized by *emaciation*, a *deeper yellow color of the skin and mucous membranes*, a tendency to *hemorrhage* and *pyrexia*, *œdema*, *serous effusions*, *extreme muscular prostration*, and *mental hebetude*. There is also said to be at times, as in lymphatic pseudoleucæmia, an intermittent or *per saltem* course in the symptoms to the extent of apparent complete restoration to health in the intervals.

An increase in the urea of the urine has been noted by Strümpell, and is regarded as evidence of increased albuminoid metamorphosis.

All clinical facts go to show that the spleen is responsible in some way for a destruction of eruthrocytes and of their capacity for carrying oxygen.

The duration of the disease is from five or six months to three years.

Diagnosis.—The diagnosis of splenic leucæmia depends upon the presence of splenic enlargement associated with the phenomena of anæmia above described, and the absence of glandular enlargement, so conspicuous in Hodgkin's disease. Anæmic symptoms attend the chronic malaria so often associated with enlarged spleen, but the history of malaria in such cases is invariably present, while the degree of anæmia is not so deep.

Prognosis.—The prognosis has been regarded unfavorable, but may be modified by the results of splenectomy, which in the cases collected by Banti appears to have been successful in three out of four.

Treatment.—The treatment is that for the other anæmias, by iron and arsenic and nutritious food. The propriety of splenectomy must be determined on the merits of each case.

SECONDARY OR SYMPTOMATIC ANÆMIA.

This form of anæmia is the direct result of trauma, accidental hemorrhage, chronic disease, or toxic agents. I may again refer to the fact mentioned in the preliminary remarks on the anæmias, that reasons are being found to show that some at least of the so-called essential anæmias may be due to agencies tending to destroy the corpuscles rather than to diseases of the blood-making apparatus. The secondary anæmias include :—

I. *Anæmias Due to Hemorrhage, However Caused.*—Traumatic hemorrhage, post-partum hemorrhage, lung hemorrhage, and gastric and

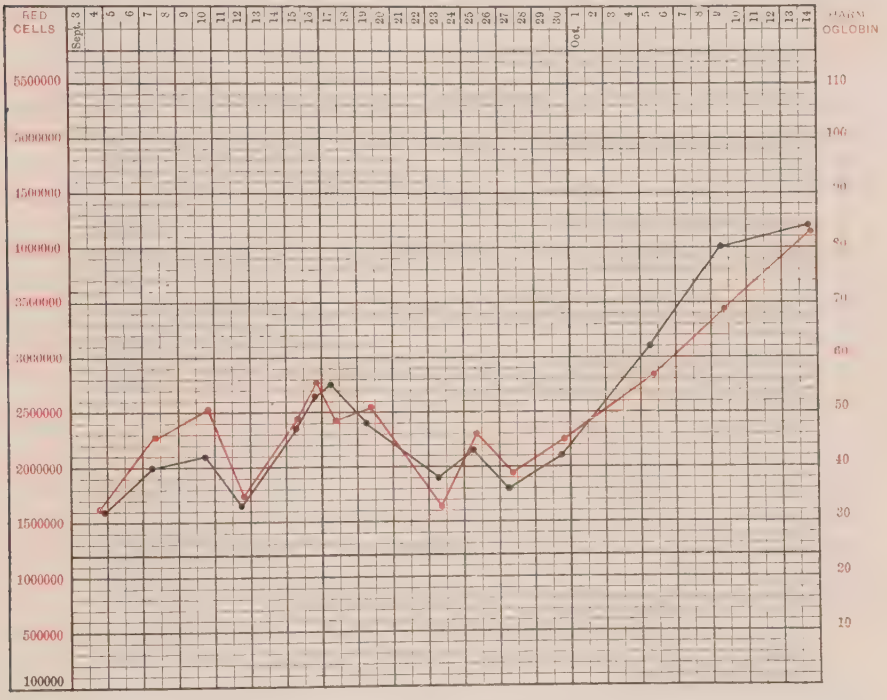


FIG. 55.—THE BLOOD IN SIMPLE ANÆMIA.

intestinal hemorrhages include most of these ; ruptured aneurisms, purpura, and the bleeding-habit furnish others.

In non-fatal hemorrhages from these causes the immediate loss of blood in bulk is rapidly made up by the absorption of water from the gastro-intestinal tract, but a long time is required even under favorable circumstances before the corpuscles and hæmoglobin are restored. At other times regeneration is quite rapid, restoration being complete in ten days. The hæmoglobin is always rather more reduced than the corpuscles, but they increase for a time *pari passu* as shown in the appended chart. The albuminous constituents are more rapidly restored.

II. *Anæmia Due to the Drain of Chronic Disease.*—Such are chronic Bright's disease, suppurative processes, or cancer, or prolonged lactation, or chronic diarrhœa. The changes are similar to those of acute anæmia with poikilocytosis rather more marked and fewer nucleated red corpuscles, though in some prolonged cases large nucleated corpuscles are met, some with karyokinetic nuclei. The colorless corpuscles are also commonly increased, rarely diminished. In this group belong the anæmias of malaria, in which the corpuscle is directly consumed by the plasmodium.

III. *Anæmia from Inanition.*—This results from starvation, which may be the practical consequence of diseases which interfere with the successful ingestion and assimilation of food, such as obstruction of the œsophagus by cancer or otherwise, or chronic and prolonged dyspepsia. Carcinoma of the stomach may also be included in this group. The last two groups (II and III) overlap.

Under these circumstances the blood mass is greatly reduced, but it is by a reduction in the blood plasma rather than the corpuscles, for the latter, though reduced, are not markedly so and may even be relatively increased.

The blood also undergoes morphological changes discoverable by the microscope. The disproportionate lowering of the hæmoglobin is explained by a more than natural paleness of the red corpuscles. Their average size is reduced while there is also a moderate poikilocytosis. Nucleated red corpuscles also make their appearance soon after a hemorrhage. They are about the size of a normal red disc, and exhibit, after staining with hæmatoxylon, a deep blue nucleus, while free nuclei are occasionally found.

The colorless corpuscles are moderately increased, such increase being represented by the multinuclear neutrophiles, while the small mononuclear lymphocytes are diminished. The leucocytosis gradually disappears with the return of the blood to its normal state. The presence of these as well as of the nucleated corpuscles is evidence of regenerative activity. In the absence of this they are absent and a permanent and ultimately fatal anæmia may ensue.

IV. Finally, there remain the *toxic anæmias.* These are the result of the presence in the blood of such substances as lead, acquired by painters or workers in lead-paint factories, typesetters, and type founders; also arsenic from dress fabrics, wall paper, and furniture coverings; mercury, and certain disease poisons, among which that of chronic malaria is the most conspicuous. Syphilis is also one of these, producing what is sometimes known as syphilitic chlorosis. The blood chart is from a case of syphilis. The anæmia of tuberculosis is sometimes classed among the toxic anæmias, but it probably belongs to group II, though it may be partly toxic also. The same may be said of the anæmia of pyrexia.

In the true toxic anæmias the poisonous substance acts directly upon the red corpuscles, destroying them, or perhaps also by increasing their consumption in the ordinary way.

Diagnosis.—In addition to the blood changes, more or less common to all of these causes of anæmia, the same general symptoms of

pallor, lassitude, debility, and faintness which characterize the essential anæmias are also present in less degree.

The distinctive feature of simple anæmia is the coequal reduction of the hæmoglobin and corpuscles. There is possibly an exception to this in the case of lead poisoning. The history of the case in the presence of one of the causes named is of itself sufficient to settle the diagnosis in many cases. The simple anæmias are not always, however, sudden or rapid in their occurrence, and a study of the body is often necessary to clear up a doubtful case.

Treatment.—The treatment of simple anæmia is eminently satisfactory. The simple administration of nourishing food with rest is followed by a very rapid coequal rise in the hæmoglobin and corpuscles, as is beautifully shown in the above chart made from one of my cases in the Philadelphia Hospital. And when to the treatment by nourishing food we add the use of iron there is nothing more to be desired. Full doses of iron are well borne in these cases, and we have the choice of almost any of the preparations, including Blaud's pills of the carbonate, reduced iron, tincture of the chloride, Basham's mixture, the vegetable salts. Though full doses are here indicated, it is still unnecessary to give the massive doses recommended by some, and they should be made short enough to produce constipation. The rapidity of the cure in some of these is only comparable to that of their production.

SECTION VI.

DISEASES OF THE THYROID GLAND.

GOITRE.

Simple Goitre or Struma.

Synonyms.—*Bronchocele; Thyrocele; Thick Neck; Derbyshire Neck.* (*The name is derived from L., guttur, throat.*)

Definition.—Under this name are included all enlargements of the thyroid gland other than those due to inflammation, malignant disease, exophthalmic goitre, or parasites. All simple goitres start in a true hypertrophy of the gland follicles, but assume ultimately special peculiarities on which are based anatomical varieties.

Etiology.—Simple goitre may occur endemically or sporadically, but in this country it is only sporadic. It is, however, quite prevalent about the eastern end of Lake Ontario and in the State of Michigan. It is still endemic in certain parts of Switzerland (cantons of Freiburg and Berne), in Italy (in the Southern Alps and in Savoy), in England, and in Asia in the Himalayas and Siberia. In the cantons named as many as 80 and 90 per cent. of recruits are found goitrous. It has even occurred in *epidemic* form in Finland.

The exciting cause of goitre still remains unknown, although there has long prevailed a notion as to the endemic form that some constituent of drinking water is responsible for it. That there is something in a locality is shown by the fact that removal from a territory subject to it arrests its development, while if a healthy family moves into a goitrous district the disease develops in some one or more members. A change in the water supply of a district where goitre has been prevalent has led to its disappearance, while the water in certain wells on the Continent of Europe is known to produce it. In fact, certain water is said to be drunk by men who desire to develop in themselves a goitre in order that they may be exempt from military service. What the responsible constituent of the water is, is, however, unknown.

It is much more common in women than men, according to different authorities seven to 41 times as frequent—it has been suggested because women drink more water The disease generally develops after puberty, sometimes after fifty. Congenital cases are known. It is sometimes hereditary, but heredity must be separated from the operation of one cause on different members of the same family.

Morbid Anatomy.—According to anatomical peculiarities assumed after the goitre sets in we have (*a*) *Struma mollis*, or parenchymatous, or hypertrophic goitre, in which there is a true hypertrophic enlargement; (*b*) *Struma aneurysmatica*, in which the vessels are enlarged and dilated; (*c*) *Struma fibrosa*, in which there is an excessive development of fibroid tissue; (*d*) *Struma colloides*, in which the follicles are enlarged and filled with colloid matter; *Struma cystica*, when the follicles have enlarged to cysts with liquid contents; (*e*) *Struma ossea*, characterized by calcareous infiltration, and (*f*) *Struma amyloidea*, in which there is a wax-like product caused by amyloid change. There are various combinations or intermediate types.

Symptoms.—It may be said of the majority of goitres that they cause no inconvenience, and are mainly objectionable through the deformity they cause. The size attained is various. The enlargement may but slightly exceed that of the normal gland, or the organ may be very large and pendulous. It may be one-sided or bilateral, or only affect the isthmus. It is characteristic of all goitres and enlargements of the thyroid of any kind that they rise up when the patient swallows, and tumors of doubtful locality may thus be located. The goitre is sometimes low down behind the sternum and can only be felt during deglutition.

A goitre may press on the trachea, causing dyspnœa, or upon the œsophagus, causing difficulty in swallowing. When behind the sternum it may press upon the veins in the neck, causing swelling of the face and head and sometimes headache and drowsiness. There may be pressure on nerves, especially the pneumogastric, causing spasm of the glottis, paralysis of the abductor, and even complete paralysis of one or both vocal cords.

Treatment.—The medical treatment of goitre consists principally in the topical application of tincture of iodine. It is undoubtedly efficient at times. The simple iodine ointment or ointment of the red iodide of mercury may be daily rubbed into the goiter. It is recommended that after applications of the latter the neck should be exposed to the rays of the sun. This treatment has been especially efficient in India. Injections of iodine into the cyst are also used—20 to 30 minims (1.3 to two c.c.) of a solution of one part in 12 parts of alcohol twice a week, a new point being selected each time, care being taken not to wound any vessels or nerves.

Internal treatment is also recommended. Naturally the iodide of potassium is conspicuous among remedies in the usual doses, five to 20 grains (0.3 to 1.3 gm.) three times a day. Thyroid extract is also being used with apparent success. Bruns treated 12 cases with raw thyroid glands in doses of five to ten gm. (75 to 150 grains) twice a week at first and once a week afterward. Nine were benefited. Kocher, however, thinks that the results with thyroid extract are no better than with iodine.

When large and causing dangerous symptoms, goitre falls properly into the hands of the surgeon, who treats it as exigencies demand, sometimes extirpating it, though the operation is rather formidable at times and there is some risk of its being followed by myxœdema. Cysts may be incised and drained or injected with iodine or perchloride of iron

solution. Sir Morell Mackenzie injected the latter after tapping, using two drachms (7.4 c. c.) of a 25 per cent. solution.

If the goitre is determined by local causes, a change of residence is, of course, desirable.

Exophthalmic Goitre.

Synonyms.—*Struma exophthalmica; Graves's Disease; Basedow's Disease; Cardio-thyroid Exophthalmos; Tachycardia strumosa.*

Definition.—A disease characterized especially by enlargement of the thyroid gland, protruding eyeballs, and frequent pulse.

Historical.—In the early part of the nineteenth century cases in which exophthalmos was a symptom were published by different authors. Some of these were probably examples of exophthalmic goitre. In 1825 C. Parry published several instances of a disease not previously described which were evidently cases of exophthalmic goitre. In 1828 Adelmann published a case declared on Virchow's authority to be one of exophthalmic goitre, and therefore the first described in Europe. In 1833 Trousseau reported the history of a woman affected with goitre, exophthalmos, and cardiac palpitation at the same time, of which he said afterward that he was far from supposing that this symptomatic triad constituted a special morbid entity. In 1835 Graves published the clinical lecture whence arose his association with the name of the disease, which will perhaps continue through all time. He also described it in his "System of Clinical Medicine," published in 1843. He was the first to emphasize the absence of organic cardiac disease. In 1840 von Basedow published the results of his study of four cases of exophthalmic goitre, and with it the most exact description of the disease which had yet been given. He gave to the disease the name *exophthalmic cachexia*. Although his name has also become associated with it, and the Germans insist on the priority of his studies, he seems to have been clearly anticipated by Graves. Others who described the disease were—Romberg in 1851, Schoch in 1854, and in 1855 Koeben, who first ascribed the disease to derangement of the sympathetic nervous system. In 1855, too, Stokes included the disease in his book on "Disease of the Heart," and drew special attention also to the nervous phenomena. Charcot thoroughly described the disease in 1856 and 1857, and in 1857 von Graefe described the symptom known as Graefe's. In 1859 Fischer referred the symptoms to the remote effects of anæmia. In 1860 Trousseau asserted the nervous origin of the disease and that it should be classed as a neurosis. In the same year Aran ascribed the exophthalmos to a contraction of the muscular fibres of Müller supplied by the sympathetic. In 1862 took place a notable discussion in the Academy of Medicine of Paris, when Trousseau maintained that in all cases of exophthalmic goitre there was either an antecedent or a coincident derangement of the heart, and reasserted its nervous origin. Piorry denied the unity of the disease, and claimed that the prominence of the eyeballs was the result of retarded intracranial circulation due to pressure of the enlarged thyroid upon the external and internal jugular veins.

The neurotic nature of the disease is now generally admitted, and

most recently the medulla oblongata has been selected as the site for the lesion. This view has the support of some experimental evidence, and in a few autopsies changes have been found in the medulla.

Most recently the startling facts developed in connection with myxœdema and the results of its treatment have drawn attention to a possible antithetic relationship between the two conditions. Thus we have on the one hand, associated with exophthalmic goitre, excitability of the nervous system manifested in restlessness of mind and body, a rapid action of the heart, and a moist, flushed skin; with myxœdema the opposite conditions of dulness of intellect, torpor of body, slowness of pulse, dryness of skin. From this standpoint Moebius and Greenfield have taken the position that exophthalmic goitre is such an antithetic disease. Greenfield further adduces the fact that the changes in the gland are those of an organ in active evolution, *i. e.*, proliferation with production of new tubular spaces and absorption of the colloid matrix, to be replaced by a more mucous fluid. Further are the facts that thyroid extract in excess produces tachycardia, tremor, headache, sweating, and prostration, symptoms of Graves's disease; that when administered during the disease it aggravates the symptoms, has, indeed, in an over-dose caused it, as in a case of Beclères'; and that the most successful treatment has been such as reduces the bulk of the gland. The view that it is a sympathetic neurosis has always commended itself, appearing as it does to explain the symptoms more satisfactorily than any other—the frequent pulse, the exophthalmos, the thyroid palpitation, the sweating, and the general nervousness are all best thus explained.

Etiology.—Exophthalmic goitre is more common in women than in men, according to Trousseau, as 50 to eight. Others make it twice as frequent, others ten times. I do not remember having seen more than one case in a man. It is also more common in the young, adult, and middle-aged. The average age may be put down at from thirty to thirty-one years; Byrom Bramwell says fifteen to thirty for women and thirty to forty-five for men. The oldest patient I have ever seen was forty-four. It has been observed as early as two and a half years and as late as sixty-eight. Heredity is a rare factor, but its influence cannot be denied. It sometimes happens that several members of a family are affected. Sometimes myxœdema affects one member of a family and exophthalmic goitre another. It occurs with especial frequency in neurotic families. Sudden mental shock, worry and grief, and physical fatigue are assigned as exciting causes. So are many acute diseases, of which rheumatism is especially cited, also typhoid fever. Some of these are more likely to be coincidences.

Symptoms.—Of the cardinal symptoms mentioned in the definition the cardiac and vascular usually appear first. The *palpitation* is extreme, delirious, as it were, the pulse rate being commonly in the neighborhood of 120 to 140, and is said sometimes to reach 200. The slightest excitement augments the pulse rate instantly. The cardiac impulse is strong, but the volume of the pulse small. The normal heart sounds are loud, audible to the patient and even at a distance from the body, in one described by Graves himself as far as four feet. A systolic murmur is often heard

at the base, usually soft, but sometimes loud; more rarely at the apex, when it may be due to relative insufficiency of the mitral or tricuspid.

Exophthalmos is commonly described as the second of the cardinal symptoms to appear, but it is not far behind the palpitation. The degree of this protrusion varies very decidedly. It may be so slight as to be scarcely noticeable, while again the peculiar staring effect arising from it is conspicuous and attracts attention instantly. The eyes show a large amount of white and the eyelids when closed often cannot cover the eyes. It is in these extreme cases that *von Graefe's symptom* presents itself, a condition in which, when the eye is cast down or raised, the lid fails to follow it, as it does in health. This symptom, of which so much has been said, is not a very frequent one. *Stellwag's sign*, which is less known, seems more frequently met. In it the palpebral fissure is increased in width owing to the persistent retraction of the upper lid. It may occur with or without von Graefe's. Retraction of the lower lid is occasionally seen. Moebius considers Graefe's symptom the result of *Stellwag's*. The patient winks less frequently than in health. Pulsation of the retinal arteries can be seen with the ophthalmoscope, but other changes in the retinæ are rare. The same is true of the pupils. A lack in convergence of the two eyes was pointed out by Moebius.

The *thyroid enlargement* commonly presents itself about the same time with exophthalmos. A patient of mine said almost as early as she noted swelling she observed her eyes began to protrude, showing that the events are not far apart and that the three distinctive symptoms appear almost simultaneously. The goitre itself is in no way peculiar. It is usually of moderate size, almost never reaching the dimensions sometimes attained by a simple goitre. The tumor is largely contributed to by its vascularity, though there is also an overgrowth of the proper glandular tissue of the thyroid. Pulse and thrill are both palpable, while a loud systolic murmur may be heard on auscultation.

Another symptom already alluded to, included by some as cardinal, is what is commonly known as "*nervousness*." It includes irritability, restlessness, a disposition to start at the slightest sound, wakefulness at night. As a part of this, or as due to the same cause, at least, is "*tremor*," a highly important symptom, of such frequency as to be included by George R. Murray in his definition.* It does not, however, appear, as a rule, until the other symptoms have been present for some time. It may be best studied by holding out the hand with the palm downward; even better by laying the examiner's palm lightly upon the patient's fingers when the hand of the latter is held out. The tremor affects the flexor and extensor muscles of the wrist and not the intrinsic muscles of the hand, so that the fingers do not vibrate independently but the whole hand moves. It is rapid, regular, and uniform while it lasts. It occurs from eight to nine times in a second. Its extent is small but not always the same. It may be seen, too, in the foot, and in some instances the whole body appears to tremble. It is generally equal on the two sides of the body, but has been unilateral when

* "Twentieth Century Practice of Medicine," Vol. IV, 1895.

goitre and exophthalmos have been on one side. It is variously modified by position, lying, sitting, or standing. It does not, as a rule, interfere with the movements of the hand, but when excessive may hinder sewing or writing. The tremor of general paralysis and alcoholism is less regular in extent, while the individual fingers tremble, the rate being practically the same. In the tremor of old age the rate is only one-half as rapid; in disseminated sclerosis it is from three to six times a second, and in paralysis agitans it is less regular. It resembles more the tremor of fatigue or that seen in recovery from long illness. Another symptom included in the same category is a sudden giving way of the legs, so that the patient falls to the ground without previous feeling of faintness or giddiness, and Charcot mentions also a weakness of the legs. Painful cramps sometimes occur. Localized muscular atrophy, and that peculiar nervous symptom known as astasia-abasia, in which there is inability to stand and inability to walk.

The *mental condition* has been alluded to. It may be added that fits of depression alternate with buoyancy, while the moral nature may also be changed to a degree, amounting to melancholia and mania. Active cerebral symptoms are sometimes present and characterized at least one rapidly fatal case. Dulness or stupor seems to be entirely absent. Excessive *sweating* is a frequent symptom. It may be intermittent or irregular or there may be a simple feeling of flushing without sweating. Diminished electrical resistance was pointed out by Vigouroux. This is a natural result of the constant moisture of the skin. *Polyuria* often occurs, caused perhaps as is the sweating. Albuminuria has been noted. A dark *coloration of the skin* sometimes takes place, more decided in those situations in which the pigment is naturally more abundant, such as the face and arms. Yet the flexures of the joints, the axillæ, the areolæ of the nipples, the genitals, and inside of the thighs are also affected. The skin may be uniformly bronzed or it may be darker in patches. Parts of the body which are subject to constant pressure are also disposed to take on pigmentation more deeply. *Œdematous swellings* of the skin in various parts of the body may occur and are to be carefully separated from œdema, the result of associated conditions, such as anæmia, organic heart disease, etc. It manifests itself as swelling of the feet and ankles, and has been ascribed to vaso-motor paralysis. The *nails* sometimes become thin and occasionally have a corrugated appearance.

Gastro-intestinal symptoms are frequent, manifesting themselves by attacks of diarrhœa, apparently of nervous origin, coming on suddenly without pain, with copious loose motions of which there are two or three or more in a day. With this vomiting may be associated. Acute forms are sometimes thus ushered in. The tongue, however, remains clean, and there is, as a rule, no rise of temperature. Sometimes there may be very slight fever. The skin coloration and gastro-intestinal symptoms suggest those of Addison's disease and it is not impossible it may have been associated. *Rapid breathing* is a frequent accompaniment, equaling 30 to 40 a minute. It may be associated with cyanosis of the face and swelling of the vessels of the neck. Derangements of menstruation are less frequent than might be expected, this function being normally maintained in the

majority of instances. Pregnancy is infrequent. Among complications hysteria and chorea and even epilepsy are included.

Diagnosis.—This need not detain us. The combination of the three cardinal symptoms named can receive no other interpretation. There may be some doubt at the beginning which time will shortly solve.

Prognosis.—Exophthalmic goitre is rarely fatal in itself, the patient dying of some other disease. At times the condition remains permanent, with little change, but in the course of time the majority of cases improve vastly and some get well. I have been watching for years a young woman who, fifteen years ago, had bad exophthalmic goitre, with its train of symptoms marked, who is now well except that her eyes are slightly more prominent than is strictly natural.

In a few rare instances a rapidly fatal course ensues, death taking place in a few days after the onset. The majority of cases run a chronic course, the symptoms persisting more or less for years. When death occurs it is from failure of the heart. It is generally preceded by an aggravation of all the symptoms, but previous to it the patient has been pretty well worn out. It may be sudden, as by syncope. Acute cases are reported with characteristic symptoms, following one of the cited causes, in which the symptoms lasted a few days and then disappeared completely.

Treatment.—Rest and protection from excitement are essential conditions to a successful treatment. After this the treatment is directed to the symptoms mainly. The remedies most used are the bromides and digitalis ; digitalis to slow and steady the pulse ; the bromides for two reasons : (1) as nervous sedatives, and (2) for their reputed action in producing anæmia of the nerve-centres.

In some cases, where there is no cardiac lesion and the pulse is good and strong, aconite with the bromides are of service. Ergot, for its power of contracting the calibre of blood-vessels, is also a rational remedy. There is a difference of opinion as to the propriety of administering iron. I believe the decision should be based on the condition of the patient and the presence or absence of anæmia. By German writers, galvanism of the sympathetic is claimed to be of service. Theoretically it should be. A constant current of from five to eight cells is used ; the negative pole is placed on the 5th cervical vertebra, the positive pole along the sternum. Special efficiency has been claimed for the tincture of nux vomica by J. Newton Hunsberger, of Skippack, Pa., who called my attention to it. Dr. H. gave it to a well-marked case, beginning with doses of 25 drops, increasing to 50. In three months after the treatment commenced the patient was able to do all her work and has continued well ever since.

Thyroid extract has not proved useful so far as tried,—in fact, has appeared to be harmful. The results of operative treatment have been somewhat satisfactory. Oppenheimer has collected 68 cases, of which 18 completely recovered, in 26 there was more or less improvement, in nine there was no change, five died almost immediately, and four within twenty-four hours.

MYXŒDEMA.*

SYNONYMS.—*Cachexie pachydermique* (Charcot); *Cachexia thyroidea vel strumipriva vel thyreopriva* (Kocher); *Athyrea; A Cretinoid State Supervening in Adult Life in Women* (Gull).

Definition.—A myxomatous infiltration of the subcutaneous connective tissue, characterized also by dryness of the skin, subnormal temperature, mental failure, and atrophy of the thyroid gland.

Historical.—The disease was first described by Sir William Gull in England in 1873 as a cretinoid change; further described by William Ord in 1877, and named by him myxœdema, and by Charcot, who called it *cachexie pachydermique.* In 1882 Jacques L. Reverdin called the attention of the Medical Society of Geneva to the occurrence of the symptoms of myxœdema after total extirpation of the thyroid in the human subject, naming the condition *myxœdema operatoire.* In 1883 Kocher reported to the Twelfth Surgical Congress at Berlin similar results in patients from whom he had entirely removed thyroid glands, calling the condition *cachexia strumipriva*, having previously known nothing of myxœdema as a disease. He did not, therefore, attribute the condition to the loss of the thyroid but to injuries received in the structures of the neck in the operation. Two months later Reverdin discussed the relationship between myxœdema and cachexia strumipriva, and shortly afterward Felix Semon suggested that the loss of function in the thyroid gland was probably the common factor in the production of both. Victor Horsley in 1890 found that the removal of the gland in monkeys was followed by myxœdematous symptoms instead of tetany, as announced by von Eiselberg † the same year, ascribing the tetanic symptoms to the fact that the animals were kept at a temperature of 90° F. (32.2° C.) after the operation. N. Weiss also pointed out that tetany is apt to follow operative extirpation of goitre. In 1890 Horsley published a note on the possibility of the successful treatment of myxœdema, sporadic cretinism, and cachexia strumipriva by grafting with thyroid tissue from the necks of animals, though in this he was anticipated by Kocher in 1883 and Bircher in 1889. Kocher's graft was absorbed but Bircher's patient was enabled to return to work. Victor Horsley's results were a part of a report by a committee of the Clinical Society of London, appointed in 1883, whose conclusions were as follows:—

(1) Myxœdema is identical with cachexia strumipriva.

(2) Sporadic cretinism is myxœdema occurring in childhood.

(3) Endemic cretinism is also closely allied to myxœdema.

(4) And further, that while these conditions are dependent on loss of function due to removal or disease of the thyroid gland, the ultimate

* The student is referred to five noteworthy papers on the subject of "Myxœdema and Cretinism" in Vol. VIII, 1893, Transactions of the Association of American Physicians, by Francis P. Kinnicutt, James J. Putnam, M. Allen Starr, W. Gilman Thompson, and William Osler; to the "Atlas of Clinical Medicine," by Byrom Bramwell, for admirable illustrations; and to the exhaustive article on "Myxœdema," by George R. Murray, of Newcastle, England, in the "Twentieth Century Practice of Medicine," Vol. IV, 1895.

† Von Eiselberg's experiments were made chiefly on cats.

cause of this loss of function in ordinary myxœdema is not as yet explained.

Etiology.—Some change in the thyroid gland as the result of which it becomes functionless is a necessary condition of myxœdema. Commonly it is atrophy and it may be preceded or followed by exophthalmic goitre. The inference is that the thyroid gland supplies some secretion which is essential to a normal nutrition, but exactly how this operates is unknown.

Symptoms.—Three groups of cases are recognizable :—

(1) Pure myxœdema.

(2) Myxœdema associated with congenital or sporadic cretinism.

(3) Operative myxœdema or cachexia strumipriva.

I. *Pure Myxœdema.*—This is much more frequent in women than men, at least as six to one, and occurs usually between the ages of thirty and fifty, but it is not confined to these ages, being found in those who are younger and older. Heredity is a recognized factor, acting usually through the mother. Several members of a family may be affected. The poor suffer most. It is said to have no relation to the catamenia, but has followed frequent pregnancies, injuries, severe hemorrhage, and mental disturbance. Most essential is some change in the thyroid gland. Formerly thought to be rare in this country, cases have multiplied since attention has been called to it.

The face is the chief seat, but the extremities, the trunk, tongue, and even internal organs may be involved. The *face is uniformly swollen, broadened, and flattened*, the *nose broad*, the *mouth large*, all lines obliterated, and expression gone. The *skin* of the neck above and below the clavicle is thrown into folds of fatty and myxomatous tissue. It is yellow, translucent or waxy, dry and scaly. The cheeks are flushed and sometimes the nose. True œdema may be associated, and there is rarely albuminuria and glycosuria. The *hands lose their natural shape* and were described by Gull as "spade-like," the *feet* are also misshapen ; the *gait* is slow and labored. The *mind is feeble*, slow in its action, memory is poor, while irritability and suspicion are added qualities, and sometimes there are delusions and hallucinations, ultimately often dementia. The organic functions are fairly well performed. The course of the disease is slow and the patient usually dies of some intercurrent affection. *Atrophy of the optic nerves* is a rare but possible symptom, also *synovitis* from trifling causes. *Subnormal temperature* is a characteristic symptom, though in early stages the temperature may be normal or slightly above. In winter the patient always feels cold and hugs the stove.

II. *Myxœdema Associated with Cretinism, Congenital or Acquired.*—Cretinism is a form of idiocy associated with the absence of the thyroid or with a functionless thyroid. It is myxœdema in childhood. There is almost complete arrest of mental and bodily development. The *cretin* is a dwarf. In the *congenital* form there is congenital absence of the thyroid, and the child is further characterized by its thick neck, short arms and legs, and prominent belly. The face is large, the lips thick, and the tongue large and often protruding. All the bones of the skeleton are short and broad, the epiphyses swollen but not ossified. The skull is short

and broad and the baso-sphenoid junction early ossified. The cretin resembles the rickety child and may be confounded with it.

Acquired cretinism may start before birth and be barely appreciable at birth. More frequently the infant appears normal at birth, and the changes make their appearance between the second and fifth year. The arrest of development continues, or, rather, there is very slow development, so that at adult age the man or woman does not exceed in stature a child of from five to seven years. The myxœdemic symptoms are much as described in pure myxœdema.

III. *Operative Myxœdema, or Cachexia Strumipriva.*—By this is meant a condition of myxœdema the result of removal of the thyroid. See history, page 619. It is more likely to follow total than partial removal of the thyroid, but does not follow every case, having been observed in 69 out of 408 cases. Cases of operative myxœdema are very rare in this country.

Diagnosis.—This is easy. The œdema of Bright's disease or heart disease may be confounded, but the peculiar flat face, the absence of pitting on pressure, and, above all, the absence of albuminuria and of the signs of heart disease are distinctive features of myxœdema.

Prognosis.—This was regarded as unfavorable until the hypodermic use of thyroid extract was suggested by George R. Murray, of Newcastle, England, in 1891, based upon the satisfactory effect obtained by Bettencourt and Serrano in 1890 in engrafting sheeps' thyroid in human subjects having myxœdema,—the idea being in this manner to substitute the juice or secretion of the gland. Every expectation was realized. In 1892 Howitz, of Copenhagen, and soon afterward E. L. Fox and H. Mackenzie, in England, substituted for the hypodermic use the administration of the gland itself or some preparation of it by the mouth. At the present day the effects of the administration of thyroid preparations in myxœdema are among the marvelous results of medicine. The duration of the practice has, however, been scarcely sufficient to admit of unqualified conclusions as to it.

Treatment.—The treatment at the present day is, therefore, solely by the sheep's thyroid. The gland is best administered in the shape of the glycerine extract. From 15 minims (one gm.) to 30 minims (two gm.) are to be given daily, its effect being carefully watched. If no effect follows, the dose should be doubled, and further increased if necessary. As improvement takes place smaller doses should be given at longer intervals until finally ten to 15 minims (0.66 to one gm.) are given once a week or less often. During the first week the patient should be watched with a view to guarding against heart failure, prohibiting all physical exertion on his part, this being an apparent effect of the medicine in excessive doses.

The myxœdema being removed, it is necessary, of course, to continue the treatment in this second stage by such doses as will maintain the cure; for it is to be understood that as the thyroid is still functionless, the omission of the treatment is followed sooner or later by its return. The quantity required varies in different cases, but it is found to range between five to 15 minims (0.33 to one c. c.), the more precise dose being de-

termined by trial. A single daily dose is preferred by Murray to a smaller dose more frequently repeated. A fall of temperature below normal, a slight return of swelling or of other symptoms, indicates that too small a dose is being given, while acceleration of the pulse indicates that the dose should be reduced. In a climate not subject to great variations the same dose can be given the year round. In hot weather a smaller dose suffices than in cold, and a dose that has been found sufficient during the summer may not be enough in the winter.

Of the treatment previous to the introduction of the thyroid treatment, that by jaborandi and pilocarpin should be alluded to, because the partial benefit derived from them alone is explainable by supposing that they produced increased activity in small portions of residual glandular tissue. Should thyroid extract be unattainable, these drugs may still be used.

Cretinism.

Synonyms.—*Cretinismus; Cretinoid Idiocy; Idiotie avec cachexie pachydermique.*

Cretin is Swiss patois for Chrétien, a Christian. The word is also derived by some from the Latin *creta*, chalk.

Definition.—Cretinism has been alluded to in treating of myxœdema. It may be defined as a condition due to diminished functional activity of the thyroid gland, commencing in embryonic life, or after birth before the age of fifteen. It is, as already said (p. 620), a congenital or infantile form of myxœdema. From the fact, however, that the disease may start to develop later than infancy, there results a series of types intermediate between those represented by congenital and adult cretinism. This arrest of mental and physical development is, of course, greater the earlier the disease begins to develop; whence two cretins of the same age will differ materially if one has commenced to develop at or before birth and the other not until seven or eight years of age.

Cretinism may be endemic, as in some parts of the Continent of Europe, or sporadic, as in England and America. The sporadic cases are, as a rule, without goitre, the thyroid glands being either undeveloped or atrophied, while one-third also of the endemic cretins are without goitre. In either event, the gland is functionally dead, even though it may appear natural in size, the original true gland tissue having been replaced by an indifferent element. Endemic cretinism occurs in localities where goitre is also endemic,—in the shut-up valleys of mountainous districts of Europe and Asia.

Congenital Cretinism.—True congenital cretinism, that is, cretinism which is evident at birth, is very rare. Some cases of intrauterine rickets may have been cretinism. In most cases the child does not long survive its birth.

When examined the thyroid is found either undeveloped or completely atrophied. The body is short and broad. The peculiar elastic swelling in the subclavicular fossæ common to other forms of cretinism is found. The skin is thick and lies in folds. The sub-

cutaneous adipose tissue is well developed. The other symptoms are those described on p. 620. In another form described by Horsley the disease is supposed to begin shortly before birth, but develops slowly, so that at birth it has not attained the degree described, and the child can live. In this there is usually a goitre at birth. The body appears large and there may be myxomatous swelling of the subcutaneous tissues, the nose is flat, tongue large, the neck short and thick. Mental development is slow, and later in life the symptoms become more decided *pari passu* with progressive degeneration of the thyroid gland.

Sporadic and Endemic Cretinism.—In this form the child is apparently healthy at birth and may continue to develop in a normal manner for months and even years before the disease manifests itself.

Etiology.—The affection is the result of disease of the thyroid gland, but what excites this disease is not known. It is much more common in girls. Heredity plays no part, as cretins are not sexually developed. Yet it occurs in members of the same family, which only goes to show that one cause operates to produce it. Many are the children of neurotic persons, while Langdon Down considers that alcohol is responsible, especially if the alcoholic habit is present at the time of procreation. The sporadic form occurs in any country, including America, but the endemic form seems confined to the Continent of Europe, as stated on p. 622. At one time, in 1847, a number of cases—some 24 out of a population of 350—prevailed in Cheselborough, Somerset, England, but it has died out. The endemic form is commonly ascribed to the use of certain drinking waters, but no responsible constituents have been isolated. The child, being normal at birth, remains so until the change begins in the thyroid, when it becomes less lively, development is arrested, and the conditions described on p. 620 slowly develop. The cretin may reach the age of thirty to forty years, but ceases to change after the twentieth year, whether sporadic or endemic.

Morbid Anatomy.—The morbid changes in the cretin after death are those described as characteristic in life, but autopsy has also disclosed the thyroid absent in nine out of ten cases examined, confirming the theory of its origin. Enlargement of the *hypophysis cerebri* was found in six cases by different observers, and Horsley says that the convolutions of the brain are ill-defined and the blood-vessels small, even in proportion to the rudimentary condition of the nervous system.

Treatment.—This is by the thyroid extract, as also described under myxœdema. It will be remembered that its continuous administration is necessary to produce a cure. Relatively larger doses of thyroid extract can be given to children than to adults. The liability to syncope during treatment is less marked than in myxœdematous adults, but it is advisable not to allow any unusual exercise during the first part of the treatment. In the absence of the extract, the sheep's raw thyroid may be administered, giving one-eighth to one-quarter lobe twice a week.

Thyroid grafting is also employed for the treatment of cretinism with the same results and same shortcomings and is carried out in the same way. It is especially with a view to more permanent results, that

the successful application of this method is desirable, and symptoms have been kept in abeyance for some time by it. Sooner or later, however, they return, and the operation has to be repeated or the extract administered. If grafting, which is usually done into the peritoneal cavity, is practised, it should be preceded by a course of thyroid extract, and the symptoms of myxœdema removed as far as possible before the operation is done, usually for two or three months. The details of operative treatment are left to the surgeon.

The results of treatment are as marvelous as in myxœdema. They include not only the removal of the hideous deformity and the restoration of intellect, but also an increase in height, which, though not amounting to a restoration of the normal height, is still appreciable. The earlier treatment is commenced the more marked is its effect.

TUMORS OF THE THYROID.

The thyroid is subject to a variety of morbid growths, among which may be mentioned :—

(1) *Adenoma,* which occurs as an encapsulated growth, varying considerably in size. There may be nodules in both lobes. Metastases of growths resembling thyroid tissue are reported to have been found in the lungs and bones of the body.

(2) *Primary medullary* cancer, as a rare growth with a tendency to invade the trachea and œsophagus, developed from the epithelial cells of the follicles. *Secondary* cancer has also been reported.

(3) Tuberculosis, always supposed to be a possible but rare disease, has been found by Chiari less so than supposed, who found it in seven out of 100 post-mortems on persons who had had tuberculosis. Bruns refers to six cases of tuberculous goitre.

(4) Hydatid disease,

(5) Syphilis, including gummy growths, and even

(6) Actinomycosis have been found in the thyroid.

Abscess of the thyroid is an occasional event.

The **treatment** of these abnormalities is surgical, except in the case of syphilis, which demands the usual antisyphilitic remedies.

SECTION VII.

DISEASES OF THE URINARY ORGANS.

GENERAL REMARKS ON ALBUMINURIA.

The importance of the relation of albuminuria to diseases of the urinary organs makes its consideration an absolutely essential preliminary to a study of these diseases.

Extra Renal Albuminuria.

It is well known that albumin may enter the urine from other sources than the secreting substance of the kidney. The pelvis of the organ, the ureters, the bladder, and the urethra, and in the female the vagina and uterus in addition, are these sources. In all of them except the uterus it is almost invariably the serum of *pus* formed during inflammation of their mucous surfaces which furnishes the albumin. The presence of pus-corpuscles, therefore, in sufficient number in the urine is usually sufficient to explain the source of such albumin, which is, moreover, usually small in quantity, never more than about $\frac{1}{10}$ the bulk of urine even with the most copious sediment of pus. It must not be overlooked, however, that the two sources, that of kidney disease itself and the mucous surfaces, referred to may coexist, in which event careful microscopic examination will sooner or later discover tube-casts, while the quantity of albumin will be larger than can be accounted for by the presence of pus alone.

Menstrual or lochial blood need only be referred to as sources of albumin in the urine hardly likely to be overlooked by any physician; while hemorrhage from any one of the mucous surfaces referred to, as well as the kidney itself, would be a source of albuminuria. It is usually, though not always, comparatively easy to determine whether a hemorrhage has its source in the kidney on the one hand or the mucous membranes above mentioned on the other. In the former, coagula are rarely present, for the blood entering the pelvis of the kidney slowly becomes intimately mixed with the urine. It imparts to it, too, when acid in reaction a *smoky hue* which is very characteristic. The coloring-matter of the corpuscles is mostly dissolved out by the urine which is thus tinged, and on standing the stroma of the corpuscles sinks to the bottom in the shape of a brownish sediment. The microscope reveals

these corpuscles shrunken, almost colorless, and often crenated. I have said that the smoky hue is present only in acid urine. When the latter is alkalinized, either by spontaneous or artificial change in reaction, it assumes a brighter red hue, the degree of which depends upon the quantity of blood. The same cause, acidity, produces the smoky hue of blood which is vomited, and therefore mixed with gastric juice. When blood comes from the pelvis of the kidney or the ureter in any quantity, coagula which are moulds of the ureter are sometimes found, the descent of which is often attended with severe pain.

Another source of albuminous urine, though not likely to cause error, should be mentioned, viz., the so-called chylous urine, or chyluria, in which, in consequence of some as yet imperfectly understood communication between the lymphatic system and the urinary tracts, chyle enters the urine and imparts its physical and chemical characters thereto. These are a milk-white appearance, due to the presence of fat in a molecular state, and a large amount of albumin.

The kidney itself may be the seat of suppuration, and contribute through the pus thus added to an albuminuria.

The Immediate Cause of Renal Albuminuria.—Delayed circulation of the blood through the glomeruli of the kidney is doubtless of itself sufficient to cause albumin, an otherwise non-osmotic substance, to pass through the walls of the capillary blood-vessels, though it is more than probable that an altered state of the epithelium covering the capillaries also contributes a facility of transit. The necessary obstruction is produced by any cause which sufficiently resists the movement of the blood through the kidneys, whether it resides in the organ itself or the venous system beyond it, whence the albuminuria which so often attends extreme valvular disease of the heart. The comparative smallness of such albuminuria as contrasted with that in parenchymatous nephritis, where the renal cells are an early seat of change, is strong evidence in favor of some active participation of epithelial change in causing albuminuria. In chronic albuminuria another important influence operates to facilitate the transudation of albumin. This is the hydræmic state of the blood, which is itself a consequence of albuminuria.

Renal albuminuria also occurs as a secondary symptom in diseases other than renal. First may be mentioned the albuminuria of fever, such as that of typhoid fever, small-pox, etc., and even in local inflammations other than renal, attended with much fever, as pneumonitis. The albuminuria of diphtheria and scarlatina, due to an intercurrent parenchymatous nephritis, is, of course, not referred to. The febrile albuminuria alluded to is not usually large, and disappears with the decline of the disease. It is in great measure the result of irritation of the kidney by the infectious agent; possibly also, in part, the result of diminished cardiac force with which the blood is driven through the kidneys and of the resulting turgor.

Other conditions in which albuminuria thus occurs are anæmia, leucæmia, diarrhœa, cholera, lead-colic, also certain conditions of the brain and spinal cord, including hemorrhages into the brain, meningitis, epilepsy, tetanus, and others. In all of these there is probably directly or indirectly through the nervous system diminished arterial pressure.

Physiological or Functional Albuminuria.

The presence of a physiological or functional albuminuria at the present day is generally conceded. By it is meant an albuminuria which is associated with no other symptoms. There are no tube-casts or feeling of ill health. Such albuminurias are often discovered accidentally, especially by examiners for life insurance. Much care should be exercised in concluding upon the nature of an albuminuria suspected to be functional. In the first place, it should be small, not exceeding $\frac{1}{10}$ the bulk of urine tested, and though it is not necessary that it should be absent on rising, yet it is a strong point in favor of the functional nature if it is absent at this time and present only after some exertion has been made or on taking food. No tube-casts should be in the urine, the urea should be in sufficient quantity, there should be no retinal change, no hypertrophy of the left ventricle, no high tension to the pulse, nor even a suggestion of dropsy. And this condition should be maintained over a considerable length of time before the conclusion is arrived at that we have to do with a harmless functional albuminuria.

Other proteid matters are sometimes found in the urine, such as globulin, mucin, pepton, and hemialbumose, but except in the case of globulin their clinical significance is not sufficiently determined to justify their further consideration in a text-book. A certain amount of *globulin* is always associated with albumin. In an ordinary serum albuminuria the ratio commonly maintained is ten to 18 of albumin to one of globulin, the ratio in the blood being one of serum albumin to 1½ to three of globulin. A like ratio holds for pus.

This is, however, at times exceeded, especially in the case of the amyloid kidney, where I regard its presence as of diagnostic value.

To Test for Albumin.—The test which is at the same time the most delicate and reliable is the heat-and-acid test. It is best applied in the following manner :—

The Heat-and-Acid Test.—A suitable quantity of urine, filtered if not clear, is poured into a test-tube, say to a depth of five cm. (two inches), and boiled over a Bunsen flame or spirit-lamp. Then nitric acid is added, drop by drop, until ten to 15 drops are added to this quantity of urine. Any precipitate produced in the act of boiling which is dissolved by the acid is phosphates. After the total quantity of acid has been added, the tube is placed aside for half an hour, and if at the end of this time no further precipitate appears, the specimen may be declared for clinical purposes free from albumin. It is very important that the acid should not be added first, as is sometimes directed, for it often happens that where a considerable amount of albumin is present, acid albumin is thus formed which is not precipitable by heat, and thus such quantities of albumin may elude detection. The test of heat and acid thus applied is really more delicate than the contact method with nitric acid, recognizing, as it does, smaller quantities of albumin.

The Contact Method with Nitric Acid, or Heller's Test.—On the other hand, the contact method with nitric acid is free from the error possibly

occasioned by acid albumin to which the heat-and-acid test is subject. This method, also known as Heller's, is best done by placing a sufficient quantity of nitric acid in the bottom of a test-tube and overlaying it gently with urine. If albumin is present, a sharp white line will promptly form above the acid. A dark, reddish-brown color-line which forms in concentrated urines at this point will not, of course, be confounded with albumin. Furthermore, in concentrated urines a layer of acid urates, dirty white, is sometimes found at the junction between the two fluids. It behaves very differently, however, from the sharp white line of albumin, since it soon begins to rise above the surface, while the albumin remains at its position. Further, this less definite layer is promptly dissipated on the application of a moderate heat. When these two tests are jointly used in the way described, it is impossible for such an amount of albumin as is of clinical significance to elude detection. It is true that picric acid solution, Tanret's iodo-mercuric solution, and one or two others are slightly more delicate than the heat and acid, but they are subject to a now generally acknowledged source of error, the precipitation of mucin and of the vegetable alkaloids of which quinine is the type, and which always appear in the urine when administered in full doses.

The Picric Acid Test.—As a confirmatory test, however, the picric acid solution is very useful, and if its application is negative we may feel assured that there is no albumin present, while a delicate response is doubtful for the reason named. The picric acid test is also best applied in the contact method, the urine being placed in the tube first and overlaid with the saturated picric solution, because the picric acid is commonly lighter than the urine to be tested. When they are of the same specific gravity it is not easy to maintain a separation at the line of contact, but a careful examination of the fused portions will recognize a diminished transparency due to either mucin or albumin. A saturated solution of picric acid should be always kept on hand for the control purposes named.

To Test for Globulin.—Globulin may be separated by Poehl's method as follows :—

Render the urine slightly alkaline by ammonium hydrate, and after several hours filter to separate the phosphates. Then add solution of ammonium sulphate in the proportion of one volume to one volume of the filtrate. If a precipitate forms it is globulin.

DISEASES OF THE KIDNEY.

DERANGEMENTS OF CIRCULATION.

Active Congestion.

Etiology.—Active congestion occurs as the result of poisoning by cantharides or arsenic, overdoses of turpentine, copaiba, cubebs, and carbolic acid. It is identical with the first stage of acute nephritis, however caused. The kidney in acute fever has usually been considered as actively congested, but recent experiments by Walter Mendelsohn go to show that it is really anæmic, small, pale, and bloodless. So that the kidney is really comparable to the anæmic kidney of cholera, in which, too, there are albuminuria and nutritional changes of a degenerative kind analogous to the cloudy swelling affecting the renal cells in acute fever, but more advanced in degree.

As to **morbid anatomy,** the kidney of active congestion is slightly enlarged, swollen, and, after removal of the capsule, brown or mottled. On section the cortex is wider and darker than in health, the blood-vessels over-full, the Malpighian bodies distended, the cells the seat of cloudy swelling. The medulla is less intensely red and sharply defined from the cortex.

Of symptoms there are none except a scanty urine of high specific gravity and high color.

Passive Congestion or Cyanotic Induration.

Etiology and Pathogeny.—While any agency which obstructs the movement of the blood through the kidney may cause passive congestion, the causes encountered in actual practice are mostly limited to valvular disease of the heart and chronic pulmonary disease involving considerable areas of the lung, such as emphysema, interstitial pneumonia, and pleurisy, with extensive effusion or marked adhesions. Tubercular phthisis is a less common cause because of the impaired nutrition and small quantity of blood. Pressure on the renal veins by tumors, the pregnant uterus, or ascitic fluid acts similarly.

In either event the mechanism of its production is the same. The blood is crowded into the venous side of the vascular system. In mitral insufficiency the blood is regurgitated from the left ventricle into the corresponding auricle, and thence into the lungs; the latter organs become engorged, and again resist the entrance of blood from the right side of the heart, whence it is backed into the right auricle and valveless vena cava. The smaller veins of the extremities at first resist the encroachment by means of their valves. But the veins of the abdominal viscera, including the liver and the kidneys, are without valves, and are the first, therefore, to receive the brunt of the stagnation, and they become engorged with blood. In pulmonary or pleural disease the obstruction begins in the lungs instead of the heart, but the mechanism is the same. Pressure by tumor on the renal vein is even more direct.

Morbid Anatomy.—The kidney of cyanotic induration or passive congestion is hard, firm, and bluish-red as to its external surface. In the earlier stages it is enlarged simply from the presence of the large amount of blood detained in its vessels. The stellate veins are unusually distinct. The capsule strips off easily, and on section the enlargement is found to involve the cortex, but the veins of both cortex and medulla are engorged, that of the straight veins causing the medulla to appear darker in hue than the cortex. The Malpighian bodies, on the other hand, are not always engorged. The cut surface of the kidney is moist and succulent, but the microscope reveals no further changes, either in the cortex or the medulla, the epithelium being unchanged.

After some duration the kidney is slightly if at all larger than the normal organ, though rarely smaller. The other superficial characters of hardness, smoothness, and bluish-red color, however, remain. Sometimes there appears a slight tendency to lobulation. At this stage the capsule does not strip off quite as easily as usual, but may drag small portions of the parenchyma with it. There may then be seen some shallow depressions. On section, the vessels are less turgid, and the relations of the cortex and medulla are not much altered. There may be a slight overgrowth of interstitial tissue and a small-celled infiltration between the tubules. Malpighian bodies are sometimes shrivelled and the epithelium of the tubules in places granular and slightly fatty.

Symptoms.—These are primarily those of the diseases of which it is the consequence, of which I will repeat only anasarca because of its importance. To these are superadded *scanty urine* of *high specific gravity*, containing usually a small amount of *albumin* and a few *small hyaline casts.*

The dropsy is first of the lower extremities, in the area drained by the inferior vena cava. There also occur, however, effusions into the pleural sac and peritoneum, and the hands and arms may be involved.

The urine is scanty, and of high specific gravity, often 1030 to 1035, and even higher. It is turbid with urates, depositing a copious sediment of them and of uric acid. The albumin is usually small in quantity, but may become larger if the obstruction to the movement of the blood is great. The casts are small, transparent, or faintly granular, and not numerous—indeed, often absent. Blood is rarely present; indeed, its presence may be considered evidence of a superadded nephritis. The solids are secreted in normal amount. In fact, such kidneys can apparently be restored at any time by proper treatment to their normal function. *Uræmia* is *exceedingly rare.* Very rarely does it happen that an interstitial nephritis is induced.

Diagnosis.—Passive congestion exists to a certain degree in all cases of valvular disease without compensation, but higher degrees may be suspected when the urine becomes scanty and albuminous, and when all the symptoms of the cardiac affection become aggravated. When the physician sees the patient after the symptoms of passive congestion have become marked, it is often a nice question to decide which of the two conditions are primary, the cardiac or the renal condition; but this subject will be further considered in treating of the relations between kidney disease and heart disease.

Prognosis.—With the addition of the renal complication, the inconveniences and annoyances of the cardiac disease become greatly aggravated, while the difficulties in the way of successful treatment are greater. Yet the results which sometimes follow appropriate and energetic treatment and the substitution of favorable for unfavorable hygienic surroundings, such as succeed the admission of a neglected outcast to the wards of a hospital, are often astonishing. Under these circumstances it is not unusual for the dropsy to decline, the albumin and casts to disappear, and the patient to be restored to comparative comfort, without, however, any change in the original lesion, which upon the slightest provocation may re-excite all the symptoms.

Treatment.—As intimated under prognosis, the substitution of favorable for unfavorable hygienic surroundings, if the former exist, is the primary requisite. Shelter, warmth, rest, good food, are indispensable. After this digitalis is the sheet-anchor for evident reasons. We have here to deal with a dilated, weak, failing heart, unable to drive the blood forward. Its power must be increased, and we have a remedy capable of doing this in digitalis. Sufficient doses must, however, be given, whether of the tincture, powder, or infusion. A half ounce (15 c. c.) of the infusion may be given every four hours to an adult, of the tincture not less than ten minims (0.65 c. c.), or 20 drops, to be reduced when diuresis sets in. Under such doses, if the cardiac disease is not too advanced, the urine may increase, become clear, its albumin and casts decline, and with these also the dropsy, dyspnœa, and restless, sleepless nights. All that has been said under the treatment of cardiac valvular disease of substitutes for digitalis is applicable here, and the reader is referred to that section.

Due attention must also be paid to the bowels for the sake of securing prompt action of the diuretics as well as to secure the elimination which their free action secures. The hydragogue cathartics, such as elaterium and the salines, are often excellent adjuvants.

ACUTE PARENCHYMATOUS NEPHRITIS.

SYNONYMS.—*Acute Nephritis; Acute Diffuse Nephritis; Acute Desquamative Nephritis; Acute Tubal Nephritis; Acute Bright's Disease; Acute Catarrhal Nephritis; Croupous Nephritis; Albuminous Nephritis; Hemorrhagic Nephritis; Acute Albuminuria; Acute Renal Dropsy.*

Definition.—Acute parenchymatous nephritis is an acute inflammation of the kidney, the tubular, vascular, and interstitial tissues being simultaneously affected in different degrees in different cases.

Etiology.—Most cases of acute parenchymatous nephritis are caused by scarlet fever or diphtheria, and occur, therefore, in children. A certain number originate in exposure to cold, especially cold and moisture, while the body is warm and perspiring. The latter cause is particularly potent if the person be fatigued or exhausted.

When acute nephritis supervenes on scarlet fever, it is usually not until the end of the second week, often when convalescence is well estab-

lished. It may occur as early as the tenth day, seldom, if ever, later than the thirty-first.

Other grave infectious diseases, as small-pox, acute endocarditis and acute articular rheumatism, typhus and typhoid fevers, pneumonia, malaria, are also occasional causes. Measles, erysipelas, pyæmia, jaundice, and diabetes have been known to cause it. Skin diseases, as well as extensive burns of the skin, are acknowledged causes; the former rarely, but the latter almost always if the burns be sufficiently extensive. Simple follicular tonsillitis is also a not infrequent cause. I have known an abscess in the thigh succeeding the lodgment of a splinter in the toe to be followed by typical acute nephritis. Pregnancy is the cause of a good many cases of acute parenchymatous nephritis, to which, in turn, most cases of puerperal convulsions are due.

Most cases of acute nephritis due to other causes than scarlatina, diphtheria, cold, and pregnancy are mild in degree; and even in cases due to pregnancy, if the patient is once through with her confinement, recovery is usually rapid. In looking for the evidence of nephritis in acute infectious diseases, it must not be forgotten that intense febrile movement may cause albuminuria, independently of any structural change in the kidney due to the toxic agent. When thus caused the albuminuria is always small.

Certain specific poisons of vegetable and mineral origin are capable of producing acute nephritis. Among the best known of these substances are cantharides, turpentine, oil of mustard, worm-seed oil, and phosphorus; in a less degree the mineral acids, arsenic, nitrate of silver, lead, and mercury. Very large quantities of alcohol when swallowed have caused acute nephritis.

The microbic origin of nephritis has also been invoked. At least pathogenic organisms have been found in the urine of cases pure cultures from which by Mannaberg produced intense nephritis when injected into the blood of animals.

Oertel claimed fifteen years ago that in renal diseases following diphtheria he found "great numbers of micrococci and exuberant proliferations of the same," both in the renal tubes and Malpighian bodies; Heller that he had repeatedly found the blood-vessels and their branches in acutely inflamed and swollen kidneys from cases of pyæmia greatly dilated and plugged with masses, which under low powers presented a peculiar greyish-yellow appearance, and with high powers were found to consist of extremely minute, highly refracting granular particles. These particles he considered spherical bacteria, and the resulting masses bacteria-emboli.

As may be inferred from the etiology, acute nephritis is often a disease of early age, although when caused by cold or any one of the causes named except scarlatina, it is as more likely to affect adults as these latter are more frequently subjected to such causes. It is rare after forty, almost unknown after fifty. More males are attacked than females in adult life, evidently because they are more frequently exposed to the causes. But even in childhood there is a slight preponderance of cases in boys affected, which can hardly be thus accounted for.

Morbid Anatomy.—This varies with the stage of the disease as well as its severity. In the first place, as ordinarily caused, it is symmetrical, both organs being alike involved. The alterations may be so trifling as not to be recognizable by the naked eye. But the kidneys are generally sooner or later enlarged, in the latter stages always, sometimes to more than twice their normal volume, and they may weigh from eight to 12 ounces (240 to 360 gm.), those of children reaching the former and those of adults the latter.

The capsule strips off easily, without dragging any of the parenchyma with it. Bereft of its capsule, the kidney itself is softer, inelastic, doughy. Its surface is smooth and exhibits a peculiar mottled appearance, which is due to the fact that the little circlets of veins which form the boundary of the lobules are distinctly injected, while the area included by each circlet is paler than in health, and in the more advanced stages even yellowish-white in color. This "irregular mixture of congestion and anæmia," as Sir George Johnson early called it, is further contributed to by the injection of other veins indistinct in health. Spots of hemorrhagic extravasation may also be found scattered over the surface.

On section it is evident that the enlargement is due to change in the cortex and the interpyramidal convoluted portion. The cut surface is smeared over with a dark red or chocolate-hued blood, but on scraping or washing it away the vessels are found injected like those of the surface, and between them the same paleness or yellowish-white hue. The Malpighian bodies are enlarged and distinct, dark red, sometimes pale. Punctiform hemorrhages may also be present, as on the surface of the organ. The pyramids are dark red.

Minute Changes.—These are confined almost solely to the cortex. They by no means always correspond in degree with what would be expected from the symptoms, being often entirely inadequate to explain them. The changes are *tubal*, *glomerular*, and *interstitial*.

I. *Tubal Changes.*—These vary a good deal with the stage of the disease. The earliest change assumed by the cells is cloudy swelling, a result of increased nutritive activity. In this state the cells are swollen and "cloudy" from a deposition of albuminous granules, which may obscure the nucleus. Although kidneys removed after death from cases of acute parenchymatous nephritis have, as a rule, advanced far beyond this stage, yet it is often possible to find points at which cloudy swelling exists alongside of more advanced stages, while alongside of these again may be tubes in which the epithelium is normal. As a result of the cloudy swelling, the cells are larger, and the tubes are therefore distended, broader than in health, but a stage later they are still more distended with granular cells, granular debris, and often red blood discs and leucocytes. Under a low power the tubules appear as black, more or less opaque lines. A closer examination of the *cells* at this stage, as obtained by scraping, shows them to be granular in various degrees. In some the nucleus is still visible, in others demonstrable by the aid of staining fluids only, and in others still, entirely obscured. Occasionally a few fat-drops may be present. In other situations the cells are so closely packed in the tubules that they cannot be differentiated, being apparently fused in

one continuous, dark, granular mass. It is to these tubules distended with granular cells and their debris, dark by transmitted light, but white by reflected, that the pale or white color seen between the injected blood-vessels is due. Casts of the uriniferous tubes are also found *in situ*, usually blood casts or small hyaline casts. Minute extravasations of blood visible to the naked eye have been referred to. They occupy the tubules, and in the "hemorrhagic" form the interstitial tissue.

II. *Glomerular Changes.*—In all cases in which the nephritis is due to a toxic substance which enters the blood the glomerule suffers first. The capillaries of the *tuft* are distended with blood, which bursts through into the Malpighian capsule, distending it with red blood corpuscles and leucocytes. In a more advanced stage the glomerules may be paler in consequence of the proliferation of the cells lining the capsule and covering the glomerule (glomerulo-nephritis).

These glomerule changes are present in almost all cases. They include swelling and desquamation of the capsular epithelium and an accumulation of cells in the interior of the capillaries, probably due to a proliferation of their endothelial lining or an accumulation of white blood cells; or thickening and hyaline degeneration of the capillary walls and their contents. These are especially frequent in nephritis after scarlet fever or diphtheria.

III. *Interstitial Changes.*—In mild cases there is no interstitial change, no formation or deposit of new material between the tubes. In others there is a serous transudate with a few leucocytes in most cases, and red blood discs. In severer cases there is a large outwandering of cells, and a small-celled infiltrate settles itself between the convoluted tubes and around the capsules. In cases of extreme severity a diffuse nephritis involving both tubes and intertubular tissue may be present from the outset. In such event the latter is uniformly infiltrated, or pervaded more intensely in certain places by leucocytes.

The epithelium lining of the straight tubes of the pyramids is unchanged, but the tubes themselves often contain cellular and granular material, which has descended from the convoluted tubes.

Serous infiltration and effusion are present when the patient is dropsical at the time of death. Among other tissues sometimes thus infiltrated are the membranes of the brain, constituting what is known as œdema of the brain.

The mucous membrane of the pelvis of the kidney may be injected but is otherwise unchanged.

Symptoms.—The mode of onset of acute nephritis is not uniform, but among the symptoms earliest noticed is slight *swelling* or *puffiness in the face*, below the eyes. This œdema rapidly extends to the upper extremities and trunk, and thence, if the disease does not abate, into the lower extremities and abdominal walls. In the male, the scrotum and prepuce are favorite seats of swelling. The great serous sacs are the last to fill with fluid in acute nephritis, although in bad cases ascites not unfrequently occurs, while there may also be transudation into the pleural and pericardial cavities. The degree assumed by the general anasarca

is sometimes enormous, resulting in the extremest distortion. The eyes may be actually closed by the swelling, and movement of the lower limbs rendered almost impossible. Dropsy does not always follow the order here named. Much depends upon the position of the patient. Thus, if he be upon his feet, the latter may be the first to swell, or if he be lying in the recumbent position, the back may be the seat of the first swelling. While dropsy is a very frequent symptom in acute nephritis, it is not, however, always present. It is more particularly in the nephritis after scarlet fever and exposure to cold that it is a decided and almost invariable symptom. After the other infectious diseases it is frequently absent.

Changes in the Urine.—Simultaneously with, and sometimes earlier than, the dropsical symptoms is a *diminution in the quantity and alteration in the quality of the urine*. The former may amount to actual suppression. The urine is darker than natural, and often smoke-hued from the effect of the natural acid reaction on a small quantity of blood. Should the urine become alkaline the color becomes a brighter red. The smoke-hue also becomes red if the quantity of blood is large, which is not often the case; but here again the peculiar tint returns if the blood is allowed to subside. If the urine is very small in quantity, and there is much sediment, the former is turbid and may have a brownish tinge. The blood may disappear, to return again.

The *specific gravity* of the urine at first is high, 1025 to 1030, mainly due to the diminished quantity, while the solids remain nearly normal. Later, if the symptoms abate, the specific gravity diminishes with the increase in the quantity; or if the disease lasts for any length of time, or passes over into the chronic form, a similar reduction in weight occurs; this may result in a specific gravity as low as 1010.

The chief alteration is the *presence of albumin.* This is generally copious, the urine often solidifying on the application of heat and acid, while it constantly contains more than half its bulk. This albumin is derived in part from the extravasated blood and in part is a result of the inflammatory action. If estimated by weight it will equal ½ to one per cent., and, in rare instances only, to 1.5 per cent.

Next in importance is the reduction in the twenty-four hours' secretion of *urea*, which is invariable until convalescence sets in. The percentage remains as high or higher than in health, but the twenty-four hours' quantity is diminished. There is a good deal of range, within the limits of health, in the quantity of urea eliminated in twenty-four hours—from 20 to 40 gm. (300 to 600 grains) in adults. More frequently the amount is reduced to ¼ or ½ the normal. The phosphates and chlorides are also reduced.

As to *sediment*, the urine of all cases of acute parenchymatous nephritis deposits a sediment which, in the early stages at least, is copious and brownish or reddish-brown in hue; later, it may diminish in amount and assume a lighter color. *Microscopical examination* reveals this deposit to be made up mainly of casts of the uriniferous tubules, free cells from these same tubules, blood corpuscles, red and colorless, and very constantly crystals of uric acid, together with granular urates. The casts include the varieties known as epithelial casts, blood casts, hyaline

casts, waxy and dark granular casts. Pus casts and numerous leucocytes are also sometimes present. The hyaline casts are probably pure fibrin. The epithelial consist of the same material, to which epithelial cells of the tubules are attached and blood casts have blood corpuscles caught in the coagulated exudate. The epithelium thus attached, as well as that which is found free in the urine, is variously altered. Some of the cells are merely the seat of cloudy swelling, others are decidedly granular, while others again are converted into compound granule-cells or granular fatty cells by complete fatty degeneration. Casts containing a few oil-drops may also be present; but much oil is not found until the case has continued for some time—in fact, become chronic.

Along with the diminished quantity of urine is often met a disposition to *frequent micturition*, the efforts at which are only partially successful, resulting in the emission of from a few drops to a tablespoonful. This frequent desire to pass water is a purely reflex symptom, the bladder being free from disease. It sometimes precedes, in point of time, all other symptoms. It is by no means constant.

Fever is not a marked symptom in acute nephritis; indeed, it is generally absent, unless as a part of the disease causing it. To a less degree the same is true of *pain*. It is mostly absent, and when present amounts only to a dull ache, as a rule. *Chilliness* and *rigors* sometimes introduce the disease. *Nausea* and *vomiting* are not infrequent in the beginning. Sometimes these symptoms usher in the disease.

The *pulse* is quite characteristically altered. While not materially changed in rate it exhibits, especially in sphygmogram, a decided increase in tension, as shown by the broader apex and diminished dicrotic element.

Uræmia.—At almost any time in the course of an acute nephritis the patient is liable to *uræmia*, and while the *train of nervous symptoms* usually known as *uræmic*, and ascribed to the accumulation of excrementitious substances in the blood, is not confined to this form of Bright's disease, and is by no means invariably present, it may be conveniently considered here. When present, however, it adds a phase of extreme gravity.

It is not unlikely that the loss of appetite, nausea and vomiting, and headache which sometimes usher in an attack of nephritis may be the result of retained excrementitious matter. At all events, these same symptoms may be the initial ones of a uræmia. Headache is often occipital, extending down the neck. To it is often superadded dizziness.

An early symptom is *drowsiness*, which may be sudden or gradual in its onset and may be slight or decided. From the latter degree the transition is easy to the next symptom, that of *coma*, from which the patient may or may not be temporarily aroused. Alternating with the latter may be epileptoid *convulsions*, which are the most alarming and dangerous symptom of Bright's disease. This is not always the succession of these symptoms. Convulsions may succeed drowsiness, but as often precede coma, and they may occur without warning. Indeed, there may be no suspicion of Bright's disease whatever until a convulsion suddenly happens. Drowsiness, in like manner, may be the first symptom

of the renal disease to attract attention, others being overlooked or possibly even absent. The convulsions exhibit every grade of movement, from the slightest twitching to the most violent epileptiform spasm.

Suppression of urine, an almost constant symptom, is often the initial one which should at once excite suspicion. Accompanying it is often a breath of urinous odor, and when vomiting accompanies scanty or suppressed urine, the vomited matters sometimes have a urinous odor, the elements of urine being thus supplementally eliminated.

Impairment of vision or *actual* blindness suddenly occurring is another symptom of acute uræmia which sometimes supervenes upon other symptoms, or it may itself usher in the complication. This blindness, it must be remembered, is something altogether different from that which is the result of organic retinal changes, which are rare in acute nephritis, but common in some of the chronic forms. It may be due to retinal hemorrhage but is often unassociated with demonstrable retinal change.

Itching of the skin is another symptom sometimes present in uræmia. It is probably due to the irritant action of the urea upon the nerves of the skin as it is being supplementally eliminated by that organ. That such increased elimination takes place is attested by that rare but still unquestioned occurrence, in which the entire integument is covered with a frost-like coating which has been found upon analysis to be crystals of pure urea.

Another symptom of uræmia, which belongs rather to the uræmia of chronic renal disease, is shortness of breath,—*uræmic asthma* it is called. This is an asthma which differs from bronchial asthma in the absence of spasmodic contraction of the bronchi, and its uræmic origin, at least in every case, I think, is extremely doubtful. I believe it is more frequently what I have described as cardiac asthma, due to heart-failure, the result of dilatation succeeding on hypertrophy. It may happen that true spasmodic asthma is produced by uræmia, but it must be exceedingly rare. To it alone, however, should the name uræmic asthma be given. Paroxysms of dyspnœa are also caused by œdema of the lung, which is not infrequent in acute nephritis. It is recognized by the presence of fine, moist râles. Of course, it is not impossible for nephritis to happen to an asthmatic, whose attacks would then occur as before, independent of the uræmic cause, or might be increased in frequency or rendered more unmanageable by the latter.

Cheyne-Stokes breathing is also a symptom of uræmia and may last for a long time. It is not necessarily associated with coma.

The question of *temperature* in uræmia has not been satisfactorily worked up. My own experience points to the absence of any elevation, with at times a tendency to subnormal. I am aware that studies have been published which go to show that an abnormally high temperature is characteristic, and this may be the case exceptionally. But in true, uncomplicated uræmia I believe in the majority of cases the temperature will not much exceed the normal. It is true that toward the very end there is commonly a rise, but this attends dissolution in so many diseases that it cannot be considered characteristic.

The *pulse* is often slow in uræmia before the appearance of severe symptoms, sometimes as infrequent as 40 to 50, but with severe symptoms it becomes frequent.

Acute mania and *delusional insanity* may also be symptoms; also rarely melancholia and paralysis, including hemiplegia and even monoplegia. These may occur independently of a convulsion or succeed it, in which event there may or may not be any coarse lesion found at necropsy. True uræmic palsies are of rare but undoubted occurrence.

The duration of acute nephritis is very variable—from a few days to several months, while the acute form may become chronic. The former class of cases are fatal, for none which recover do so in a few days. The most rapid usually require a month. As to the cases of longer duration, the possibility of recovery at any time cannot be denied, but nothing is better determined than that the longer the duration the more difficult the cure. Of course, such cases are no longer acute.

Complications.—These are not numerous in acute as contrasted with chronic Bright's disease, and some which are described as complications are not really such, but local symptoms. Thus, *œdema of the lungs* occurs as a part of the general tendency to dropsy, and may be a grave symptom resulting in death by suffocation. It is not the result of an intercurrent bronchitis. *Pneumonia*, on the other hand, is an occasional true complication. *Inflammation of the serous membranes* is more truly a complication, but not every case in which there is effusion into a serous cavity is of such a nature. Such effusions are local dropsies. But inflammation of serous membranes is rather more prone to occur, while the exudate may assume a purulent character, thus also increasing the gravity of the case. Pleurisy is the most frequent form of this inflammation, pericarditis next, and peritonitis next. The tubercular origin of the graver forms of pleurisy occurring in Bright's disease has been suggested.

Hypertrophy of the left ventricle is not a frequent complication of acute nephritis. It is a well-recognized one of chronic Bright's disease. Time is an essential condition to its production. It is not, therefore, until the kidney disease has existed for some time that it can ordinarily occur. It occasionally happens, however, that the hypertrophy occurs earlier. Thus Dickinson reports a case recognized at eight weeks, and v. Leube one at ten days succeeding the first symptom. The infallible sign of hypertrophy is sharp accentuation of the aortic second sound with or without demonstrable enlargement of the normal area of dulness.

Allusion has been made to the gastric symptoms which very commonly attend acute nephritis, especially after scarlet fever. Samuel Fenwick* and Wilson Fox† have shown that these may be associated with organic changes in the structure of the stomach. Dr. Fenwick characterizes these as gastritis, as evidenced by increased vascularity of the mucous membrane, distention of the tubes by a confused mass of cells and gran-

*Fenwick, Samuel, "The Morbid States of the Stomach and Duodenum," 1868, p. 177.

†Fox, Wilson, Medico-Chirurg. Transac., Vol. XLI, p. 361.

ular matter, and occasional thickening of the basement membrane. To these Dr. Fox has added thickening of the intertubular tissue.

Notwithstanding the frequency of convulsions in acute nephritis, structural *alterations* in the *brain* are almost unknown. Apoplectic effusions do not occur in most cases, probably because of the comparative structural integrity of the blood-vessels of the brain in the young, in whom the disease mainly occurs. In like manner the blindness which not infrequently occurs as a symptom of uræmia is unattended by retinal changes, nor does that condition known as albuminuric retinitis occur in acute parenchymatous nephritis except with the extremest rarity.

Diagnosis.—The diagnosis of acute parenchymatous nephritis is ordinarily quite easy. The previous history, the usually easily recognizable cause, the suddenness of the attack, the scanty and bloody urine with its high specific gravity, the copious albuminuria, the blood and epithelial and dark granular casts, the blood corpuscles, free epithelium and granular fatty cells in the urine,—these are a combination of symptoms which admit of only one interpretation. At a later stage the absence of one or more of these symptoms may somewhat increase the difficulty, but it is scarcely possible to err if those which remain are duly considered. It must be remembered also that an acute condition, such as this described, may supervene upon any one of the chronic forms of Bright's disease to be described, and this may give rise to some difficulty of diagnosis, but if there be hypertrophy of the left ventricle it is likely that there was chronic disease before; in the latter case, too, there is apt to have been anæmia existing for some time, previous œdema, headache, and other symptoms of chronic Bright's disease.

Febrile albuminuria is quite often mistaken for acute nephritis by those who have had little experience, though the distinction is easy. In pure febrile albuminuria the quantity of albumin is very small, and while there may rarely be a few hyaline casts there are no blood discs, no epithelial casts. The absence of dropsy is of no significance, for in the acute nephritis of the infectious diseases, except scarlet fever, there very seldom is dropsy. There is often febrile albuminuria in scarlet fever which is quite different from the nephritis occasioned by this disease. It occurs early, and in this stage the other features of febrile albuminuria obtain, while the scarlatinal nephritis does not come on, as already stated, until after the end of the second week.

While the glomerule changes referred to are more usual in scarlatinal nephritis, there is no certain way of recognizing such condition, and the term glomerulo-nephritis which is applied to the nephritis associated with these changes is scarcely justified from the clinical standpoint, because there are no symptoms by which it can be recognized.

The diagnosis of uræmia, commonly easy, is sometimes difficult. This is especially the case when instead of the usual complex list of symptoms appearing there are but one or two. By no means every nervous manifestation coincident with Bright's disease is uræmic, and I am inclined to believe that some are ascribed to it which should not be. Such are localized convulsions and hemiplegias, which are much more likely to be due to some anatomical lesion in the brain. Given,

however, a case of sudden convulsions or coma or even muscular twitching, if it is associated with scanty urine and diminished urea excretion it may be ascribed to uræmia provided there is no cause which will explain it more satisfactorily. Uræmia has been mistaken for opium and alcoholic intoxication, and it must be admitted that the coma in all three is very much alike. But one need only be forewarned to prevent such error. In opium-poisoning the pupils are contracted, in alcoholism they are dilated, in uræmia they vary.

Prognosis.—Grave as is justly considered this disease, recoveries from it are numerous and the prognosis is generally favorable. Even without treatment cases may recover, and more recoveries follow a judicious treatment. The prognosis should, however, always be guarded, as insidious causes may produce death when it is least expected. Among the most important of these is uræmia.

Bartels says that death from uræmia has never occurred in his experience, except when the disease has resulted from scarlatina or diphtheria; but Dickinson narrates a fatal case resulting from exposure, in which death was preceded by coma and other symptoms of evident uræmic origin, and I believe I have seen such.

Pulmonary œdema is a cause of sudden death, the patient drowning, as it were, in his own secretions. Its onset is characterized by shortness of breath, frothy expectoration, and abundant small râles. Purulent exudation into the serous cavities may also precipitate death.

The symptoms of gravest import are therefore those of uræmia, manifested in any one or all the various ways, the presence of any of the complications alluded to, and especially suppression of urine. Cases should not, however, be despaired of even when there is complete suppression of urine. Always, however, suppression of urine is the gravest of symptoms, and death generally ensues within a couple of days after it sets in. The possibility of sudden death should always be borne in mind, and mentioned to the relatives of the patient, although the number of cases in which this occurs is not very great. Of course, the longer the duration of the case, the less the likelihood of recovery.

Treatment.—Many cases of acute nephritis recover under the conditions of rest, quietude, and warmth. And it is further certain that, whatever other means of treatment are used, these three conditions are absolutely necessary to recovery. A patient with acute Bright's disease, therefore, whatever its mode of origin, should be put to bed, kept quiet, and warmly covered. We should seldom, however, be satisfied with this treatment alone. The selection of other measures will depend somewhat upon the severity of the case. If the urine be suppressed, dry cups, or in severe cases cut cups, to the loins may divert the blood and relieve the stagnation which always exists in the acutely inflamed kidney. Cups should be followed by a warm poultice to the same region, which, indeed, should be used under any circumstances, whether the cupping is necessary or not. Poultices should, therefore, always be resorted to, and if the symptoms are at all severe,—that is, where there is complete or almost total suppression of urine, nausea, headache, or delirium,—should be preceded by cupping. Dry cups should not be

allowed to remain on one spot longer than to secure a bright redness, after which they must be withdrawn or moved to another spot in the vicinity. By allowing them to remain too long, the blood is stagnated in the capillaries, its onward movement prevented, and there is, therefore, no derivation of blood from the involved organ.

The above means have for their object the direct relief of the congestion of the kidney. This is further accomplished by purgation, which supplements the action of the kidney. But a purgative is early employed not more for this purpose than to promote the action of other remedies. Absorption is slow when the blood-vessels are congested and there is a sluggish current. The cathartic relieves this turgor and after its effect prompt absorption and action of other remedies may be looked for. The purgative most suitable is a saline. A simple dose of bitartrate of potassium, or simple magnesia for children, or citrate of magnesium, or epsom salts for adults will be sufficient. The indication is to get a watery stool as soon as possible. In view of the fact that the stomach is often sensitive, it is desirable to use an aperient which is not nauseous or irritating, and to this end some one of the delicate effervescing preparations so common in modern pharmacy may be used.

Next, or simultaneously, the action of the skin should be promoted. This is done by maintaining warmth and avoiding cold, as already insisted upon. But we are not confined to these protecting measures. The skin may be made to do the work of the kidney itself, and thus one of the most alarming dangers of Bright's disease, uræmic intoxication, averted, while at the same time the congestion of the kidney is also relieved. The class of remedies which produces this action are diaphoretics (the warmth described is one of these); and, of the simple remedies, none is better than the ordinary sweet spirit of nitre, especially if it be combined with neutral mixture and small doses of ipecacuanha. If more active measures are required, some one of the preparations of jaborandi may be used, the dose varying with the effect it is desired to obtain. If moderate diaphoresis only is desired, doses of from ten to 15 minims (0.65 to one gm.) of the fluid extract may be given to adults every two hours, and increased, if necessary, until the effect is brought about. To children, five to ten minims may be given in the same manner. Or pilocarpin may be given in doses of $\frac{1}{24}$ to $\frac{1}{12}$ grain (0.0027 to 0.0054 gm.). The further use of this important remedy will be again referred to in treating uræmia.

Another method of accomplishing the same end is by warm baths, or, better still, by the warm pack, in which the patient is wrapped in a wet sheet and then enveloped in a sufficient number of blankets. Perspiration is thus copiously induced, and when thus caused is agreeable, and never attended by the faintness which sometimes follows the use of the hot-air bath. In an ordinary severe case of acute Bright's disease, a single pack of this kind will often remove all urgent symptoms and happily inaugurate the convalescence. It may, however, be repeated daily if necessary.

Diuretics are, perhaps, the first means thought of by most practitioners in the treatment of Bright's disease, and they are indicated where

there is dropsy and scanty urine. Digitalis is the diuretic most to be relied upon. It is necessary, however, to have a reliable preparation, and unless one is sure of the quality of the tincture it is best to use a freshly prepared infusion. I have already explained on p. 542 why I believe more efficient results are obtained from the infusion than the tincture. Digitalis should therefore be given in sufficient doses,—½ to one fluidrachm (two to four c. c.) of the infusion to children, and two fluidrachms to half fluidounce (eight to 16 c. c.) to adults,—repeated every three hours until an appreciable effect is produced on the rate of the pulse, when it should be diminished. Not until then can we look for a diuretic action. I prefer at first to give it alone. Later it may be combined with acetate, citrate, and bitartrate of potassium. The diuretic action of these salts probably depends upon the impetus they give to osmosis of fluids holding them in solution, thus filling the blood-vessels, which in their turn give out water to flush the kidney. To adults 20 grains (1.3 gm.) of either may be given every two or three hours freely diluted, because water itself is an excellent diuretic; five to ten grains (0.32 to 0.648 gm.) to children as often. An important object, too, is to maintain an alkaline urine, which tends to dissolve exudates. For this purpose the alkaline mineral waters are also useful, or what is commonly known as cream-of-tartar tea may be drank instead of water. A teaspoonful of potassium bitartrate is put into a pint of boiling water and taken cold as drink is wanted.

Another admirable diuretic combination, including all of these elements, is Trousseau's diuretic wine, which consists of:—

℞. Junip. Contus., ... ℥x (40 gm.)
Pulv. Digitalis, ... ℥ij (8 gm.)
Pulv. Scillæ, ... ℥j (4 gm.)
Vin. Xerici, ... Oj (½ liter)
Macerate for four days and add
Potas. Acetatis, ... ℥iij (12 gm.).
Express and filter.
S.—Tablespoonful three times a day for an adult.

Infusion of digitalis may also be used in the shape of fomentations. Cloths wrung out in hot infusion of digitalis and laid over the abdomen of the patient have been known to produce diuresis when all other measures have failed.

The diet of patients with acute Bright's disease should be of the simplest and easiest of digestion and should contain a minimum of nitrogen. The irritability of the stomach in this disease has been alluded to, and it is important that food should be adapted to it. Milk may be considered the typical food, not merely because of its easy assimilation and nutritious character, but because there is abundant testimony to prove that albuminuria diminishes under its use, while the amount of nitrogen contributed to the blood is less than by animal flesh. The combination of lime-water, and still better of carbonated water or Vichy, with milk is an eminently suitable one. While solid animal food is not to be recommended, weak animal broths may be permitted to break up the monotony of a pure milk diet. *Beef teas and extracts should be prohibited as harmful.* Rice and farinaceous preparations generally are suitable adjuvants to the milk diet.

Treatment of Acute Uræmia.—The alarming and dangerous character of the symptoms of this condition demand a separate consideration of the measures required in their treatment. The treatment which has just been described is such as would be called for by an ordinary case of acute nephritis of a decided character. The tendency of it will be to prevent the retention of those effete matters, whatever their precise nature, which constitute the cause of uræmia. But all efforts in this direction sometimes fail, and we are called upon to contend with convulsions or coma, or more frequently both in alternation. How shall they be met? The indication has already been explained. Elimination is demanded. The kidneys are not acting and the secretion of urine is suppressed. There remain, therefore, but the bowels and skin to operate upon. But the patient is unconscious and cannot swallow voluntarily. Such remedies must therefore be used as do not require his co-operation. These are *croton oil* and *elaterium.* Of the former two drops, slightly diluted with plain oil or glycerin, may be carried into the back part of the throat, or in case of extreme necessity, undiluted, may be introduced into the mouth, whence it is quickly absorbed. Its operation may be facilitated by a rectal injection. Of elaterium a quarter of a grain in solution may be administered by the mouth.

In like manner the skin may be made to substitute the action of the kidney. The vapor or hot-air bath or hot pack should at once be availed of. The vapor may be conveyed by a pipe two or three inches in diameter, carried from a vessel containing water, under which a spirit-lamp is placed, under the bed-clothing, the patient being well covered with a mackintosh and blanket, excepting the head. An ordinary rain-spout may be used. Hot air may be similarly conveyed, but does not act as quickly. Its action may be favored by moistening the skin. The hot pack is also very efficient and perhaps less enervating.

Jaborandi or its active principle, pilocarpin, may be used, the latter preferably because available hypodermically. One-third grain (0.02 gm.) of the muriate may be thus administered, and if perspiration does not set in in a half-hour it may be repeated. Its action is also greatly facilitated by warmth applied to the patient. A freshly-prepared infusion may be injected into the rectum with almost equally prompt results. Four ounces (120 c. c.) of hot water should be poured on a drachm (four gm.) of jaborandi leaves, and, when sufficiently cool, strained and injected. The doses here mentioned are intended for adults. Where less urgency is required, a fluidrachm (four c. c.) may be inspissated and made into a suppository. Should œdema of the lungs occur, it may be overcome by the hypodermic injection of atropin, $\frac{1}{60}$ grain (0.001 gm.).

If the convulsions continue, blood-letting may be practised, or it may be done at once if the patient is not very feeble. No one doubts the efficiency of bleeding in puerperal convulsions, and if puerperal convulsions are uræmic, as they are, with few exceptions, then bleeding should be of service in the uræmic convulsions of acute Bright's disease.

The hydrate of chloral should not be forgotten; indeed, it is one of the most valuable remedies for the convulsion and should be one of the first measures tried. In the case of an adult, a drachm (four gm.) in

solution may be injected into the rectum; 15 to 30 grains (one to two gm.) for a child. Its use is sometimes followed by the promptest favorable results. Chloroform may also be used to control the convulsion while the eliminating measures are acting.

The use of opium requires to be alluded to. The caution which has always been suggested in its use I believe to be, in the main, a wholesome one, and I should prefer to produce hypnotic, sedative, and antispasmodic effects by chloral and the bromides whenever it is possible. At the same time there can be no doubt that opium is less harmful in acute nephritis than in chronic, and especially chronically contracted kidney, and when other measures fail to control convulsions it may be used cautiously. It was in the convulsions of acute nephritis that the late Professor Loomis, of New York City, recommended it, although its wider use has grown out of this suggestion. His practice was to treat cases of uræmic convulsions in acute nephritis with hypodermic injections of large doses of morphia, ½ grain (0.033 gm.) or more.

These same measures which have been detailed, excepting the general blood-letting and chloral, may also be employed in the treatment of suppression of urine or of obstinate dropsy without uræmic symptoms, with such modifications as circumstances may suggest, due regard being paid to the strength of the patient. They will be further referred to when discussing the treatment of the chronic forms of Bright's disease.

Sooner or later, also, in the treatment of acute parenchymatous nephritis supporting treatment is rendered necessary to repair the losses which the blood suffers by the albuminuria, and to some extent also by the depleting measures of treatment. These effects should indeed be anticipated by proper diet, tonics, quinine, especially iron, wine, malt liquors, whiskey, or brandy, as indicated. These measures will also be more particularly alluded to in the treatment of chronic Bright's disease.

Treatment of Complications.—Complications should be treated by remedies called for by such conditions independent of the renal cause. Effusions into the pleural cavities and abdomen are often best relieved by paracentesis, or aspiration; pneumonia and bronchitis by counter-irritation.

CHRONIC PARENCHYMATOUS NEPHRITIS.

SYNONYMS.—*Chronic Diffuse Nephritis; Chronic Tubal Nephritis; Chronic Catarrhal Nephritis; Large White Kidney.*

Definition.—A chronic diffuse hyperplastic process in the kidney, involving the epithelium, glomerules, and interstitial tissue.

Etiology.—This cannot always be traced. While it is frequently a continuation of acute nephritis, more frequently it originates *de novo*. To cases in the former category, scarlatina and pregnancy contribute the greater number. To the second class belong insidious cases, the cause of which is often not traceable. Habitual exposure to cold and dampness, such as residence in damp, cold houses, may cause some. Tubercular disease of the lungs is an undoubted cause. Great stress is

laid by German writers upon malarial poisoning as a cause. In this country it is not a frequent cause. One or two cases pretty well founded with others of more doubtful authenticity include my experience with this cause. S. C. Busey and I. E. Atkinson have, however, also assigned this as a cause in special papers devoted to the subject. It may be the case in more southern parts of the United States, where malarial poisoning is more intense than in the Middle States. Alcohol is a cause, and the nephritis of confirmed drunkards and the employees of breweries may be thus accounted for, though it cannot be denied that the exposure to which some of the former class are subjected may be responsible. Males, and of these young adults, are the more frequent subjects.

Morbid Anatomy.—There are two distinct stages in the morbid anatomy of chronic parenchymatous nephritis if the disease is of sufficient duration, viz., the stage of enlargement, represented by the *large white kidney*, and that of contraction, or the *fatty and contracting kidney*. A special variety is *chronic hemorrhagic nephritis*.

I. *Stage of Enlargement.*—There are few more striking objects in morbid anatomy than a typical example of the *large white kidney*. The kidney is large, smooth, white, or slightly tinged with yellow; weighs generally from seven to ten ounces (217 to 310 gm.), but is often much heavier. It is usually doughy, sometimes elastic in consistence. The capsule, which may be thinner than in health, strips off easily, but occasionally drags a little of the parenchyma with it. When the smooth white surface thus uncovered is examined, the little capillary circlets bounding the lobules in the normal organ are in some places indistinct, in others conspicuous; the same is true of the stellate veins of Verheyn. Numerous yellow specks are seen scattered over the surface. Hemorrhagic extravasations are also occasionally present, but very much more rarely than in the acute form. Alongside of these the greater translucency of more nearly normal areas results also in a characteristic mottled hue. *On section* it is evident that the enlargement resides altogether in the *cortex*, which is also anæmic, its intense white contrasting strongly with the pink hue of the cones, which, though paler than in health, are much less so than the cortex. Closer examination of the cortex reveals the same yellow specks found on the external surface. They contribute, with similar less decided alterations, to form a series of dull white striæ which alternate with somewhat broader translucent striæ radiating toward the surface; the former correspond to the area of the convoluted tubules and Malpighian bodies—the labyrinth,—the latter to that of the medullary rays.

The pelvis of the kidney in chronic parenchymatous nephritis is the seat of catarrhal swelling and a slight degree of hyperæmia.

Minute Change.—Microscopic examination of thin sections shows the involvement of both *tubes, blood-vessels, and intertubular substance*. Of the former, many are found choked with granular cells and the granular debris of cells, causing them to appear, under the microscope, as black, opaque lines by transmitted light, very similar, indeed, to the tubes in acute nephritis. In other situations the tubules are filled with fat-globules and fatty cells. In places the lumen of the tubes is preserved, in others not. Other cells are the seat of hyaline change. Others still are nearly normal.

The parts presenting a yellow tinge are those in which the fatty elements have replaced the normal, and this is the composition of the yellow specks already alluded to as visible to the naked eye. They represent a coil of tubules filled with oil-drops or fatty cells.* Certain tubules contain casts, usually of the waxy kind. Sometimes, indeed, they are very numerous. Rarely hemorrhagic extravasations are also found in the tubules.

The *capillaries* of the cortex are completely or nearly empty of blood, which has been expressed from them by the distended tubules. To this and to the fatty cells is due the extreme whiteness of these kidneys, whence the name by which they are known. Many of the glomerules are enlarged, their capsules thickened, their vessel walls thickened and hyaline, their capillary and glomerular epithelium proliferated and degenerated.

The *pyramids* in chronic parenchymatous nephritis are more changed than in the acute form, but the changes in them are quite secondary. They are sometimes a little paler, owing partially to a granular and fatty alteration in the cellular lining of the straight tubules, and partly to the presence of cells pushed down from the convoluted tubules above them. On the other hand, they may be congested and darker in color. The straight tubes of the cones as well as the looped tubes of Henle often contain waxy casts.

In chronic parenchymatous nephritis the *interstitial tissue* is always altered, and, it may be said, as a rule in proportion to the duration of the disease. It has already been said that sooner or later interstitial overgrowth always presents itself, although it is difficult to say when this overgrowth begins in any given case. Langhans reports a case in which death occurred five weeks after the appearance of the first symptoms, directly traceable to a thorough wetting, in which the stroma was *markedly thickened*. And in a case of Dickinson's already alluded to, intertubular cellular formation, "though approximating as much to pus as to fibre," was found within six weeks of the onset. Again, cases of much longer duration may be entirely without it. Interstitial *fibrosis* may, however, be considered as a superaddition of chronicity, and wherever a case is distinctly chronic it may be inferred, with tolerable certainty, that it is present. In this overgrowth the quantity of the connective tissue between the tubules varies extremely, being sometimes so slight as to be discoverable only on microscopic examination of thin sections; at others it is appreciable to the naked eye. Minute examination shows the thickened trabeculæ to consist of numerous round and oval nuclei, between which may be a homogeneous or more or less distinctly fibrillated intercellular substance.

II. *The Stage of Atrophy—The Fatty and Contracting Kidney, or Small White Kidney.*—The interstitial new formation above referred to possesses the properties usual to new connective tissue. Produced pri-

* No satisfactory explanation has yet been offered of the great differences in the degree of fatty degeneration in the different kidneys of chronic parenchymatous nephritis or in different parts of the same kidney. Dickinson says the cells have a greater tendency to be fatty when cold is the cause.

marily to replace destroyed tubular structure, it shrinks and gradually contracts the previously enlarged organ, while obliterating in turn a certain amount of the tubular structure. The extent of contraction varies greatly, increasing with the duration of the process. The kidney may continue as large and even larger than the normal organ, though smaller than the large white kidney, and its surface is uneven, lobulated, rough, and granular. Its capsule does not strip off easily, as from the large, smooth organ, but drags with it considerable of the tubular structure. The capsule removed, however, the surface of the kidney exhibits between the constrictions the same pallid, speckled appearance, distinct stellate veins, etc., already described; and on section the cortex exhibits the same anæmic appearance but may be narrowed. Microscopically, sections exhibit the same alternation of groups of normal and choked tubules already described, alongside of other places in which the tubules together with the Malpighian bodies at their extremities are obliterated. Between them is found a large amount of interstitial tissue, and the Malpighian bodies are surrounded by concentric layers of the same. Even minute cysts, the result of obstruction of tubules by the constricting tissue, are found. The secondary origin of this form of kidney is not conceded by every one. An independent primary origin is claimed for it.

III. A special form of this stage is chronic hemorrhagic nephritis. In this form brown hemorrhagic foci are scattered throughout the cortex between and into the tubes. The organ is still larger than normal and presents in other respects the histology of this stage.

It not infrequently happens that along with the changes constituting chronic parenchymatous nephritis are found also those of *lardaceous disease*. Thus in a large white kidney the Malpighian bodies will often strike the mahogany-red reaction with iodine, characteristic of this condition, although the alteration may not be recognizable by the naked eye. Occasionally the change may even affect the afferent and efferent vessels.

Symptoms.—There are few distinctive symptoms of chronic parenchymatous nephritis. It often begins insidiously, and, after a variable period of indescribable ill health, including, however, often *digestive derangements*, an *anæmic, waxy appearance* develops, with *puffiness of the face* and *swelling of the feet*. Ultimately the anasarca may become general, involving the face, hands, feet, legs, thighs, and trunk. The serous sacs also frequently contain fluid, almost always in severe cases. It is not, however, always so. It may be confined to the extremities or to the face, and may even be confined to more unusual situations, as the scrotum. Indeed, dropsy is often entirely wanting, but as a rule it is manifest sooner or later, and no symptom gives the patient so much inconvenience. In advanced degrees his legs and thighs are twice their normal dimensions. They are so heavy he cannot lift them, while they are often excoriated, and moist with exuding serum, and smarting with irritation. Very frequently, as the result of spontaneous rupture of the skin, the discharge of serum is profuse, saturating the bedclothing and even dropping upon the floor; occasionally, also, with relief to the patient.

Another very constant symptom is anæmia, producing a peculiar translucent waxy appearance, quite characteristic and often alone sufficient

to suggest the disease. But there may be very slight degrees of it which are not at all peculiar. Again, the debility of those suffering with advanced degrees of this condition is very striking. If able to walk at all, they soon get out of breath, are soon exhausted. Locomotion is often impossible in consequence of the extreme swelling, even if the strength otherwise permit it.

The Urine.—The urine is diminished to about 300 to 1200 c. c., though somewhat variable in quantity; often turbid, reddish-yellow, specific gravity normal or below, highly albuminous, and deposits often, but not always, a bulky, cloudy sediment. The quantity of urine also increases as the patient improves or as the stage of contraction is entered upon, so that it may even exceed the normal. The *albumin*, while also large, varies as to its percentage amount with the quantity of urine passed—from 0.5 to two per cent., or from one-half to three-fourths the bulk of the urine tested. The amount of albumin lost in the urine is sometimes very large. It has even occurred that the percentage proportion of albumin in the urine has exceeded that in the serum of the blood from the same patient. The quantity of albumin has very little effect upon the specific gravity. Indeed, the lighter urines are generally those which have the larger amount of albumin, because highly albuminous urines often contain little urea.

The sediment is made up of variously granular casts, among which the dark granular are conspicuous by their numbers and size, and especially their width. There are also found oil-casts and casts containing entire and fragmentary epithelial cells, which are also granular and oily. Finally, yellow waxy casts are also found. Casts vary in numbers, being sometimes scanty, but as a rule increase with the development of the disease and grow less as it amends. Compound granule (granular fatty) cells and other forms of fatty renal cells are often numerous. Leucocytes are also often very numerous, while red corpuscles also occur and in the hemorrhagic form are very numerous.

The normal constituents of the urine are *generally diminished* in quantity. The most important of these is *urea*. To the reduced amount of solids, and particularly of urea, the reduced specific gravity is due.

Notwithstanding the small proportion of urea which is excreted in this affection, uræmia is infrequent in chronic parenchymatous as compared with acute nephritis and contracted kidney. It is more frequent after the stage of contraction is reached.

Are there any symptoms by which we can recognize the stage of secondary contraction, which takes place sooner or later, provided the patient lives? The most reliable evidence that this has occurred is the presence of hypertrophy of the left ventricle and accentuation of the aortic second sound, although the possibility of an earlier hypertrophy cannot be denied. The increased vascular tension mentioned as presenting itself even in acute nephritis continues in chronic to stimulate the heart to more forcible contraction, which must sooner or later result in hypertrophy. As already stated, time is required to reach this stage, and by the time hypertrophy is developed contraction of the kidney is likely to have occurred. Long duration of the disease also affords presumptive evi-

dence that contraction has taken place. If a case of undoubted parenchymatous nephritis continues under observation for a year or more the process of contraction is likely to have commenced.

The dropsy diminishes and may disappear as the stage of contraction is entered upon. So, also, the urine changes in its properties. The quantity, previously small, is increased, while the specific gravity falls below normal, 1010–1015; the quantity of albumin is also much less than during the stage of inflammation. In these respects—absence of dropsy, larger amount of urine, and smaller amount of albumin—it resembles the true contracted kidney of interstitial nephritis, with which, indeed, it may be confounded in the absence of a previous history. But the casts continue pretty numerous, and exhibit much the same character that they do in the stage of enlargement, although they too may become scanty; and if we have not a knowledge of previous history the diagnosis between contraction secondary to previous enlargement, and primary contraction the result of interstitial nephritis, may be impossible. Uræmia is more common in the stage of contraction than that of enlargement.

In the hemorrhagic form the urine almost constantly contains blood. The quantity varies somewhat and is diminished while the patient is in bed, but reappears the moment he rises.

The *duration* of chronic parenchymatous nephritis is very variable. Many cases terminate unfavorably within a year after they have been established; but I have one now under observation in the stage of contraction which I have known to exist for twelve years at least.

Complications.—The complications of chronic parenchymatous nephritis are the same as those of acute. Œdema of the lungs, bronchitis, pneumonia, and inflammation of serous membranes are all liable to occur. Hypertrophy of the left ventricle is more common than in acute nephritis for the reasons already referred to, but, still, very much less so than in interstitial nephritis. Derangements of digestion are very frequent, probably due to a more advanced stage of the structural changes described under acute nephritis. The acute blindness, unattended by retinal changes, described as occurring in the uræmia of acute nephritis, rarely occurs here, while retinal changes are rather more frequent, but still uncommon compared with interstitial nephritis, under which they will be described.

Diagnosis.—The diagnosis of the inflammatory stage or stage of enlargement is ordinarily easy. The extreme pallor of the patient, the diminished urine of medium specific gravity, the usually large amounts of albumin, the numerous dark granular, oil and waxy casts of large diameter, free fatty cells, and fatty granular cells, especially if we are able to trace a history of long duration, all point to the disease. And if there is an antecedent history of scarlatina or exposure to cold, pregnancy, or long exposure, probability becomes certainty.

The symptoms of amyloid or lardaceous kidney very closely resemble those of the large white kidney, and it has been mentioned that the same causes are capable of developing both. It is often impossible to say which form of disease is present. It has usually been considered that if

there is enlargement of the liver and spleen, or persistent diarrhœa, and the cause is one which may produce lardaceous disease, it is certain that the latter condition exists; but recent observation has shown that the first two, at least, may be present, together with all the causes and other symptoms which are regarded as favoring lardaceous disease, and yet the disease be parenchymatous nephritis;* while the usual causes of lardaceous disease may operate to produce it in the liver, while the kidney may remain intact. As a rule, there is not so much dropsy in lardaceous disease, casts are more scanty, and generally hyaline, granular, and waxy; hypertrophy of the heart and uræmia and albuminuric retinitis do not occur. Often, too, the two forms of disease coexist, either as the result of the same cause, or the amyloid disease may be the result of long-continued parenchymatous nephritis.

The stage of contraction is more difficult of recognition unless we have had the case for some time under observation and are able to trace its continuation with the stage of inflammation. The resemblance to the contracted kidney of interstitial nephritis may otherwise be very close. But here again the albuminuria is apt to be larger and the casts more numerous, and to include the numerous varieties mentioned instead of the scanty, small hyaline casts which attend interstitial nephritis. In the latter the quantity of urine exceeds the normal, while in the former, although the quantity is larger than in the stage of enlargement, it is still less copious than in true interstitial nephritis.

Prognosis.—This is unfavorable so far as recovery is concerned. Well-marked cases terminate usually within two years, and sometimes within a few months. Many cases, however, may be very much prolonged by treatment, and if prolonged to the stage of contraction, the patient may be tolerably comfortable for some time, seems, indeed, to have another lease upon life. But sooner or later the dropsy returns, the heart fails, and the patient dies of exhaustion. Or some one of the complications, or uræmia, intervenes to carry him off. Of the former, œdema of the lungs or of the glottis and pneumonia are particularly dangerous. In the stage of enlargement, uræmia, while it is of rarer occurrence, is also less apt to end fatally than in the stage of contraction.

Treatment.—While it occasionally happens that spontaneous recoveries from acute nephritis occur, this is not the case with the chronic form. Here the expectant plan of treatment does not suffice. The patient with chronic parenchymatous nephritis, if left alone, grows steadily worse, and although measures of treatment may not frequently result in recovery, they often, if judicious, cause marked improvement and long avert the fatal end. There is always an intermediate stage between that of acute nephritis and the condition of the large white kidney, from which recovery often takes place, which calls for a modification of, or an addition to, the treatment described for the acute.

The chief indications in the treatment of chronic parenchymatous nephritis are two :—

* See an article by Dr. Paul Fürbringer, "*Zur Diagnose der amyloiden Entartung der Nieren.*"—Virchow's Archiv, Bd. 71, 1877, S. 400.

(1) To improve the quality of the blood, which may have become anæmic and contaminated with urea and allied effete matter; and

(2) To combat the symptoms and complications which form a source of great inconvenience and danger to the patient.

I. The first of these indications is chiefly fulfilled by the use of iron, salts of quinine and strychnine, nourishing food of the *right kind*, and proper hygienic influences, and also by depurating the blood of its retained urea. Iron is regarded by many as almost a specific in chronic parenchymatous nephritis and is prescribed constantly in the most reckless and thoughtless manner. *Large doses of iron should not be given.* They are useless, lock up the secretions, cause headache, and increase the danger of uræmia. The well-known Basham's mixture is a great favorite. It is really a solution of acetate of iron, and, made by adding to tincture of the chloride of iron acetic acid and solution of the acetate of ammonia, has the advantage of at least tending to diuresis, while it also strengthens.* But the tincture of the chloride of iron alone is an efficient preparation which is always accessible, and when combined with the sweet spirit of nitre, and freely diluted, is perhaps as efficient as the Basham's mixture. Only a few drops should be given. To either, the quinine and strychnine may be added, if desired, while to the latter the infusion or tincture of quassia makes a compatible addition.

With regard to diet, while it is true that a sufficient amount of food of good quality is desired, those articles should be selected which contain a minimum of nitrogen. Experience has shown that where the appetite is good and large quantities of meat are eaten, uræmic convulsions have been more frequent, whereas, when the appetite has been bad and little food taken, uræmic convulsions in chronic nephritis are very rare. While, therefore, it is not necessary to omit all such food, it *is* desirable to limit it to moderation, and, while drawing upon the vegetable kingdom for food, to make up the deficiency in meats by the free use of milk. The good results of the milk treatment in cases of chronic nephritis are now among the best acknowledged in the treatment of the malady, as evidenced in the diminution of albuminuria, decline in dropsy, increase in the quantity of urine passed, and general amelioration of symptoms. And of the different methods of employing milk, the pure milk diet has been most satisfactory. From 2½ to 3½ quarts (2.3 to 3.3 liters) a day meet the requirements of an adult male, and less is often sufficient. The milk should not be skimmed but diluted, for by retaining the cream the casein or proteid constituent is maintained in smaller proportion. Rich milk is not desirable—indeed, it is better taken diluted with Vichy water or carbonated water and taken at stated intervals, say six to eight ounces every two hours. It is not, of course, always necessary to confine the patient to a pure milk diet, but it should at all times constitute a large part of the food.

* The more usual formula for Basham's mixture is as follows: ℞. Tinct. ferr. chlorid., f℥ij (7.4 c. c.); Ac. acet. destillat., f℥ij (7.4 c. c.); Liq. ammon. acetatis, f℥iij (90 c. c.); Curaçoæ or syrupi simpl., Aquæ, āā q. s. ad f℥vi (180 c. c.): M. et S. Teaspoonful or dessertspoonful twice a day, in half a tumbler of water. If it becomes turbid it is probably because the acetic acid has evaporated, when a few more drops may be added to clear it up.

Next to diet, rest is a most useful measure to ameliorate the symptoms of chronic nephritis, and an albuminuria reduced to a minimum while the patient is up may often be further reduced by putting him to bed. The beneficial effect of rest upon œdema from any cause is too well recognized to require other than an allusion. The advantages of rest in bed are, however, sometimes more than counterbalanced by the disadvantage to the patient of confinement and want of fresh air and out-door life. These, of course, must be weighed, and that one adopted which serves the patient best.

Under hygienic measures is included suitable clothing. That next the body should be of wool; for it must be remembered that, on the one hand, the skin is a powerful adjuvant to the kidney in its eliminating operations, and, on the other hand, that any interference with or suppression of the action of the skin must throw more work on the kidney. Cold is the agent which produces such suppression, and warmth the means by which the action of the skin is encouraged; and no texture prevents the former or secures the latter more effectually than wool. For the same reason, while the maximum amount of fresh air is desirable, cold and dampness should be avoided or sufficiently guarded against.

II. The second indication is to depurate the blood of accumulated impurities, as well as to combat the symptoms and complications which cause inconvenience or jeopardize life. These symptoms are those of dropsy, effusions into the serous cavities, and congestions. The patients suffering from them are usually confined to the house, or go out of it at such great inconvenience as to make it intolerable to do so. Of dropsy there is abundant evidence to the naked eye; but of the necessity of depuration there is, unfortunately, no direct means of estimation except by a volumetric analysis of urine, which has been rendered very much more easy of late by ready methods for determining urea. Fortunately, too, the means which are best calculated to relieve the one are most likely to relieve the other. These measures are, in addition to diuretics, such as promote a more decided action of the skin than any yet alluded to and certain purgatives.

With regard to diuretics, nothing need be added to what has been already said under acute nephritis. But as to measures which promote a decided action of the skin, I desire to add a little more. These are the warm bath, the Turkish bath, warm-pack bath, and the hot-air or vapor bath already alluded to. Any of these may be used as convenience or the patient's choice may determine, while the frequency with which they should be used depends on the urgency of the case.

The "warm" or "cold-pack" is a very pleasant form of bath. The patient is wrapped up in a wet sheet, either warm or cold, and further enveloped in a sufficient number of blankets. A very comfortable sweat generally ensues, which is continued for an hour. In the use of the warm bath the patient is immersed in it at a temperature of about 104° F. (40° C.), and kept there from half an hour to an hour. He is then removed and wrapped in blankets. It has been said under the treatment of acute parenchymatous nephritis that these effects are more

conveniently and as efficiently brought about by the use of *jaborandi* and its derivative, *pilocarpin*. The directions for their use, given in connection with acute nephritis, need not be repeated. They may be used about as often as the baths, usually on alternate days, occasionally daily, with advantage.

The judicious use of aperients is an efficient means of depurating and reducing dropsy. The selection must depend on the urgency of the case, as sufficient has been said in connection with acute nephritis.

But in many cases of chronic nephritis a stage is finally reached at which all treatment of the kind described fails to relieve the dropsy, which becomes eventually the sorest burden of the malady. The body becomes greatly increased in weight, the integument of the extremities is stretched almost to bursting, and sometimes it does rupture, followed by leakage, which, although in one way inconvenient, is in many senses a great relief to the patient by diminishing the tension referred to. Acting upon this, physicians have long been in the habit of puncturing the swollen parts to produce the required leakage. It is a common practice to make a number of minute punctures with a needle or sharp-pointed bistoury, but free incisions may be made on the inner or outer side of the ankle of each leg. Free drainage is thus secured, often with great relief. Or Southey's tubes may be used at convenient places. They are introduced by means of a little trocar and fine India-rubber tubing attached and carried to a suitable vessel outside the bed. Some remarkable recoveries have followed this treatment. Great care should be taken to keep the tubes clean, as they are liable to become dirty and clogged.

The treatment of the complications is in no way different from that of the same conditions under other circumstances. The point to be impressed is the importance of being constantly on the lookout for them. Effusions into serous cavities are probably the most important. Œdema of the glottis requires separate allusion, as a complication most alarming and threatening to life. Inhalations of steam may be tried, but prompt punctures or incisions are the only certain means of relieving the patient and saving his life.

I know of no measures directly curative in acute or chronic nephritis, that is, remedies which by their direct action remove the morbid state, and I believe none exist. All that can be done is to place the patient in a condition most favorable for nature's kindly offices, which are always exerting themselves toward cure. This is accomplished by the measures recommended, which also eliminate the mechanical and poisonous products which interfere with recovery.

INTERSTITIAL NEPHRITIS.

SYNONYMS.—*Contracted Kidney; Chronically Contracted Kidney; Renal Cirrhosis; Cirrhotic Kidney; Granular Degeneration; Granular Kidney; Red Granular Kidney; Gouty Kidney; Renal Sclerosis.*

Definition.—Interstitial nephritis is a chronic process resulting ultimately in a shrunken kidney, in which there has been extensive destruction of the tubular substance and overgrowth of intertubular connective tissue.

Etiology.—Of the recognized forms of Bright's disease, interstitial nephritis shares with chronic parenchymatous nephritis a large number of instances in which the cause is undiscoverable. There are, however, some well-determined causes. Among the most tangible of these is gout. Gout is associated with so many cases of contracted kidney, that the term gouty kidney has come to be a well-recognized synonym for the product of interstitial nephritis. There are probably no cases of gout, which have continued for any length of time, which are not accompanied by interstitial nephritis. Uric acid in the blood is probably the exciting cause. Another well-recognized cause is lead in lead poisoning, the absorbed lead acting like the poison of gout. Hence painters, glaziers, workers in lead in any shape, are frequent victims. Dr. Dickinson considers it safe to assert that of painters at least one-half eventually die of granular degeneration of the kidneys. This is certainly not the case in this country. Long-continued cystitis, especially following gonorrhœa, is a cause in a few instances, the inflammation traveling up the ureter to the pelvis of the kidney and thence to the intertubular tissue. The more usual result of such extension is suppurative nephritis.

Among the causes the operation of which cannot be so directly proven is anxiety or business care and worry. Dr. Clifford Allbutt, quoted by Dr. Robert T. Edes,* goes so far as to attribute "twenty-four out of thirty-two cases in private practice to some long-continued anxiety or great grief." This also is contrary to my experience. It is true this disease very often exists for a long time undiscovered in business men who have lived under a state of constant mental tension. The effect of this cause is augmented if its subjects combine too liberal eating and drinking with the hard work and anxiety.

Hereditary influence is occasionally a cause of the contracted kidney. A remarkable instance of this occurred in my own practice. I was consulted by a man, aged thirty, who has granular kidneys. His father and mother both died of Bright's disease, aged fifty-six and sixty-three years respectively. The mother had convulsions. A brother died of Bright's disease, without convulsions, at the age of thirty-seven. Two children of this brother had Bright's disease when four and seven years of age. A second brother died at the age of twenty-nine with convulsions. A third and fourth brother, aged twenty-three and thirty-

* Edes, Robert T., "Some of the Symptoms of Bright's Disease," Boston Med. and Surg. Jour., Vol. CIII, No. 2, July 8, 1880.

two years respectively, have had Bright's disease for six years. A sister, aged thirty-six, has had Bright's disease for five years. A brother, aged twenty-six, and a sister, aged thirty-four, have as yet no signs of Bright's disease. A maternal cousin died of undoubted Bright's disease, and other members of the family, belonging to previous generations, died with symptoms which suggest Bright's disease. The patient himself has undoubted granular kidney, discovered in August, 1880. An examination of his urine in 1876 revealed no evidences of the disease. There is no gout in the family. Dr. Dickinson also relates the history of a family in which a hereditary albuminuria existed independent of gout.

Prolonged passive congestion, due to valvular heart disease, may become a cause of granular kidney. The same may be said of stone in the kidney causing numerous attacks of nephritic colic. I have known typical chronic interstitial nephritis ensue on such attacks of nephritic colic.

Pregnancy, so frequently a cause of acute parenchymatous nephritis, is rarely, if ever, a cause of interstitial nephritis and chronic parenchymatous nephritis. It is true, contracted kidney is sometimes found in autopsies of women dying of puerperal nephritis, but I believe it more frequently precedes than follows the pregnancy.

Interstitial nephritis is commonly a disease of middle age, the majority of persons in whom it is discovered being past forty. A few cases occur under thirty. The youngest patient whom I have ever had was twenty-six. Still younger are reported.

It must be remembered that there is a tendency to overgrowth in the interstitial tissue of the kidney, as of other organs, in old age. Hence the term *senile atrophy* of the kidney. It is not safe, therefore, to call every instance of atrophied kidney met in the post-mortem room a case of interstitial nephritis. The clinical history, or some one of the well-marked symptoms of the disease, as albuminuria or uræmic symptoms, should have preceded to sustain the diagnosis.

As to *sex*, nearly twice as many males have the disease as females, because of the more frequent exposure of the former to the causes of the affection.

Morbid Anatomy.—In interstitial nephritis both kidneys are involved, but there is often a marked difference in the amount of disease in each.

Macroscopically the organs are evidently smaller than in health, often less than half as large. I have seen them less than five centimeters (two inches) in length. Next to this reduction in size, the most striking feature of the contracted kidney is its uneven or granular surface, which is, however, not always recognizable until after the capsule is removed. Very characteristic also is the presence of cysts with more or less clear watery or gelatinous contents, often visible through the capsule. These are not invariably, but quite frequently present. The capsule, itself thickened, strips off with difficulty, dragging portions of the secreting structure with it. Owing to the resistance which the blood meets in its passage through the kidney, a larger portion of it passes out of the organ by

way of the capsule. Hence the blood-vessels of the latter are dilated, as are also the lymph spaces.

Bereft of its capsule, the kidney is hard, granular, tough, and usually darker than in health, whence one of its names, the "red granular kidney." This color is in strong contrast to the white or slightly yellow tinge of the fatty and contracting kidney, and although it is not always marked, and sometimes even substituted by a paleness, it is still easily distinguished from that of the contracting kidney of parenchymatous nephritis. The granules on the surface of the contracted kidney are distinct round and oval elevations of the surface, ranging in size from that of a pin's head to a pea, or from $\frac{1}{25}$ to $\frac{1}{5}$ inch (one to five millimeters). Those of smaller size are most numerous, and at first correspond with the lobules, the bases of which are visible on the surface of the normal organ. The larger ones result from the coalescence of two or more of the smaller. The granules themselves are of a lighter color than the depressed circlets between them, which are tinted with vascularity, and have a purplish or faint red hue. The cysts already referred to are now more distinct (after removal of the capsule) and vary greatly in size. While equaling in minuteness the smallest of the granules, some of them are as large as a walnut. The larger are apt to be ruptured on stripping off the capsule.

On section it is at once evident that the reduction in size of the kidney is largely due to a narrowing of the cortex, although the medulla is also contracted. The former may not be more than $\frac{1}{8}$ to $\frac{1}{6}$ inch (three or four millimeters) in width, and exhibit every degree between this and the normal. The Malpighian bodies are smaller, less numerous, and can scarcely be detected by the naked eye, while the small arteries are more prominent from thickening of their walls. The increased density and firmness of the organ are apparent. In a gouty subject, linear chalk-marks of sodium urate may be present, more particularly in the pyramids of straight tubules, and are contained within as well as between the tubules. The little cysts referred to as seen on the surface may also be scattered throughout the section from cortex to papillæ, but they are more numerous in the former. They are not always present. The pelvis of the kidney may be unaltered. It is sometimes enlarged and the calyces elongated from retraction of the pyramids. On the other hand, if the kidney is very much reduced in size the capsule may be pursed up and proportionately smaller.

Minute Structure.—Minute examination of thin sections through the cortex clearly reveals the condition to be an excess of connective tissue, with destruction of the tubules and blood-vessels. The process is best studied if the sections include the capsular edge as it progresses from without inward. In these may be seen extensive tracts of connective tissue separating the tubules, which, in sections of healthy kidneys, are closely in contact without appreciable intertubular substance. The *tubules* themselves appear in places quite normal; in others they are represented by fragmentary portions in which the cells are still unchanged; in others again the cells exhibit a granular degeneration; some tubes are evidently dilated; others still are completely shriveled, while it is evident from the

larger areas of connective tissue that many have completely disappeared. In a few tubules waxy casts are present. The Malpighian bodies are surrounded by concentric layers of nucleated connective tissue. Many of them are shriveled and atrophied, and an attempt to inject them with colored injecting fluids fails either partially or completely. Some thus altered lie detached from the tubules, with which they should be continuous. The granules on the surface of the kidneys are resolvable by the microscope into tubules, some of which are in a tolerably perfect state, some decidedly dilated.

The *cysts* originate partly in dilatations of obstructed segments of the uriniferous tubules and partly in dilated Malpighian capsules. Proof of the latter mode of origin is found in the fact that compressed capillary tufts are sometimes found lying up against one side of the wall of the cyst. The same overgrowth of connective tissue may be seen in the pyramids, but it appears later, extends more slowly, and never reaches the degree found in the cortex.

The *blood-vessel* of the contracted kidney is the seat of important changes. In the first place, it shares with the tubules the compressing effect of the contracting new formation. As the result of this, a part of the capillary system is destroyed, and in the part thus destroyed are many capillary coils in the Malpighian bodies. Hence, as many afferent arterioles send their blood directly into the second capillary network, which is also cut down by the pressure. The vessels which remain are often sclerotic, dilated, and twisted, and in consequence of the destruction of numerous Malpighian bodies send much of their blood out through the capsule. The intima is thickened and the media and adventitia invaded by hyperplastic connective tissue, but always to a less degree. Even arterioles whose walls have thus been thickened become involved in the atrophic processes affecting the glandular tissue of the organ and ultimately disappear.

Associated with these changes is a general arterio-sclerosis and hypertrophy of the left ventricle, sometimes also of the right. The final effect of these alterations is to produce a brittleness in the arteriole walls, which disposes them to rupture on very slight increase of intravascular pressure. Hence, the frequent fatal termination of cases of interstitial nephritis by apoplexy, also the frequent nasal and retinal hemorrhages which characterize the disease.

The *retinal changes*—retinitis albuminurica—symptoms of which form so important a part of the symptomatology of chronic interstitial nephritis are various and vary with the stage of each case. Many cases are first diagnosticated by the ophthalmic surgeon. The changes include serous swelling of the disc and surrounding retina, hemorrhagic extravasations, dirty white splotches representing fatty degeneration, and dilatation of the veins and capillaries, with fatty degeneration and sometimes hyaline thickening of their walls.

Symptoms.—The great obscurity as to the origin of a large majority of cases of contracted kidney is only equaled by that of the insidiousness of their approach. The beginning of the disease is certainly not characterized by any distinctive symptoms; and its progress

is often unmarked by any, until those of uræmia mark the beginning of the end. To the observing physician, some obscure symptom may suggest an examination of the urine; or the peculiar tense and bounding pulse of hypertrophy of the left ventricle, or the more tangible symptom of a slight swelling of the feet or ankles, recognizable only at night or through the unexpected tightness of a boot, may lead to the same examination.

Changes in the Urine.—Attention being called to the *urine*, it will be found to present characters which are more or less distinctive and lead easily to a diagnosis. It is, when freshly passed, acid in reaction, copious, often exceeding the normal amount, never scanty except in the last stages of the disease. The quantity may reach 90 ounces (2700 c. c.). The patient very commonly has to rise at night, probably not more than once or twice, to pass his water. There may be corresponding thirst. Consequently, the urine is light in color and of low specific gravity,—1005 to 1015,—and contains a trifling or moderate flocculent sediment. It is generally albuminous, but the albumin is small in amount and may be temporarily absent, or it may be absent before a meal and present after it. Later, however, it becomes constant. It seldom exceeds $\frac{1}{10}$ the bulk of fluid tested, and is very constantly a good deal less, showing a delicate line of white by Heller's nitric acid test. Tube-casts are present, but not usually numerous. They are almost solely hyaline, and pale granular. Some of the hyaline casts are delicately so, requiring nice illumination for their detection; others are distinct and sharply cut; others still contain two or three glistening oil-drops. Casts may at times be absent and again reappear, as is the case with albumin. Toward the termination of cases of interstitial nephritis the urine diminishes in quantity, the specific gravity increases, and the casts become much more numerous, and include among them highly granular or dark granular, and occasionally even blood-casts in addition to those alluded to, and there are sometimes a few blood discs earlier. The *urea* is also diminished sooner or later, and in this manner the lower specific gravity is contributed to. This fall becomes marked toward the close, accounting for the uræmic symptoms which often first announce the disease. It may be as low as 15 grains (one gm.), and may be anything between this and the normal twenty-four hours' quantity, which may be put down at from 20 to 40 grams in an adult. All the remaining normal constituents may be said in general terms to be diminished.

As to the other symptoms, a feeling of *unaccountable weakness* or of being tired is very often present, but it is a symptom which occurs in many conditions, and should only be considered as suggestive. Slight œdema about the feet and ankles is often present, being so slight as to escape detection, or is discovered accidentally. When present it is significant, but it is often entirely wanting.

Hypertrophy of the left ventricle of the heart without valvular disease is so constant as to be alone suggestive of the disease. No case of interstitial nephritis has existed for any length of time without this condition supervening, and as few cases are discovered until they have existed a good while, few are found without hypertrophy. In more than

one-half at least of cases there is evident hypertrophy. It is recognized not at first so much by the resulting enlarged percussion area as by the sharp accentuation of the second aortic sound. Corresponding to this, the pulse is hard and resisting, indicating high tension and thickening. These two symptoms have, therefore, great diagnostic value. Sclerosis is distinguished from tension by obliterating the blood current by pressure and feeling the artery beyond this point. The sclerosed vessel continues tangible, that of high tension disappears. As a symptom of this stage is often an uncomfortable *pulsation* felt in the head and even other parts of the body.

As the disease becomes more advanced there are added cardiac distress, dyspnœa, palpitation, and reduplication of the first sound. The latter is probably due to a want of synchronism in the systole of the two ventricles. There is usually no murmur, because there is no valvular disease. The latter may be present. The patient may have had valvular disease prior to the renal malady, or the latter itself, by its long continuance, have produced endocarditis and atheroma with an aortic systolic murmur, or there may be a mitral murmur due to relative insufficiency. The hypertrophy of the heart is conservative and all goes well as long as the power of the heart lasts. When the latter begins to fail and dilatation appears the blood-pressure diminishes, and with it begins a train of symptoms, among which diminished secretion of urine and dropsy are the most conspicuous along with gallop rhythm, dyspnœa, palpitation, and dizziness. These symptoms may again be averted for a time by hypertrophy of the right ventricle, which is a signal of disturbed compensation.

Dimness of vision due to retinitis albuminurica, already described on p. 657, is a characteristic symptom. It is often the first recognized, and hence the diagnosis is frequently first made by the ophthalmologist. It is a serious symptom, generally considered a sign of advanced disease, as, indeed, it usually is, yet I have seen it improve under treatment. Some assign two years as the limit of life after its recognition. The atheroma of the blood-vessels is the cause of another symptom which frequently determines the mode of death—rupture of a blood-vessel in the brain; in a word, *apoplexy*. This accident is more usual late in life, but Dickinson reports a case in which cerebral hemorrhage occurred in a girl of twelve. The proportion of cases of recognized interstitial nephritis in which this happens is not large, but many cases of apoplexy are directly traceable at autopsy to unsuspected renal cirrhosis. Dickinson believes that of fatal cases of apoplexy one-half are preceded by this form of disease. Hemorrhages in other situations are referable to this same altered state of the blood-vessels, as, for example, into the retina, from the nose, and even into the stomach. Hence *sudden blindness*, in addition to the dimness of vision due to retinitis albuminurica, is a symptom which occasionally presents itself. Amaurosis and amblyopia also occur and may disappear, but dimness of vision due to retinitis albuminurica, is a permanent symptom. Auditory disturbances also occur, such as ringing in the ears with dizziness and more or less deafness.

The termination by *uræmia* occurs more frequently in this than any

other form of Bright's disease. Bartels says that nearly all the patients he has seen die in the extreme stage of atrophied kidneys sank under the symptom of chronic uræmia. He is probably correct, and it is frequently the first intimation of the existence of any derangement, and manifests itself in any one or more of the forms already described under acute nephritis. Headache, drowsiness, convulsions, stupor, delirium, maniacal excitement, renal asthma, restlessness, nausea, vomiting, any one of these symptoms may usher in the dreadful train which is so apt to be fatal. E. C. Seguin especially has called attention to occipital headache as a symptom of uræmia.* V. Leube considers that even the intermittent headaches which occur in this disease, and which very closely resemble migraine, are probably due to uræmia. Temperature follows the same rule as in uræmia from other forms of nephritis (see p. 637). The *convulsion* is commonly associated with a rise. Dyspeptic symptoms with obstinate vomiting, particularly in the morning on rising, are apt to usher in a chronic uræmia. Diarrhœa is less common, but also sometimes occurs toward the close, when it may be very difficult to control.

The duration of this form of renal disease is indefinite. Always a chronic process, it may last for years undiscovered, and when discovered before it is too far advanced, the knowledge of its presence will suggest measures of precaution and treatment which may so prolong life that it need only be determined by its natural limit or some other disease. Yet complete recovery from well-established interstitial nephritis is probably unknown.

Complications.—These include bronchitis, pericarditis, pleurisy, pneumonia, and, more rarely, endocarditis, peritonitis, intertubular gastritis, and even inflammation and ulceration of the bowels. But all inflammatory complications except bronchitis, pleurisy, and pericarditis are less frequent than in acute nephritis. Bronchitis is said to be the most frequent complication, while pericarditis is the most dangerous, being almost invariably fatal. The former occurs in about 33 per cent. of the cases, and the latter in, according to W. Howship Dickinson, 25 per cent., and to Sir T. Grainger Stewart seven per cent. My own experience accords more nearly with the latter. Pleurisy and pneumonia are also of tolerably frequent occurrence, Dr. Stewart finding the former in 15 per cent. of his cases and pneumonia in seven per cent. Acute endocarditis and peritonitis occur, but very seldom.

Diagnosis.—The diagnosis of an interstitial nephritis is usually easy, if in any way an examination of the urine is suggested. The increased quantity, the low specific gravity, small albuminuria, delicate hyaline, pale granular casts, and hypertrophy of the left ventricle, even in the absence of other symptoms, are sufficiently distinctive. The conditions which should suggest such an examination are a feeling of constant weariness, slight swelling of the feet, drowsiness, frequent headaches, confused intellect, dyspeptic symptoms, obstinate nausea, delirium, coma, and convulsions. High arterial tension should always suggest examination of the urine.

* Archives of Medicine, Vol. IV, No. 1. New York, August, 1880.

Prognosis.—The prognosis is unfavorable as to recovery, but favorable as to prolongation of life if the diagnosis be made sufficiently early. Cases with casts and small albuminuria may continue under observation for a dozen or more years. If not made previous to the setting in of uræmic symptoms, little may be expected. But even at this stage energetic treatment may still avail to avert the immediate danger and prolong the patient's life. The possible sudden occurrence of convulsions and coma, and of death therefrom, should always be remembered and impressed upon the relatives of the patient. These constitute unfavorable symptoms, to which toward the end Cheyne-Stokes breathing may be added.

Treatment.—From what has been said under prognosis, it is evident that the most hopeful result to be expected from treatment is the protection of the patient from the consequences of his malady, rather than the restoration of the kidney to its normal condition. Our power in the former respect depends largely upon the stage at which the disease is discovered. If detected at a period in which the urine is tolerably abundant, the albuminuria small, the casts few, and there is no œdema, the indications are :—

(1) To maintain the integrity of the blood, by preventing the accumulation of urea and allied compounds ; and

(2) To treat as they arise the accidents and complications which are often so dangerous to the patient.

The first of these is best accomplished by dietetic and hygienic measures, aided by the use of a few remedies. First, as to food, all that was said under chronic parenchymatous nephritis is more than applicable to interstitial nephritis, because the appetite is still good and a suitable selection can be exercised. As the urea has its chief source in the azotized elements of food, it is plain that the larger the quantity of such food consumed the larger is the accumulation of urea to be eliminated by the kidneys. Now, while it is not possible nor perhaps desirable to exclude all nitrogenous food, it may be largely reduced. This is accomplished by the substitution of all or a part of animal flesh by milk, while drawing the elements of a mixed food from the vegetable kingdom. The so-called vegetarians have proved conclusively that it is possible to live and maintain good health upon milk and an otherwise exclusively vegetable diet. And while this diet may not be compatible with the highest mental and physical development of which man is capable,* which is not at all proven, the resulting life is perfect enough for all its objects, and will doubtless be acceptable to those who prefer to live. On such a system I have known the patient with contracted kidney to maintain apparently perfect health for many years.

With regard to habitual beverages, the use of strong alcoholic drinks is harmful, and brandy, whiskey, champagne, and sherries and ports should be prohibited. The light wines, and especially the red wines and lighter alcoholic drinks, as lager beer, porter, etc., may be used in moderation. I have already referred to the very great value of the alkaline mineral

* See "Carpenter's Physiology," 7th English ed., 1869, p. 77.

waters, such as those of Vichy, Vals, and Kissingen; to these a little claret may be added at dinner.

What was said of clothing, fresh air, and exercise in connection with chronic parenchymatous nephritis is even more applicable to interstitial nephritis. Warmth of the body, maintained by woolen garments next the skin to encourage its action, and the avoidance of damp and cold, which check it, are peremptory. The wetting of the body by rain, or of the feet alone, has frequently been the exciting cause of a uræmic attack which was fatal. India-rubber overshoes should be worn in all damp weather.

In this connection sea-bathing requires allusion. It is well known that sea-bathing sometimes induces albuminuria in normally constituted persons, or at least in individuals at other times free from albuminuria. This is probably due to a temporary congestion of the kidney, from introversion of the blood kept up by the duration of the bath. Still more mischievous, therefore, must be the effect of prolonged sea-bathing upon one whose kidneys are already damaged and incompetent to perform their office. Sea-bathing, therefore, or, indeed, any form of cold bathing, should be interdicted to the patient with contracted kidney or, indeed, with any form of chronic nephritis. Sea-bathing is especially mentioned because it is considered so healthful, and permits remaining in the water so much longer at a time. On the other hand, a daily warm bath at bedtime and especially an occasional Turkish bath is advantageous.

For the same reason residence in a warm, equable climate is often of signal service in interstitial nephritis; and cases are reported where the albumin has disappeared and symptomatic recovery taken place during such residence.

Prolonged bodily or mental fatigue should also be avoided by these patients, as they have been known to be the exciting cause of uræmia and death; especially are they when associated with free eating and drinking. The patient should live a life as easy and as free from any of these causes which have been considered as his circumstances will permit.

As to drugs, they are of limited utility. The moderate use of tonics, including quinine, strychnine, and iron, is useful to combat the tendency to anæmia and weakness, which sooner or later follows. In this form of Bright's disease even more than in chronic parenchymatous nephritis is the indiscriminate use of iron to be guarded against. Iron in contracted kidney as often used is a harmful drug. It locks up secretions, causes headache, and increases the danger of uræmia. Only when there is evident anæmia should it be used, and then only in very small doses. I hold that as long as iron blackens the stools or constipates the bowels the doses are too large and they should be reduced until the effect on the stools is very slight or not noticeable. Elimination is favored by stimulating the skin secretion, and this is best accomplished by an occasional warm bath, or especially a Turkish bath, with thorough friction and protection from cold by woolen underclothing. The Turkish bath is an admirable remedial measure, especially before the disease is too far advanced.

Diuretics are not indicated in the earlier stages, because the secretion of urine is already free. The bowels should be kept regular by the use

of the natural aperient waters, the Hunyadi, Friedrichshalle, and Rakoczy, or an occasional blue pill, or a dose of magnesium sulphate. Of course, later in the disease, when the heart begins to fail and the urine is scanty, both diuretics and purgatives are indicated. The same principles are to govern us in using them as have already been laid down under acute nephritis. Very high arterial tension sometimes demands treatment. A certain amount is a result of the conservative train of symptoms, beginning with hypertrophy of the left ventricle, and is necessary. But where a resulting throbbing is unpleasantly appreciable, especially if there is throbbing headache with flashes of light at each pulsation, tension should be lowered. The remedy par excellence is nitroglycerine, which should be given in doses of $\frac{1}{100}$ grain (0.00066 gm.) and upward. The best preparation is the one per cent. alcoholic solution, of which one drop represents $\frac{1}{100}$ grain (0.00066 gm.), the granules being more convenient but less reliable. The dose should be rapidly increased until the physiological effect is obtained and then reduced to what is found best to effect its purpose. Either the susceptibility of different persons varies greatly or the drug varies greatly in quality. I have repeatedly given it every two hours for days together in doses of $\frac{1}{100}$ grain (0.00066 gm.). It may be associated with digitalis, with which it coöperates by relaxing the arteriolar spasm which digitalis produces, and which interferes with its happiest effects.

The second indication mentioned, the treatment of the complications and accidents incident to the condition, resolves itself into the treatment of the bronchitis, the pericarditis, the pleurisy, pneumonia, endocarditis, gastric and intestinal disorders which have been named as occurring, and especially of the most serious calamity of all, uræmia. The treatment of the complications is that of the same conditions under other circumstances. Paracentesis is a measure which is often of signal service in effusions into the chest, and occasionally of the pericardium.

Dyspeptic symptoms are best treated with pepsin and acids and such other remedies as may be symptomatically required. Opium should be cautiously employed, if at all, not only in the gastro-intestinal troubles, but under all circumstances, as it undoubtedly increases the dangers of uræmia. This has been abundantly proven. It need not be discarded altogether, for there is sometimes no substitute for it in certain bowel affections and conditions of severe pain, but it should be given in smaller doses than usual and its effects watched. In like manner hypnotic, sedative, and antispasmodic effects, when desired, should be produced by sulfonal, trional, chloral, and bromides if possible.

Finally, as to the treatment of uræmia, the measures described in the treatment of uræmia in acute nephritis are to be used. Apoplexy, which is not an infrequent termination of the disease, in consequence of the atheromatous state of the blood-vessel walls, is recognized by the paralysis, general or partial,—most frequently hemiplegia,—which accompanies the unconsciousness. Remedies are here generally futile, but such may be used as are indicated for apoplexy elsewhere. The upright position, bleeding, counter-irritation, and, if the patient survives the im-

mediate accident, iodide of potassium, with a view to promoting absorption of the extravasated clot, may be used. Hemorrhages in other situations, as from the nose or alimentary canal, are treated by the same measures as when they occur under other circumstances.

As to special treatment, or treatment directed to the removal of the interstitial overgrowth in the kidney, there is none. Theoretically, the iodide of potassium ought to be of service. Unfortunately, the peculiar requirements of its administration, viz., the length of time during which the patient must take the remedy before any results may be expected, and the consequent difficulty in accumulating a sufficient number of cases, are such that it is almost impossible to determine whether it can be of any service or not. Owing to these difficulties it is doubtful whether its exact possibilities have as yet been determined. There can be no disadvantage in administering it if the dose is made so small as not to derange the stomach. Very rarely can more than a few grains daily be given. I believe I can say, however, of the bichloride of mercury that I have seen its long-continued use in doses of at first $\frac{1}{24}$ grain (0.0027 gm.), and later $\frac{1}{50}$ grain (0.0013 gm.), kept up a long time, followed by improvement. I have also seen improvement in the impaired vision of albuminuric retinitis follow its use. Certainly in the event of a clear syphilitic origin the iodide of potassium should be used.

LARDACEOUS DISEASE OF THE KIDNEY.

SYNONYMS.—*Amyloid Disease; Albuminoid Disease; Waxy Kidney; Depurative Disease.*

Definition.—A pathological state of the kidney in which its structural elements are more or less infiltrated with a substance of albuminous composition and of bacony lustre, but best recognized by its striking a deep mahogany-red with a solution of iodine.

Etiology.—The most frequent cause of lardaceous disease is profuse and long-continued suppuration, such as occurs in chronic bone disease, whether tubercular, syphilitic, or traumatic in origin; or such discharge as constitutes the expectoration in cases of chronic phthisis and chronic bronchitis with bronchiectasis. Syphilis itself, independently of the tertiary conditions which it produces, is a frequent cause of lardaceous disease. Cachectic states of any kind, chronic dysentery, ulceration of the bowels, and chronic albuminuria are possible causes.

Either sex is equally subject to lardaceous disease, but as men are more frequently exposed to its causes, it is in them rather more common. Very young children are rarely affected, for evident reasons, but in young persons from eleven to thirty it is most frequent. After thirty it grows gradually rarer. Tubercular hip-disease in children is, however, a cause.

Morbid Anatomy.—The incipient stages seldom present alterations recognizable by the naked eye unaided by reagents. But if, after section of the kidney, the cortex be treated by a solution of iodine and iodide

of potassium,* numerous mahogany-red points make their appearance; or if by a solution of violet-anilin, as many red or pink points. These are the Malpighian bodies, whose capillary tufts are the first to be affected by the change. The kidney in this early stage is normal in size or very slightly enlarged. Its capsule strips off readily, leaving an organ which exhibits no changes, or a paleness or translucency which readily escapes notice, but may be recognized at the edges of a thin section. Very often, too, they are completely overshadowed by other alterations, for amyloid kidney is most frequently a superadded event in the course of chronic Bright's disease, while the same event may happen in interstitial nephritis. But the large white kidney of chronic parenchymatous nephritis is especially apt to exhibit a slight degree of lardaceous change, which may altogether escape notice unless iodine is used. Hence *iodine should be tried upon all kidneys* removed at autopsy.

In a more advanced stage of uncomplicated lardaceous change the kidneys are both enlarged, usually symmetrically, but the extreme degrees of enlargement are usually associated with fatty degeneration of the epithelium. Such organs were a pair weighing 23 ounces (715 gm.) which came under Dr. Dickinson's † notice. Dr. Johnson ‡ refers to a case in which the two kidneys weighed 28 ounces (870 gm.). Rindfleisch§ has seen a single instance of that very rare condition, complete lardaceous infiltration, that is, in which the basement membrane of the uriniferous tubes, as well as the capillaries, were infiltrated, where the kidney was enlarged to nearly twice its normal size. In the uncomplicated forms of lardaceous disease the capsule is not adherent, but if interstitial changes exist to any extent it is adherent. It leaves the surface of the kidney pale and anæmic; occasionally the stellate veins are conspicuous. The characteristic translucency may even be recognized in the organ in bulk, but in sections is more striking. When the change is present in high degree the edges of a thin section are almost as translucent as a similar section of bacon. On bisection the cortex is seen to be enlarged; it is pale, anæmic, waxy, firm, and resisting. The pyramids are normal in hue and area. The iodine solution added to such a kidney produces its peculiar coloration, not merely in the Malpighian capillaries, but also in the afferent and efferent vessels and the vasa recta of the pyramids. In a still later stage, that of atrophy, the kidney becomes contracted, diminished in size, rough, and even distorted in shape. The capsule is adherent, and on section the cortex is found narrowed, sometimes as much so as in the contracted kidney of interstitial nephritis.

To microscopic examination in the first stage, in which the naked eye often fails to detect anything abnormal without the aid of iodine, the

---

* *The Iodine Test Solutions.*—The best test solution for macroscopic purposes is one made by dissolving 2½ grains (0.162 gm.) iodine by the aid of five grains (0.324 gm.) of iodide of potassium in one fluidounce (30 c.c.) of water. The solution contains about ½ per cent. of iodine. For microscopic preparations a solution weaker than the above, or a ¼ per cent. of iodine dissolved by twice the quantity of iodide of potassium, is more suitable, and sometimes a solution containing as much iodine as water alone will take up answers best.

† Dickinson, *op. cit.*, p. 249.

‡ Johnson, *op. cit.*, p. 104.

§ Rindfleisch, "Path. Histology," New Syd. Soc. Trans., 1873, Vol. II, p. 167.

Malpighian bodies exhibit a lustrous or waxy appearance. They are also enlarged and the capillary walls thickened. At this stage there is no visible alteration in the tubules or their epithelium. In the second stage larger vessels are involved, the vasa afferentia and efferentia in the cortex, and also the vasa recta of the cones; also the second capillary network of the cortex, and an exudation occurs into the tubules of a glistening material which forms casts. This is undoubtedly at times the amyloid material, for such casts sometimes strike the mahogany reaction. At other times they have the composition of ordinary hyaline casts. It is to be remembered, too, that similar waxy casts are found in the tubules in other forms of chronic renal disease.

The arteriole walls are thickened by an involvement of both interna and media. This thickening is attended by an extraordinary distinctness of the muscular fibre cells of the circular coat. Later the basement membrane and epithelial lining may also be invaded, the cells being swollen, translucent, and apparently fused. It is also quite usual for the epithelium of the cells to be fatty, and the capillary walls to contain aggregations of fat-globules, while the urine in the later stages contains oil-casts and fatty cells.

In what has been called the third or contracting stage of lardaceous kidney, but which may be the ordinary contracted kidney on which the amyloid change has been engrafted, minute examination reveals, in addition to the appearances described, the hypernucleated intertubal overgrowth. Cysts are occasionally present for the same reason that they are found in the granular contracted kidney and the surface assumes also a certain degree of granulation.

Symptoms.—An individual who has had syphilis, or who has phthisis, bone-necrosis, or other affection causing an exhaustive drain, may acquire this form of kidney disease without appreciable addition to his symptoms. There may be a growing frequency in micturition, but this symptom may be totally absent. Accidentally, perhaps, a somewhat copious albuminuria is discovered. At first the urine may be quite clear and no casts are met, or they are exceedingly scanty, and hyaline or faintly granular. Later, a slight œdema of the feet may appear while the patient is up and about, but disappears during the night while he is in bed. The albuminuria is now copious but still varies, and casts may be more numerous, or may still be scanty and continue hyaline or faintly granular. The urine is now decidedly increased in quantity, 53 to 80 ounces (1600 to 2500 c. c.), its specific gravity low, 1005 to 1015. More rarely it is scanty. A cachectic anæmic condition develops. There is sometimes a peculiar fetor of the breath. Still later, all these symptoms increase; the dropsy is persistent, the urine loaded with albumin, and, in addition to the ordinary delicate hyaline casts, may contain the glistening waxy casts. Senator announced some years ago that serum-globulin is increased in the urine of amyloid kidney. During the past winter I have been enabled to make some observations which make me confident that he is right and that it is quite a valuable diagnostic sign. Fatty casts and free fatty epithelium from the tubules of the kidney may be superadded, as well as free oil-drops. Epithelial casts are rare, as is, also, blood.

Œdema of the lungs may also occur as a serious complication. Toward the close of the disease, the urine, which had been increased, becomes diminished in quantity, but is seldom suppressed. Of the solid chemical constituents, it may be said of all that they are, as a rule, slightly diminished, but not sufficiently to influence the course of the disease. It is in consequence of this that uræmia is almost unknown in lardaceous disease, the urea and extractives being eliminated in sufficient amount to avert this evil. Nor do we find hypertrophy of the left ventricle or high arterial tension.

But lardaceous disease of the kidney almost never occurs alone. It is always accompanied by similar changes in the liver, spleen, and often of the intestinal canal. Hence, evidences of alterations in these organs are more or less marked. Thus the percussion areas of the liver and spleen are almost always enlarged, and the blood-vessels of the stomach and intestines are often involved. In the former event obstinate vomiting and in the latter equally obstinate diarrhœa results. The latter is far more frequent than the former.

As to *duration*, the disease generally runs a very chronic course, which is limited only by the malady of which it is a complication. As such it is always of shorter duration than interstitial nephritis, and may be shorter than chronic parenchymatous nephritis, although the latter affection and lardaceous disease more closely resemble each other in respect to duration. When obstinate diarrhœa and vomiting supervene the end usually is not remote.

Diagnosis.—There are some instances in which lardaceous disease is easily recognized. If a patient has had syphilis with secondary and tertiary symptoms, or has long been a victim to phthisis, and he is discovered to be œdematous, and to have a large albuminuria, with waxy hyaline and fatty casts, and an enlarged liver and spleen, and obstinate diarrhœa, there can be little doubt but that lardaceous disease is present. But where neither of these two general diseases are present, or the phthisis has not existed a very long while, or there is not decided evidence of enlarged liver and spleen, we cannot be certain. While it is never safe to diagnose lardaceous disease without the presence of enlarged liver and spleen, the presence of these enlarged organs along with large albuminuria, and the other symptoms which attend it, do not necessarily imply amyloid kidney. The symptoms and course of the disease, particularly in its latter stages, are so like those of chronic parenchymatous nephritis that it is often impossible to distinguish the two. Further, there is every reason to believe that chronic nephritis is sometimes caused by the same dyscrasic conditions as produce the lardaceous disease. In such cases, therefore, a diagnosis is impossible. Finally, the two conditions exist jointly.

The only other form of renal disease which it is at all possible to confound with lardaceous disease is interstitial nephritis. But in this we have the almost total absence of dropsy, small albuminuria, and scanty sediment, in which granular and hyaline casts are found. While the quantity of urine is increased in both these forms of chronic Bright's disease, the quantity is larger in interstitial nephritis. Hypertrophy of the left ven-

tricle, an almost invariable symptom in contracted kidney, is very rare in lardaceous disease, while enlargement of the spleen and liver are common and do not occur in interstitial nephritis. Contracted kidney may also be associated with lardaceous disease.

Prognosis.—In the matter of prognosis much depends upon the presence or absence of the original disease causing the lardaceous change in the kidneys. If the former cannot be cured, the effect of the latter can only be to hurry on the unfavorable termination, although it is subject to the abatements as well as exacerbations of that affection. If the original disease is curable and the patient young, there are no limits to the possible improvement, although it is scarcely likely that the diseased structures are ever restored to their normal state. But as it is unlikely that all the renal vessels are involved in the change, and the organ itself, especially before its complete development is attained, is one capable of assuming an extraordinary degree of supplemental function, it is not impossible that there may be a complete restoration. If the patient is past middle life, even if it should happen that the original disease has disappeared, the probabilities of recovery are a minimum, while a decided degree of improvement is not impossible. If the stage of alteration of the blood-vessels of the stomach and intestines, as attested by obstinate vomiting and diarrhœa, is reached, the disease is necessarily rapidly fatal.

Treatment.—Of lardaceous disease it may be said with greater emphasis than any other form of renal disease, "an ounce of prevention is worth a pound of cure." A due appreciation by surgeons and syphilographers of the causes of lardaceous disease would prevent the occurrence of many cases, the timely amputation of a limb long the seat of suppuration and the thorough treatment of syphilis being all that is necessary to accomplish this. To this end also frequent examinations of urine should be made by the surgeon in charge of cases of the kind so often referred to, and the slightest indication of albuminuria should be the signal for prompt interference, if such be possible, while the possibility of the occurrence of this renal complication should always be before the surgeon's mind. Especially watchful should surgeons be who are in charge of children with hip-disease.

In syphilis the faithful and persistent use of remedies for a sufficient time after all symptoms of the primary and secondary affections have disappeared is essential. To this end the "continuous," rather than the "intermittent," treatment of syphilis by small doses of mercurials long continued is the plan most likely to secure the eradication of the disease, and this should be kept up for at least six months after the disappearance of all syphilitic symptoms. By small doses are meant doses of $\frac{1}{50}$ to $\frac{1}{25}$ grain (0.0013 to 0.0026 gm.) of the bichloride.

If the causing disease continues to exist, the treatment of the amyloid disease is the treatment of the former,—if it is syphilis, iodide of potassium and mercurials; if phthisis, cod-liver oil, iron, quinine, an abundance of nourishing food, in which milk and cream should be conspicuous, alcohol, and restorative measures generally, together with fresh air and suitable exercise. Supposing the original disease to have disappeared, the measures of treatment indicated are precisely those of

chronic parenchymatous nephritis, for the details of which the reader is referred to the chapter on that disease.

SUPPURATIVE INTERSTITIAL NEPHRITIS AND PYELO-NEPHRITIS.

SYNONYMS.—*Septic and Pyæmic Nephritis; Interstitial Suppurative Nephritis; Surgical Kidney.*

Definition.—Suppurative nephritis due to invasion of the kidney or its pelvis by pathogenic bacteria, either by way of the circulation or the urinary tract.

Most frequently this form of nephritis starts in the pelvis of the kidney as a pyelitis, and thence extends into the interstitial tissue of the organ. Such a condition is preëminently a *pyelo*-nephritis. It may also happen that the nephritis starts in the interstitial tissue of the substance of the organ as the result of infectious embolism or traumatism or obstruction of the tubules by concretions. In either event both the kidney and its pelvis are sooner or later involved, and it is scarcely possible to separate the two conditions in diagnosis, and I do not, therefore, separate the two diseases.

Etiology.—The most frequent medium of invasion by bacteria is retained decomposed urine. Retention may be due to stricture of the urethra or even phimosis, to stone in the bladder or ureter or pelvis of the kidney. Perhaps there are always bacteria ready to avail themselves of sufficiently favorable conditions, but a favorite route of introduction is by unclean catheters. In many of these cases inflammation of the bladder is an intermediate state. Calculous concretions in the substance of the kidney also furnish conditions favorable for the action of bacteria of suppuration.

Infectious emboli cause a small number of cases of suppurative nephritis. These emboli are usually derived from the valves of the heart in cases of mycotic endocarditis, but they may also arise in putrid wounds, stumps, or other seats of putrid inflammation. The abscesses found in the kidney in common with other organs in pyæmia are thus produced. Tubercle bacilli are also causes, entering by either of the routes named, producing tubercular pyelo-nephritis. Parturition is a not infrequent medium of introduction of pathogenic bacteria, while the infectious fevers are recognized causes.

Traumatic agencies, such as blows, kicks, or penetrating wounds in the neighborhood of the kidney, or falls from a distance and striking upon the sharp edge of a fence or similar object, may also cause suppurative nephritis.

Suppurative nephritis may occur at any age subject to the operation of the cause. The youngest patient I ever had was two years old.

Among the organisms found in the urine and held responsible are, besides tubercle bacillus, the bacterium coli commune, the proteus Hauser, the streptococcus and staphylococcus.

Morbid Anatomy.—The appearances vary necessarily with the stage of the disease and also somewhat with the cause. In an earlier stage, if the inflammation pass from below upward, as is most frequently the case, the mucous membrane of the pelvis is first affected, being swollen and dirty-grey in color, sometimes visibly congested. Later the pelvis and calyces may be dilated and the papillæ flattened. The distention may go on at the expense of the kidney until the whole organ is converted into a pus sac bounded by a varying remnant of renal tissue. Such sac may be a constant source of pus, or if complete obstruction occurs the pus may become inspissated and cheesy. The ureter is also often dilated, sometimes resembling in consequence of such extreme dilatation an intestine.

In tuberculosis extending *viâ* the urinary tract the apices of the cones are also invaded, it may be from the mucous membrane by continuity, or by direct lodgment of the bacillus. Successive portions of the kidney substance break down and the ultimate product will be the same,—a sac filled with liquid pus or cheesy, putty-like material.

In other instances, especially when the kidney is invaded by way of the vascular or lymphatic system, as in pyæmic abscess, foci of suppuration a millimeter and upward in diameter are scattered in the cortex, separated by sound renal tissue. They are surrounded by an intensely red border, are often visible through the cortex, and ruptured by dragging of the capsule. On section at an early stage linear streaks of pus may be found in the medulla.

At a later stage these little collections of pus unite to form larger ones, these again to form others still larger, destroying the tubular structure of the kidney as they encroach upon it; and it is at this stage that cases of pyelo-nephritis not unfrequently terminate unfavorably and the specimens come under observation. At first each of the abscesses thus formed is confined to the region of a single pyramid, and it not unfrequently happens that a kidney is partitioned off into spaces corresponding with these. Before this occurs, however, the abscess bursts through the papilla and calyx into the pelvis of the kidney. Thus, in an opposite direction from that first described, the kidney may again be converted into a purulent sac, but these cases generally terminate fatally long before this is attained.

Where the abscess is embolic in origin its seat is at first occupied by an area of intense hyperæmia, resulting in hemorrhagic extravasation, which takes place also into the tubules, causing bloody urine. To this succeeds suppuration. The size and number of the abscesses depend upon that of the plug obstructing the blood-vessel, which is usually one of the interlobular arteries or a vas afferens. The embolic abscesses may also be multiple in consequence of the breaking of the embolus into a number of minute fragments. Where the cause is traumatic the process is not so easily defined. Circumscribed abscesses may occur, or the kidney may be converted into a soft, pulpy mass, a mixture of pus, blood, and broken-down renal substance.

Symptoms.—The symptoms of this condition are not numerous, and, apart from the characters of the urine, are not very distinctive.

In milder degrees of pelvic inflammation before the kidney is invaded there may be none. *Pain* and tenderness are the most constant, but considerable inroads may be made upon the structure of the kidney before pain results. On the other hand, it is often very severe, while the *tenderness* over the region of the kidney is pronounced. This tenderness is the most distinctive and valuable symptom. Usually the severest pain is in the renal region, whence it radiates toward the front of the abdomen and groin, and may be accompanied by retraction of the testicle. Where the condition is the result of impacted calculus, the seat of the impaction is the primary seat of pain. It may be between the umbilicus and the abdomen when the stone is lodged low down in the ureter. The pain is always intermittent to a degree, sometimes totally so, but generally it is more or less constant, paroxysmally increased. Various positions are assumed by the patient with a view to easing the pain, among which that on the face is not infrequent.

A distinct *tumor* can sometimes be discovered in the region of the kidney by palpation and percussion. This implies an enlargement of the organ, due either to its conversion into a purulent sac or an augmentation of its size, owing to the distention of its pelvis with pus or calculi or both. Very frequently it is due to perinephric invasion.

Fever is also an intermittent symptom. Possibly in a very few latent cases it may be altogether absent, but except in these there is always elevation of temperature, with corresponding frequency of pulse. These latter at times become decided, and in advanced stages the fever is septic, being followed by profuse *sweats*. In acute cases, especially pyæmic, the beginning of suppuration is often marked by a *chill* and *high fever* or succession of chills, but in other instances it is quite impossible to recognize the beginning of the suppurative stage.

The *characters of the urine*, as intimated, are more distinctive. This, except in acute infectious cases, almost invariably sooner or later contains pus, and unless it does contain pus no certain diagnosis can be made. *Blood* is also a very constant constituent of the urine from cases of suppurative nephritis, but while such urine is scarcely ever examined by the microscope without discovering a few blood-discs, yet the quantity is often not large enough to be recognizable to the naked eye. The quantity of *pus* also varies greatly. While it may be so copious as to produce a heavy white opaque deposit, one-sixth to one-fifth the bulk of urine, it may be represented by little more than a trace. This variation will also occur at different times in the same case. Pus from the kidney and its pelvis is usually distinguished from that formed in the bladder by the absence of that glairiness so characteristic of the latter, due to admixture with mucus and decomposition products. Pus from the pelvis of the kidney is also rarely fetid as compared with pus from the bladder.

The urine is also usually *diminished in quantity*. Complete suppression is not uncommon toward the close of cases presenting extreme degrees of destruction. Notwithstanding such diminution, the color may be pale and the specific gravity low, owing to the small proportion of urea present, the range of specific gravity in a single case being from

1003 to 1016. In reaction the urine is faintly acid, neutral, or alkaline, and though often prone to rapid decomposition, this is a much less characteristic feature than in cystitis. It *is always albuminous*, but the quantity of albumin is never excessive, and varies generally *pari passu* with the quantity of pus and blood. When more albumin is present than is thus accounted for, it is evidence that the tubular structure of the kidney has become involved. Such cases are, therefore, more serious. In such cases, too, *tube casts*, ordinarily very rarely found in suppurative nephritis, may appear.

It sometimes happens that there is a sudden increase in the quantity of pus in the urine, followed by a gradual diminution, or the urine, previously clear, may suddenly become loaded with pus. Such occurrences indicate the probable period of *rupture of an abscess* through a papilla and a pouring out of its contents into the pelvis of the kidney, or of the removal of a temporary obstruction to the descent of the pus. It may happen that a small portion of the substance of the kidney is thus discharged with the urine, when it may be recognized by microscopic examination, which will discover the tubules and glomerules of the kidney. Occasionally, also, the abscess, instead of rupturing into the pelvis of the kidney, perforates into the perinephric tissue, burrowing in different directions and producing fistulous openings. Perforations may thus take place posteriorly in the lumbar region, or anteriorly at the groin, into the colon, and more rarely into the lungs and liver, and even peritoneal sac.

The *course* and *duration* of suppurative nephritis are very various. Traumatic cases are comparatively rapid, either toward recovery or death. Pyæmic cases may run their course in forty-eight hours, and are invariably fatal. But cases due to other causes, viz., impacted calculus, tuberculosis, stone in the bladder, or cystitis, may be prolonged indefinitely, while some terminate without being discovered. Sooner or later the patient generally succumbs to exhaustion, but life may be sustained for years with paroxysms of the severest suffering and a surprising degree of destruction of the kidneys. The greatest danger to those thus affected is intercurrent illness, which is always more serious and more apt to terminate unfavorably. It is then that the kidneys, previously surprisingly sufficient in eliminating power, give way in this respect, the symptoms of uræmia supervene, and the patient dies of this complication. It is well known that the operation for stone is much more apt to be followed by a fatal result when the subject happens to have a surgical kidney.

There are no *complications* peculiar to suppurative interstitial nephritis other than those mentioned as causing it or as resulting from unusual accidents of rupture, of abscess, and perforation of neighboring organs, or from uræmia.

Diagnosis.—The diagnosis of suppurative nephritis may be easy or difficult. It is easy when there is the history of a traumatic cause followed by hæmaturia, and later purulent urine, with tenderness and pain over the region of the kidney. On the other hand, while prolonged inflammation of the bladder, stone in the bladder, nephro-lithiasis, or other causes of obstruction and decomposition of urine always afford

good reason for suspecting that suppurative nephritis exists, it is not easy to find data on which to rest a positive belief in this. If, however, the urine contains pus constantly or intermittently, and in addition to this there is pain or tenderness in the renal region, suppurative nephritis may be averred with reasonable certainty. I know of no distinctive cellular elements from whose presence in the urine it may be asserted that pus comes from the pelvis of the kidney. For though the little columnar or pear-shaped cells are referred to the pelvis of the kidney and ureter, they also come from the urethra and bladder.

As to differential diagnosis, the only certain means of recognizing the tubercular form is by the recognition of the bacillus in the purulent urine, and this is facilitated by the use of the centrifugal apparatus. Should the symptoms described occur in a case of tuberculosis of the lungs, the tubercular nature of the nephro-pyelitis becomes quite probable.

Pyelo-nephritis is distinguished from paranephritis by the more circumscribed shape of the tumor, the absence of œdematous infiltration of the lumbar region, and by the presence of purulent urine, unless it happens, as it rarely does, that the paranephric abscess breaks into the kidney and discharges by the ureter. Pain on flexing and rotating the thigh is characteristic of paranephric abscess because of the involvement of the psoas muscle. Pyæmic abscesses of the kidney may be suspected if a pyæmic process is present and a chill supervene, followed by more or less of the renal symptoms described.

Prognosis.—Operation has done much to improve prognosis of late years. Yet so far as recovery is concerned, it is still in most cases unfavorable. Traumatic cases may recover if the injury is not too extensive, while very grave injuries are usually rapidly fatal. So may cases succeeding infectious fevers and pregnancy. Cases due to obstruction of the ureters cannot get well as long as the obstruction and irritation continue, and as their removal is often impossible such cases gradually grow worse. On the other hand, their fatal termination may be indefinitely delayed. It is often a matter of astonishment, on viewing the post-mortem appearances of cystic purulent kidneys, that the patient has lived as long with the extreme structural changes which present, the barest remnant of secreting structure being sometimes found. It is impossible to say how far repair may take place if the cause can be removed. Conditions of this kind occur where a stone has been removed after having been long present either in the bladder itself or in the kidney or ureter. It is scarcely necessary to say that such persons are in imminent danger from the effect of cold, acute disease, or other cause which tends to suppress the action of kidneys already crippled in their function.

Treatment.—As operation offers the best chance of cure in many cases, a surgeon should be early called. There is no curative treatment by medicine for suppurative nephritis without a removal of the cause, and as the latter is often impossible, it follows that medical measures are mainly palliatives. One of the most frequent indications is the relief of pain, which is often so severe as to call for powerful anodyne measures,—opium and its alkaloids being absolutely essential. Hypodermic injections of morphine in doses of $\frac{1}{8}$ to $\frac{1}{3}$ grain (0.008 to 0.022 gm.) repeated,

if necessary, are favorite and effectual methods of relieving the intense pain, which is often due, not so much to the inflammation as the impacted calculus or other cause of obstruction. Suppositories of ½ to two grains (0.03 to 0.13 gm.) of the extract of opium may be substituted. Hot fomentations and simple counter-irritants, such as mustard, are also valuable adjuvants.

The catarrhal process in the kidney, its pelvis, and the ureter, and also in the bladder, may be treated with varying success. Diluents are decidedly indicated, and for this purpose any one of the numerous negative mineral waters can be used. The alkaline mineral waters are contraindicated, as they inspissate the pus and make it more difficult to pass. The only remedies I have found of any use are the balsams and benzoic acid. Of the former I prefer sandalwood oil, because better borne by the stomach than copaiba. Given in gelatin capsules, each containing ten minims (0.65 c.c.), of which one or two may be taken three times a day, it sometimes has often a decidedly beneficial effect upon the catarrhal inflammation, seen in the diminished amount of pus. Benzoic acid fulfils another indication, that of securing an acid reaction of the urine, which is very often either alkaline or so faintly acid that it rapidly becomes alkaline, and thus predisposes to decomposition. The benzoic acid is best given in the form of capsules. For an adult three or four five-grain (0.32 gm.) capsules daily are usually sufficient to keep the urine acid. Larger doses than these may be given. It may be given either alone or in conjunction with the sandalwood oil, the former being given before and the latter after a meal. To children smaller doses may be given. To them one may begin with one grain (0.006 gm.) three times a day and increase it. The various vegetable diuretics, as buchu, pareira brava, etc., are rather harmful than useful in suppurative nephritis and pyelitis.

The constant and inevitable tendency in these cases to run down in general health, in consequence of the drain and wear and tear to which they are subject, demands tonics such as quinine, iron, and strychnine, while milk and other nutritious articles of diet are always indicated. The dangers to which the patient is subject from exposure, cold, and dampness should be averted by suitable care and woolen clothing.

I desire to repeat that many of these cases are saved at the present day by a timely operation of nephrotomy, by which the pus is discharged, the kidney drained, and calculi, if present, removed. I look back upon many cases allowed to perish whose lives would at least have been materially prolonged by operation.

PARANEPHRITIS OR PERINEPHRIC ABSCESS.

Definition.—Paranephritis is an inflammation invading the connective-tissue about the kidney, terminating almost always in suppuration.

Etiology.—A number of causes may be responsible for perinephric abscess. Thus there may be rupture of a nephritic abscess through the capsule of the kidney; perforation of the bowel, most frequently seen in appendicitis; extension of suppurative disease from the spine, as in caries of the vertebræ, or from a burrowing empyema; finally, blows and injuries may terminate in suppuration about the kidney.

Morbid Anatomy.—At autopsy the kidney is found surrounded by pus, which is usually posterior to it, rarely in front between the kidney and the peritoneum. The pus has often a fecal odor from contact with the large bowel. It may burrow in various directions and even burst into the pleura and be discharged by the lungs; or it may work its way to the groin and appear at Poupart's ligament. It in turn may perforate the bowel or rupture into the peritoneum, bladder, or vagina. Occasionally the fatty bed of the kidney is found to be converted into a fibrous capsule fused more or less closely with that of the true kidney capsule.

Symptoms.—Most cases are secondary to disease in the neighborhood. *Pain* and *tenderness* in the region of the kidney are the most constant symptoms. In addition, there is a peculiar *œdematous* or *boggy condition* in the same locality, giving rise to pitting on pressure. The thigh is flexed to relax the psoas muscle, tension of which, especially in adduction, increases suffering. The patient, if able to walk, relies as much as possible on the opposite leg, on which he leans, assuming also a stooping posture with the spine fixed. The whole attitude and behavior of the patient remind one of hip-joint disease, while the pain may even be referred to the knee, as in this disease. These symptoms do not, however, appear at once, and the approach is often insidious. At other times suppuration is ushered in with chills, fever, and sweats. The plastic form of fibroid paranephritis is without distinctive symptoms.

Diagnosis.—The diagnosis from nephritic abscess, with which it is most likely to be confounded, has been considered. The attitude of the patient in lying or standing is like that in hip-disease, but the history elicits that in its incipiency the pain is much higher up in perinephric abscess, while examination shows that the swelling and tenderness are above the hip and not over the hip-joint itself. As most cases except those due to injury are secondary to disease in the neighborhood, it does not much matter about separating the two classes. Secondary forms are more sudden in their onset, though this is not always the case. Doubtful cases may be settled by the exploring needle.

Treatment.—This is by section and free drainage, for though spontaneous rupture sometimes takes place it is apt to be preceded by destructive and dangerous burrowing, which should be anticipated by operation.

TUBERCULOSIS OF THE KIDNEY.

Morbid Anatomy.—Tuberculosis presents itself in the kidney in two forms :—

(1) In the shape of miliary granulations which are a part of a general tuberculosis, giving rise to no special local symptoms.

(2) As primary foci of localized tuberculosis which in time may fuse to form larger areas, which undergo caseation and liquefaction, transforming the whole kidney at times into a sac of purulent or cheesy matter. Such tuberculosis may start in the prostate, bladder, ureter, or pelvis of the kidney, and may extend also into the testicle and epididymis in men and the ovary and fallopian tubes in women.

Symptoms.—The first form is without special symptoms. There may be none at all or *they may simulate closely those of nephro-lithiasis.* Those of the second, so far as the neighborhood of the kidney is concerned, are not distinctive or constant. There may be none or there may be *fulness*, *tenderness*, and even in extreme cases *fluctuation.* Frequently subjective symptoms are reflected to the bladder, and they include frequent micturition, pain, and tenderness in the region of the bladder. There is also purulent urine, but commonly this differs from that of cystitis. It is more constantly acid in reaction, and contains pus less contaminated with mucus. Blood is much more frequent than in simple cystitis, and correspondingly albumin. Tube casts are very rarely found. Cheesy masses are sometimes found and the tubercle bacillus, which is the only pathognomonic sign. It should always be sought. The method for its recognition is the same as for the tubercle bacillus in sputum. A negative result does not, however, exclude tuberculosis. In such event Damsch suggested inoculation with the pus from the urine into the anterior chamber of the rabbit's eye. At the end of three weeks tubercular nodules should make their appearance if the pus is tubercular. Sometimes, also, shreds composed of white fibrous and elastic tissue representing the disintegrating kidney or mucous membrane are found in the urine, but are not diagnostic, since they may be found in other varieties of destructive disease of the organ. The features of the urine described are almost characteristically intermittent, that is, the urine is sometimes almost or quite clear and again becomes purulent.

In the absence of such conclusive proof as bacilli in the urine, the presence of tubercle elsewhere, as in the lungs or nearer parts, as the testicle and prostate in man or the ovaries and fallopian tubes in women, affords suggestive evidence. The latter may be investigated through the vagina and rectum, while catheterization of the ureters may also be practised in women and stenosis of the ureter due to tubercular infiltration of the pyelo-uretal wall thus recognized. Even in men the thickened ureters may rarely be felt through the abdominal wall. In other cases where the lungs are not primarily tubercular they may be secondarily invaded. Hydronephrosis, it is said, may result from complete obstruction of the ureter by tubercular infiltration.

Treatment.—Beyond the general restorative and palliative treatment

useful in general tuberculosis there is no medical treatment of tubercular kidney. As soon as the diagnosis is made the surgeon should be called and nephrotomy done. Life is almost invariably prolonged by it, and if the operator be so fortunate as to find only a few isolated nodules on section, they may be scraped away. In cases in which the whole organ is involved a persistent renal fistula must be expected. Exploratory operation may even be justified under circumstances which must be determined in each case.

NEPHRO-LITHIASIS—STONE IN THE KIDNEY.

Definition.—Nephro-lithiasis means "stone in the kidney." But the term is a general one, which covers the presence in the kidney, its pelvis, or ureter of concretions large enough to justify the term "stone," of smaller masses appropriately known as "gravel," and fine particles known as "sand."

Morbid Anatomy.—Except in the case of "sand," which includes particles made up either of pure uric acid or oxalate of lime, gravel and stone as found in the kidney and its pelvis always have an organic basis through which are distributed the mineral matters which go to make up their bulk, and which remains as a framework after the mineral matters are dissolved out. The matters thus precipitated, in some one of the shapes named, in the order of frequency are :—

(1) Uric acid and its compounds of sodium, ammonium, and potassium.

(2) Oxalate of lime.

(3) The phosphates of calcium and of ammonium and magnesium.

Only in the case of uric-acid stones, of small size, and of oxalate-of-lime stones, likewise moderate in size, do we have the bulk of the stone made of a single constituent. More frequently it is the case that uric-acid or oxalate-of-lime stone forms the nucleus, and about this aggregate in concentric layers the phosphates, which make up the great bulk of all large stones as well as some stones in their entirety. More rarely a uric-acid nucleus is surrounded by oxalate of lime or the reverse. Not only may the sediments named be the nuclei of stones, but foreign matters, such as a clot of blood or a fragment of any kind of matter accidentally reaching the urinary passages, may also play a like rôle.

The steps for determining the more precise composition of stones will be found in appropriate manuals on the examination of urine, but the three principal varieties present certain physical characters by which they can with considerable certainty be determined. Thus *uric-acid stones* are usually smooth or lobulated, of a dark red or reddish-brown color, hard in consistency, and rarely acquire a size of a centimeter (0.4 inch) in diameter, while many of them are no larger than a lentil. They may be multiple. *Oxalate-of-lime stones* are very hard and uneven, so characteristically so that they have received the name mulberry calculi, from their resemblance to this fruit. Their hard-pointed projections produce exquisite pain in transit from the kidney through the ureter into the bladder. They attain about the same maximum size as uric-acid stones and are also

often multiple. The *phosphatic stones* are white in color or grayish-white, quite soft, easily disintegrated, may often be crushed in the fingers, though at other times they are much harder. They form the largest stones, attaining the size of a hen's egg or larger. When stones lie in the ureter rather than the pelvis of the kidney they are apt to be more elongated, or sometimes spindle-shaped, and present sometimes a spiral marking which is characteristic. Other stones are moulded to the shape of the pelvis of the kidney with a prolongation for each calyx, which may be further branched—the dendritic or coral calculi. Rarer forms of calculi are made up of cystin, xanthine, carbonate of lime, and urostealith.

Symptoms.—It sometimes happens that a stone is found *post-mortem* in the substance of the kidney or in the pelvis which was not suspected, but it is hardly likely that even in these cases symptoms were not present. They were simply overlooked and ascribed to some other cause. The most constant symptom of nephro-lithiasis is *pain in the region of the kidney* associated with more or less *tenderness*. Like the pain of stone in the bladder, it is *aggravated by motion*, especially rough motion, and there are certain positions of the body in which the patient is made more or less uncomfortable. Quite often the inflammation caused by the stone proceeds to suppuration, and the whole of the kidney, more or less, is substituted by a pus sac. The pain is often suddenly aggravated when a large stone so lodges as to plug up the ureter and interfere with the descent of urine, or a small one descends through the ureter into the bladder. Under these circumstances come the attacks of so-called nephritic colic, characterized by a pain which may equal in severity any to which man is subject. It has other distinctive features. It radiates downward into the groin and the neighborhood of the bladder and down the inside of the thighs and into the testicle, which is often retracted. Sometimes it extends upward toward the diaphragm, and it is not always easy to separate the pain of nephritic colic from that of hepatic colic when the former is on the right side. It may happen that both kidneys are the seat of impacted stone, though this is a very rare event. There is often nausea and vomiting, a cold sweat appears, and the patient is collapsed.

After the pain and colic the changes presented by the *urine* may be highly distinctive, aiding greatly the diagnosis, at other times the urine is absolutely negative. It very frequently contains blood, though the quantity is often small, and may be demonstrable only by the microscope. Especially in connection with fresh attacks of nephritic colic is there blood. Pus is almost always present in small or large amount, but less constantly than in stone in the bladder. Cylindroids or mucous casts may be found, true casts very rarely. In some cases, too, uric-acid crystals in the shape of red or brickdust-like particles either before or during an attack of nephritic colic point to the uric-acid nature of the stone. The same is true of oxalate-of-lime crystals. In the case of the last two substances the urine is acid in reaction. Phosphatic stones may be suspected if the urine is alkaline in its reaction, as it is only possible for phosphatic sediments to form in the presence of an alkaline urine, while uric-acid crystals can only remain permanent in an acid urine. Oxalate of lime, the most insoluble of all crystals, occurs, however, in either acid or alkaline urine.

In cases of gravel or sand as contrasted with stone, there are no symptoms as a rule between attacks, as the stone must reach an appreciable size before it produces the constant or almost constant pain characteristic of it. In some cases there is complete suppression of urine, even where the kidney on the opposite side is normal, though more frequently when it is diseased, and death from uræmia may occur in consequence.

Diagnosis.—It has been intimated that nephritic colic may be confounded with biliary colic. Usually the symptoms of each are sufficiently distinctive. The presence of jaundice in biliary colic is invariable and comes on very soon after the obstruction begins. The stools are without bile, greyish-white in color. Usually the pain is more toward the epigastric region as a centre, and thence through the upper abdomen and perhaps through to the right shoulder-blade. The urine is also bile-stained and responds to the tests for the coloring-matter of bile. Other substances, however, than stone may produce nephritic colic. Thus clots of blood may obstruct the ureter and give rise to all the pain occasioned by an impacted stone, and in the absence of a history of stone and of hemorrhage there are no symptoms by which the two causes of colic can be separated. In the case of hydatid cysts of the kidney, fragments of these, too, may be discharged, and in suppurating kidney inspissated pus may block up the ureter. I have in my possession some striking moulds a centimeter in diameter and several centimeters long, composed of such matters, which plugged up the ureter for a time and were subsequently discharged. Renal colic has been mistaken for intestinal colic, but such confusion is dissipated as the condition continues. Renal colic is simulated when from any cause the ureter is compressed, as by a twist in the ureter of a floating kidney or compression by a tumor. The symptoms of stone in the kidney sometimes closely resemble those of stone in the bladder, but the pain in the latter though radiating to the back goes into both sides; in stone in the bladder the urine contains more mucus and is alkaline or becomes readily so, while in nephro-lithiasis the pus is purer and the urine acid. In all cases pointing to the presence of stone in the bladder the sound should be promptly used.

Prognosis.—The prognosis of stone in the kidney is very much more favorable now than it was a dozen years ago in consequence of the safety with which operations can be performed. The kidney may be exposed, split open, and the parts reapposed and restored with perfect recovery, and whenever the diagnosis of stone in the kidney is made an operation should be done; while where many severe attacks occur an exploratory operation is sometimes justified for the sake of diagnosis. Where the sand or gravel is so small as to pass the ureter the suffering terminates with its entrance into the bladder.

Treatment.—It is needless at the present day to say there are no medicines which, when administered, are capable of producing solution of a stone if it be of any size. In cases where there is no such formation and we have simply to contend with gravel or sand, therapeutic measures to prevent its further formation and even in some cases to promote solution may be availed of.

It is, however, a matter of extreme importance before treatment is

instituted to know exactly the sort of sediments which are to be treated, and to this end the urine should be carefully studied. Thus uric acid is dissolved by alkalies, and the object of treatment should be to alkalize the urine as much as possible with a view of keeping the uric acid in solution. On the other hand, to give alkalies to the patient with phosphatic stone or sediment is only adding fuel to the flame. As to oxalates, they are equally insoluble in both acids and alkalies, but there is reason to believe that they are produced under the same conditions which produce uric-acid sediments, and the treatment is therefore the same for either.

The required alkalinity of the urine for the solution of uric acid may be produced by any one of a number of ways ; perhaps the most desirable of all is the free use of alkaline mineral waters represented by the foreign waters of Tarasp, Vals, Vichy, and Ems, and in this country approximated by Saratoga Vichy and geyser waters, which may be termed alkalo-saline waters. It is unfortunate that in this country we have no more sources of the alkaline mineral waters, at least east of the Rocky Mountains, and the alkaline waters of the West are so strong that it would not be possible to use them without dilution, and very few physicians seem to have had any experience with them. In the absence of true alkaline waters the numerous negative mineral waters which are so strongly recommended by their owners may still serve a useful purpose as diluents and solvents, which are very necessary in the treatment of these diseases. They are almost too numerous to mention, but the Bedford, Poland, Waukeshaw, and Buffalo lithia waters are among the best known. In a few cases they alone may be sufficient if used in sufficient quantity. They may be rendered more active by combining alkaline salts, such as the citrate of potassium and the bicarbonate of potassium and lithia. The lithia waters particularly for sale in this country contain uncertain, at most very small, quantities of lithia. I think it much better, under the circumstances, to add a definite amount of lithium carbonate or citrate to a glass of water, say five or ten grains (0.3 to 0.6 gm.). At least one quart (two liters) of any of the true alkaline waters should be used every twenty-four hours by an adult subject to uric-acid sediments, and a corresponding or larger quantity of the more negative waters referred to. Liquor potassii of the U. S. Pharmacopœia is a very excellent means of counteracting acid tendencies. It may be given in doses of 15 minims to ½ a drachm (one to two c. c.), and milk is a very suitable vehicle. Piperazin, a comparatively recently introduced solvent for uric acid, undoubtedly possesses this power outside of the body, and in a few cases it has also seemed to me of service when administered to patients with uric-acid gravel. Some assert and others deny its efficacy. Its costliness is in the way of its general use in sufficient quantity to test its real value. All that has been said of treatment of the uric-acid diathesis may be applied to that of the oxalate of lime.

Much also may be done by diet. Persons with uric-acid diathesis should limit the amount of nitrogenous food, using a minimum of meat and cultivating the use of milk and vegetables as a diet.

The phosphatic tendency is, unfortunately, not so easily combated, as our resources for the purpose of acidifying the urine are very limited.

Only two drugs, benzoic acid and boric acid, have this reputation, and they possess it to a slight degree only, while large doses of these drugs are not well borne by the stomach. From five to ten grains (0.3 to 0.6 gm.) each may be given three, four, or five times a day as borne by the stomach. Here, too, there is, of course, the same indication for the free use of diluent drinks, and in these cases the negative mineral waters are as good as any, while perhaps distilled water serves as the type of a suitable solvent. Where the composition of the stone is unknown it is better to use this class of waters rather than alkaline waters, lest the stone happen to be phosphatic, in which event alkaline waters will cause further deposit. While the mineral acids cannot, of course, be given in such dose as to produce an acidity of the urine, they are still useful in counteracting the tendency to high degrees of alkalinity.

In the matter of diet, too, the phosphatic diathesis requires an opposite treatment. Meats are here indicated, while milk and vegetables may form a less abundant part of the food because of their tendency to alkalize the urine.

In treating the uric-acid diathesis with alkalies it is possible to carry the effect too far and cause deposit of phosphates where previously uric-acid sediments were precipitated. It is important, therefore, to watch the urine carefully and to avoid such an administration of alkalies as will cause phosphatic sediments to make their appearance. It is well known that stones are found in the bladder especially which exhibit alternate layers, now of uric acid and now of phosphates, although the latter substance almost invariably makes up the bulk of calculi which have attained any size. The rationale of this is perhaps as follows: as soon as the uric-acid gravel or oxalate-of-lime gravel becomes large enough to act as an irritant it increases the organic matter in the urine in the shape of pus and mucus. These matters, it is well known, favor an alkaline reaction, so that the little stone becomes surrounded, as it were, by a stratum of phosphate precipitating urine, and the larger it grows the more apt is this to be the case. Where alternate layers exist it may be inferred that the natural acidity reasserts itself and throws down the uric acid and urates, which in turn form a layer about the stone.

The extreme pain of attacks of nephritic colic must be combated by anodynes, and for this purpose rarely is anything except opium and its alkaloids sufficient, while the hypodermic mode of medication is far the best, both by reason of the promptness of its action and the less serious effect upon the stomach. It is rarely the case that less than ¼ grain (0.016 gm.) of morphine thus administered is sufficient, and very frequently this dose has to be repeated very soon. It has happened to me that where the pain was less severe I could control it with full doses of phenacetin and acetanilid, or by the aid of these substances render smaller doses of morphine sufficient. For this purpose not less than 15 grains (one gm.) should be given. Local applications are, of course, useful to a certain extent in relieving pain, especially hot poultices, which may be applied to the lumbar regions and to the groins, while hot baths are of great service in relaxing spasm and allaying pain due to it.

The escape of the stone from the ureter into the bladder is followed

by unspeakable relief, and its discharge from the urethra usually follows sooner or later with little or no pain, although a stone of considerable size may lodge in the urethra and require extraction. Where stones are not discharged from the bladder, they are likely to become the nuclei of larger stones in the bladder, where, of course, the largest majority of stones acquire their growth.

Stones too large to pass *per vias naturales* should be cut out of the kidney. It should be remembered, however, that after apparent well-founded diagnoses it sometimes happens that no stone has been found on section. Yet, strange as it may seem, it frequently happens that the symptoms do not return after such negative operation. Such cases, on the other hand, may turn out to be tuberculosis of the kidney.

TUMORS OF THE KIDNEY.

Definition and Application.—The term "tumor of the kidney" is applied to almost any enlargement of the kidney due to the presence of morbid growths. Yet there are morbid growths of the kidney which are not sufficiently large to produce appreciable change in size. Thus the adenoma does not usually exceed 0.4 inch (one cm.) in diameter, though it may be two inches (five cm.) or more. The same is true of the angiomata and leucæmic tumors, of fibroma and lipoma, which sometimes form small white nodes in the fibrous tissue near the bases of the pyramids. Lymphadenoma occurs in the kidney associated with similar disease of lymph-glands, liver, and intestine. Villous papilloma sometimes grows in the pelvis of the kidney. Syphilitic gummata also belong to the group of moderate-sized tumors rarely producing symptoms.

Cysts, single and multilocular, acquire larger size, producing appreciable enlargement in the kidney. The malignant tumors, sarcoma and carcinoma, belong to this category, and with cysts are, clinically speaking, perhaps the chief occasion of the term "tumor of the kidney." On the other hand, renal abscess or pyonephrosis does not usually earn for the kidney involved the name "tumor," while hydroneprosis does.

Symptoms.—Certain local symptoms are produced indifferently by any one of the tumors large enough to become clinically appreciable. In the first place, *renal tumors grow* for the most part *forward* rather than backward because of the more yielding character of the parts in front of them than behind them, rarely producing posteriorly much more than a fulness of the back, in the course of which there is obliteration of the resonance which may be present between the kidney dulness and the vertebral spines. As this forward growth proceeds, a special effect results from the relation of the bowel to the kidney; as the ascending *colon* on the right side and the descending colon on the left lies *in front* of the corresponding kidney, the effect of enlargement of this organ is to push the bowel in front of it, the hollow viscus being recognized by the tympanitic percussion note. In this respect renal tumor differs from that of the spleen, and less invariably from that of the liver, since the bowel does not intervene between these organs and the abdominal wall, though, rarely, the

small intestine may float between the liver and the parieties. Commonly, too, the hand may slide between the tumor and the liver on one side and the tumor and the spleen on the other, while the renal tumor never loses the rounded border characteristic of this organ.

By bimanual palpation with the palm of one hand placed in the lumbar region and the other in front below the ribs, pressure being made in both directions, the tumor may be recognized. In this examination, too, it will be noted, that it is subject to a much more limited degree of mobility during breathing than is the liver, although it is not totally *immobile*. The renal tumor, too, commonly resists lateral movement.

Again, *pain* in the region of the kidney is an inconstant symptom. It is often totally absent, and again exceedingly severe, especially if the vertebræ and spinal cord are encroached upon. It sometimes happens that in consequence of the pressure of the kidney upon the 12th dorsal nerve and branches of the lumbar plexus, neuralgic pains in the abdominal walls are produced.

Diagnosis.—As to differential diagnosis, the two varieties of tumor which constitute the malignant growths, CARCINOMA and SARCOMA, alone produce hæmaturia, though it is not even a frequent symptom with them. In the event of its presence the blood may be fluid or clotted, and is often moulded in the pelvis of the kidney and ureter, an event very rare in the blood poured into the pelvis under other circumstances. Hæmaturia is more frequent in carcinoma than sarcoma. These two tumors are the most likely to produce pain. It is sometimes extreme and boring, when it indicates destructive encroachment on the vertebræ. More frequently it is dull, radiating over the flank into the thighs. These tumors also produce cachexia. Very rarely they may be recognized by the presence of distinctive histological elements in the urine. This occurred to me in at least two instances. Frequently the urine is altogether negative.

Of these forms of growth, between which no distinction was made until thirty years ago, the sarcoma is now regarded as the more frequent. Both may be primary or secondary, more frequently secondary. Sarcoma is a disease of early life, in fact, it is often congenital, when it is represented by that form known as rhabdomyoma, which contains striated muscular fibres. In more than half the cases it affects children under ten. On the other hand, renal cancer is not confined to later life, as is other forms of cancer, and it may even occur in children. May not these tumors also have been sarcomata? More usually one kidney only is affected. Such organ is uneven, soft, even to a sense of fluctuation. Carcinoma also selects one kidney whence it may invade the pelvis and ureter. It affects the general health more rapidly, hæmaturia is more frequent and copious but also intermittent. If there be a superficial primary growth elsewhere and a renal tumor is present, the presumption is that it is of the same nature, but no certain diagnosis can be made between carcinoma and sarcoma unless the rare opportunity occurs to examine fragments discharged with the urine. Unfortunately for diagnoses, the urine is too often quite free from any sediment, even of pus. Carcinoma of the kidney is apt to invade the renal veins and even the vena cava, and as such to cause metastasis in the lungs and in other organs as well.

From ovarian tumors renal tumors are distinguished by the fact that in the former the intestines lie in the flanks, giving resonance on percussion in that locality, while an enlarging kidney pushes the bowels in front of it. The ovarian tumor also grows from below upward and drags with it the uterus and appendages, as can be recognized by vaginal examination and also by rectal touch.

Much more difficult is it to distinguish the renal tumor from enlargement of the retroperitoneal glands, as the latter also pushes up the intestines in front of it, giving rise to a tympanitic percussion note. The absence of blood in the urine is constant in retroperitoneal tumor, while it may or may not be present in renal tumor. The retroperitoneal tumor may press upon the ureter and the renal vessels and thus produce obstruction to the descent of the urine and changes in it. The central situation of the enlargement in retroperitoneal tumors contrasts with the lateral growth in renal tumors.

From tumors of the liver renal tumors differ in that the former cause sooner or later a bulging of the right hypochondriac region, while the renal tumors rarely reach as high as to alter the configuration of the lower thorax. The sharper border of the liver tumor as contrasted with the rounded edge of the kidney tumor is characteristic, while the freer movement of the liver with breathing has also a value. Splenic tumors are not likely to be confounded with tumors of the left kidney. Splenic tumor protrudes from above downward and toward the umbilicus instead of from the lumbar region forward. It moves more with breathing and its sharper edge and indentation may be recognized. It is always above or outside of the colon.

Treatment.—The renal tumor is beyond curative treatment by the physician. As soon as the diagnosis is made a surgeon should be called and the question of operation considered.

CYSTS OF THE KIDNEY.

Reference is here made only to such cysts as produce clinically appreciable enlargement of the organ. They include :—

(1) *Retention* or *obstruction cysts*, solitary cysts ranging in diameter from a centimeter (0.4 in.) to ten centimeters (four in.) and larger. They may be present in one or both kidneys. These are probably primarily the result of stenosis of a uriniferous tubule behind which accumulates first urine, which is gradually substituted by an aqueous fluid in which may be found traces of urinary constituents. A trace of albumin may also be present. These cysts rarely give rise to symptoms.

(2) The *congenital cystic* kidney, in which both organs are the seat of numerous round cysts varying in size from $\frac{1}{5}$ inch (0.5 cm.) to one inch (2.5 cm.), producing tumors of large size, so large, in fact, that they have interfered with parturition. They contain sometimes a clear, sometimes a turbid, fluid, sometimes colloidal in consistence and containing albumin, cholesterin, triple phosphates, rarely urea and uric acid, and sometimes fat-drops. Persons with these cysts sometimes grow to adult life, and, indeed, such cystic kidneys have been found post-mortem when not sus-

pected. Commonly the subjects die either before birth or shortly after. The exact mode of origin of these congenital cysts is not understood, but they are probably the consequence of a defect in development.

The symptoms beyond that of an enlarged organ may be none at all, or they may be those of interstitial nephritis with its secondary cardio-vascular consequences. There may be a small albuminuria. Blood discs may be found, but no casts.

(3) *Dermoid cysts* are also occasionally met in the kidney, while a *general cystic condition* invading the liver and spleen as well as the kidney is described.

(4) Hydronephrosis.

Hydronephrosis is a monocystic degeneration of the kidney starting in obstruction of the ureter, succeeded by dilatation of the pelvis and gradual wasting of the kidney substance due to pressure of the accumulating fluid.

Etiology.—The obstruction causing this condition may also be congenital. As such it, too, may be large enough to impede labor. An oblique insertion of the ureter at such angle as to interfere with the easy discharge of the secretion may be the cause of its retention in the pelvis of the organ. Among recognized causes during life are, also, occlusion of the ureter by cicatricial adhesion, by lithiasis, tuberculosis of the ureter, by pressure on the ureter by tumors, by a retroflexed or prolapsed uterus, by bands of lymph in healed peritonitis and twists in the ureter of the movable kidney. Finally, carcinoma of the bladder and even hypertrophy of the prostate and stricture of the urethra may be causes.

The contents of the tumor may be purely aqueous, more frequently they are slightly turbid ; contain a few pus cells, more if they are the seat of inflammation ; also uric acid, urea, and albumin.

Symptoms.—Its symptoms are those general symptoms already described as common to benign renal tumors of sufficient size. An event which is almost pathognomonic is the occasionally sudden disappearance of the tumor simultaneously with the discharge of a large quantity of fluid from the bladder, followed by gradual refilling of the sac and return of the tumor. This intermittent discharge may be kept up for years. Such an event must be ascribed to a valvular obstruction in the ureter which at times yields to the pressure of the accumulated fluid ; or it may be due to the undoing of a twist in the ureter of a floating kidney.

Diagnosis.—As to differential diagnosis, ovarian tumor is the condition with which it is most frequently confounded. I have already (p. 683) called attention to the different relations of the intestine to the tumor in the two instances and other points of difference, all of which need not be repeated. The relative immobility of the renal tumor as contrasted with the mobility of the ovarian may, however, be mentioned ; also the lumbar origin of the former as contrasted with the pelvic of the latter, as determined by rectal and vaginal examination. Should the tumor disappear simultaneously with a copious discharge from the bladder the diagnosis is, of course, conclusive. Aspiration may also be brought to aid the diagnosis,

when advantage may be taken of the fact that the characters of the ovarian fluid and that of hydronephrosis are widely different. The dense, colloid fluid of the ovarian cyst, with its numerous cholesterin plates, fatty granular cells, small granular cells, and highly albuminous composition, contrast strongly with the aqueous consistence, the low specific gravity, and the general negative character of the hydronephrotic fluid. Too much stress must not, however, be laid upon the differences in these fluids, as they do not always persist. The history in the case of ovarian tumor will develop the events, menstrual and sexual derangement, absent in hydronephrosis. It is not likely that hydronephrosis will be confounded with ascites. The changes in the position of the fluid with that of the patient, characteristic of ascites, and its bilateral situation distinguish it at once from hydronephrosis.

From a circumscribed peritoneal exudate, hydronephrosis is distinguished by the different history, the greater tenderness of the former, and tympany of the subjacent intestine elicited by strong percussion. In renal abscess there is also fluctuation, but there is also fever and sometimes chills. The renal retention cyst is at times undistinguishable from hydronephrosis. Both may be congenital or due to congenital defects, but should there be intermittent emptying of the sac with refilling, hydronephrosis may be suspected. The diagnosis of hydronephrosis from hydatid cyst so far as is possible follows in the next paragraph.

(5) Echinococcus, or Hydatid Cyst.—Hydatid disease of the kidney is a rare affection, and when the enlargement caused by it is sufficient to produce physical signs they do not differ essentially from those of hydronephrosis and cystic kidney. Only in the event that the microscope recognizes hooklets or scolices or fragments of cyst wall in the urine or in the fluid obtained by tapping or discharge into other localities, such as the stomach, intestines, or bronchi, can a diagnosis be made with certainty. Such discharge into the pelvis of the kidney, if it produce obstruction, may also occasion nephritic colic, while such obstruction of the ureter may also cause acute hydronephrosis. The presence of hydatids elsewhere and of the hydatid fremitus is presumptive evidence. The chemical and physical characters of the fluid from hydatid cysts is given under hydatid disease of the liver (p. 368). Like hydronephrosis and cystic tumor, the hydatid kidney differs from ovarian tumor by its immobility, unless the disease should perchance invade a movable kidney.

Treatment of Renal Cysts.—The treatment of the whole list of affections included under cysts of the kidney is a matter for the surgeon, the chief office of the physician in these cases being one of diagnosis and relief of pain.

ANOMALIES OF FORM AND POSITION OF THE KIDNEY.

Normal Situation of the Kidney.—The normal situation of the kidney is on the quadratus lumborum and psoas muscles, the inferior end of the left kidney extending a variable distance below the edge of the 12th rib while the right extends about ¾ (20 mm.) of an inch lower down, the whole right organ being lowered by the position of the liver. The outer edge of the kidney is often in a line drawn vertically downward from the end of the 12th rib. Both kidneys descend about ½ inch (12.5 mm.) during deep inspiration. The kidney, if in its normal situation, is accessible to pressure just below the last rib at the outer edge of the erector spinæ muscle. Sometimes one or the other, more frequently the left, lies on the lumbar vertebræ, on the sacrum, or in the inguinal canal.

Congenital Absence of the Kidney.—The total *absence* of *both* kidneys is possible in connection with extreme abnormalities and defect of development, but is incompatible with life.

Congenital absence of *one* kidney is not very rare, the absent one being usually the left. Such absence may be suspected when over the normal situation of the organ a tympanitic note only can be elicited by percussion. In such event the remaining kidney supplements the work of the absent one and serious consequences only follow in the event of disease or lesion of the remaining organ. The ureter and pelvis of the absent kidney are absent also, but sometimes the remaining organ has two pelves and two ureters. Occasionally the rudiment of a ureter is present. Congenital atrophy of one kidney is even more common, but is discoverable only at autopsy.

Lobulated Kidney.—The lobulated kidney is the most frequent anomaly of form. It consists essentially in the persistence of the lobulation natural to the organ in the fœtal state. This is acquired by the end of the eighth week of fœtal life, after which it gradually disappears in normal development but is still maintained with more or less distinctness throughout the first year after birth. The lobulation is variously distinct. Usually partial and superficial, the fissures are sometimes so deep as to divide the organ into separate renculi, of which there may be seven to 20. This lobulation, a rare event in man, is beautifully seen in the kidneys of the lower animals, especially the sheep and ox.

Horse-shoe Kidney.—The most striking of the anomalies of form is the horse-shoe kidney, in which usually the lower ends of the two organs are united either by true renal tissue or a band of fibrous tissue. More rarely it is the middle segments which are united and more rarely still the upper ends. In either event this coalescence is usually associated with displacement of the organ, which is then lower down than in the normal condition, usually just above the promontory of the sacrum, more rarely in the pelvis, and at times on one side or other of the spinal column. In the fused kidney there are usually two pelves with from

two to four ureters. More rarely there is but one pelvis. The ureters pass over the front of the kidney. The renal arteries spring from the aorta at points corresponding to the situation in which the organ is found. Thus when above the sacrum the arteries spring from the back of the aorta near its bifurcation or from one of the common iliacs, while the veins enter the corresponding parts of the vena cava or iliac veins.

The horse-shoe kidney is generally recognized first at autopsy, but rarely it may be recognized in its abnormal position above the sacrum, especially in thin persons.

The Movable or Floating Kidney.

Synonyms.—*Ren mobilis; Floating Kidney; Palpable Kidney; Nephroptosis.*

Described.—The normal kidney is commonly quite firmly retained in position by its capsule of fat and a covering of peritoneum. The movable or floating organ exhibits a very different degree of mobility in different instances. It may be so slight that it can be recognized only by the expert manipulator, or so great that the organ may be easily grasped by the hand through the abdominal walls. In the latter condition there is a mesonephron or peritoneal fold loosely attaching the kidney to the spine.

Etiology.—The movable kidney is more common in thin persons than in the obese, in women than in men. Indeed, it has been said that one woman out of every four has a movable kidney. It is six times as frequent in the working classes. The right is far more frequently movable than the left. Repeated pregnancies are assigned causes, as is also mechanical violence, as a fall or tight-lacing. It is most likely, however, that the majority of floating kidneys are congenitally loose, and that this looseness may be increased by the conditions named.

Symptoms.—The floating kidney often occasions no symptoms. At other times it is responsible for a remarkable train of *nervous symptoms*, mainly reflex in character. These include *obstinate indigestion* of every grade, *flatulence*, *palpitation of the heart*, *cardialgia*, *neuralgic pain* almost anywhere in the body, but especially in the abdomen and cardiac region; also *irritable bladder* and *dysmenorrhœa*. It is an interesting fact that where the degree of displacement and the mobility are most marked, the reflex symptoms are least so. This is not without a parallel in other diseases, and in illustration may be cited the well-known fact that prolapsus uteri of moderate degree often causes decided reflex symptoms, while a complete procidentia produces often trifling local annoyance. The direct result of the displacement, so far as appreciable, is a sense of *dragging* or weight, which especially manifests itself while standing, walking, riding, or dancing, to which may be added a variable amount of *pain*. More serious symptoms sometimes manifest themselves as the result of *torsion* of the ureter, occasioned by complete rotation of the kidney, in which the renal vessels and nerves are also involved. These are agonizing pain, associated with symptoms of collapse, such as nausea, an anxious expression, scanty urination.

These are caused in part by obstruction to the ureter and the backing of the urine on the kidney. They are, in fact, the symptoms of nephritic colic. Acute hydronephrosis may also be the result of such strangulation, which may be caused, too, by inflammatory bands. This condition ends itself sometimes as suddenly as it appears. Both hemorrhage and albuminuria are reported as results of the same condition. Both are certainly rare. There may be other effects of displacement due to the location of the organ at times, of which irritation due to pressure upon the bladder may be mentioned as one. It is often very uncomfortable for the patient to lie on the side opposite that on which the displaced organ belongs.

Diagnosis.—This is variously difficult. The kidney exhibits some mobility in health, descending also always ½ inch (1.3 cm.) with each deep inspiration. Movable kidneys are sometimes so loose and movable that they may be felt through the abdominal walls with ease. Between this ready recognition and that which requires the highest manipulative skill of the examiner there is every degree. At the present day movable kidney is regarded as a much more frequent condition than was formerly thought. So frequently has the set of reflex nervous symptoms described been found associated with movable kidney that their presence should always suggest an examination for the presence of such an organ. The examination may be made with the patient in the standing posture, in one of two or three positions, or lying on her back. In the first she bends slightly forward, her hands placed on a table, and the clothing thoroughly loosened. The right hand of the examiner is then placed in front immediately next the skin, below the hypochondrium, while the left is placed over the lumbar region. The patient is directed to respire deeply and regularly, and to relax herself during expiration. The region between the two hands is carefully palpated, when, if there is any marked degree of displacement, or rather of lowered position, the organ can be felt as a firm, smooth, oval body, somewhat sensitive to pressure, which also produces a sickening pain, which is quite characteristic. More rarely the pulsation of the renal artery can be felt. The right kidney naturally moves with breathing more than the left, being pushed down by the liver. Sometimes the manipulation will be more successful in the knee-elbow position. When in this position, the movable kidney having fallen forward, a resonant note may be obtained by percussing over the normal situation of the organ. Or the patient may be placed on her back with the side to be examined toward the edge of the bed, on which the physician may sit. The hands are applied as in the standing position and manipulation practised as described.

The displaced organ is hardly likely to be confounded with anything else. The spleen, which corresponds nearly in size, is also sometimes movable. Its shape is, however, different. Its anterior border is sharp and often notched. Sometimes both the left kidney and the spleen are floating. A movable pyloric tumor has been taken for a movable kidney. The passage of a stomach-tube in case of doubt would settle such doubt.

Treatment.—As may be inferred, many cases of movable kidney require no treatment. In a few cases the symptoms are removed by

improving the general health of the patient, in others the patient is comfortable while lying on the back, and such comfort may continue for a time after rising. Where decided symptoms attributable to the kidney are present, surgical treatment for fixing the kidney—nephrorrhaphy—is alone of permanent value, and this sometimes fails. I have known an operation for removal of the kidney—nephrectomy—to be necessary after nephrorrhaphy had been attempted twice unsuccessfully. The use of pads and supports have never proved successful in my cases.

IDIOPATHIC HÆMATURIA.*

Definition.—A hæmaturia the origin of which is unknown. In addition to the various causes of bloody urine already referred to in treating diseases of the urinary organs, and in addition to malarial hæmaturia, there remains a form of renal hæmaturia of not very infrequent occurrence for which none of the causes named will account. To this the term idiopathic hæmaturia is appropriate.

Symptoms.—The characters of the urine in this form of hæmaturia are in no way different from those of renal hæmaturia from other causes. The blood is intimately admixed with the urine and is not, as a rule, found in coagula, as is so often the case when the blood comes from the bladder or pelvis of the kidney or in malignant disease of the kidney. There is the usual smoke-hue characteristic of acid urine containing a small amount of blood, becoming brighter red as the urine becomes alkaline, and darker red as the quantity of blood is increased. The microscope reveals numerous blood discs, recognizable by their usual characters, and often blood casts and casts filled with the debris of red discs, or red discs so closely packed as to make it impossible to distinguish their outline. The urine is, of course, albuminous.

Next to the change in the urine the most striking feature is the absence of other symptoms. The subject is not sick, is not weak, complains of nothing. Occasionally a dull ache in the back is felt or supposed to be felt, perhaps because the patient thinks that since there is bloody urine there ought to be pain in the back. The same may be said of weakness, but these symptoms are not usually complained of, though they may be present. Sir William Gull spoke of such hæmaturia as a "renal epistaxis." With the lapse of time, however, and the continuance of the symptom, positive weakness gradually supervenes.

Treatment.—Rest is an important and essential condition in the successful management of idiopathic hæmaturia. The usual astringents, mineral and vegetable, known to be efficient in the treatment of hemorrhagic conditions elsewhere are often without effect here, though these substances, including gallic acid, ergot, the persulphate of iron, and acetate of lead, alum, catechu, and kino, may be tried. The persulphate of iron has proved of undoubted value in my hands in doses of ¼ to ½ grain

* I realize that the subjects, Hæmaturia, Hæmoglobinuria, and Chyluria, next to be considered are not strictly renal affections, but it is difficult to classify them otherwise.

0.015 to 0.032 gm.) every four hours in a pill. The astringent mineral waters, especially the alum waters, as those of the Rockbridge Alum Springs, in Virginia, and the Jackson Spring, North Carolina, have apparently proved curative, but I have also failed with them. Recently a case which I saw, in consultation with T. Parry Tyson, of Jenkintown, resisted all measures until Dr. Tyson tried the fluid extract of hamamelis virginica in doses of 20 minims (1.32 gm.) every three hours, and in thirty-six hours the hemorrhage ceased and has not recurred. As hamamelis contains much tannin and gallic acid it may owe its efficiency to this constituent. One of my cases, after resisting every other form of treatment, finally yielded to electric baths.

HÆMOGLOBINURIA.

Definition.—In this interesting condition the coloring matter only of the blood is found in the urine, very rarely a few blood discs or their fragments. In their absence other criteria of the presence of blood coloring matter must be sought. To do this one may make Teichmann's hæmin crystals, as directed in the foot-note on page 71.

Or if the spectroscope be available, the filtered and diluted urine produces the absorption bands of oxyhæmoglobin between Fraunhofer's line D and E, or more frequently the three bands of methæmoglobin, of which that in the red near C is distinctive, should be sought. Sometimes both are present. The urine thus stained with hæmoglobin is dark brownish-red, and even black in color. It is also albuminous, and in lieu of the blood discs are sometimes found yellowish-brown, irregular, and granular flakes and sometimes cylindrical masses of hæmoglobin.

Hæmoglobinuria is always associated with hæmoglobinæmia, which is, however, less easy of demonstration. The hæmoglobin is set free from the corpuscles and imparts a red or lake hue to the blood plasma. The discs themselves are paler, and yellowish-brown particles of hæmoglobin may be demonstrated between the corpuscles. The number of corpuscles themselves may be reduced, falling to 4,000,000 and less.

Hæmoglobinæmia and hæmoglobinuria may easily be separated into two divisions, toxic and simple paroxysmal.

Toxic Hæmoglobinuria.—This is produced by toxic substances, which dissolve out the hæmoglobin from the corpuscles. Such are sulphuretted hydrogen, arseniuretted hydrogen, carbon monoxide, carbolic acid, pyrogallic acid, naphthol, nitro-benzole, potassium chlorate in large doses, and the poison of certain mushrooms; also sometimes the poison of the infectious diseases, including scarlet fever, diphtheria, pyæmia, yellow fever, typhoid fever, malaria, and even syphilis. The latter has sometimes seemed to act as a predisposing cause, subsequently to which so trifling a thing as exposure to cold has caused it. I have seen it associated with pregnancy as a probable cause. Hæmoglobinæmia and hæmoglobinuria sometimes succeed on extensive burns when the poison

is probably the retained excretions of the skin. High temperature alone is said to have caused it. In malarial poisoning the hæmoglobinæmia may be the direct result of the action of the malarial plasmodium. The blood of one animal transfused into the vessels of another acts similarly. Finally, the hæmoglobinuria of the new-born, occurring also epidemically, must be added to this group.

Prognosis.—This depends upon the dose of the toxin causing it and the other symptoms produced. Recovery is usual, but some cases are rapidly fatal.

Treatment.—This is that of the disease occasioning it. The same astringent measures may be tried as in hæmaturia and restorative medicines to rebuild the blood. Of these iron is the most important.

Paroxysmal Hæmoglobinuria.

In this intermittent attacks occur. They come on suddenly, preceded by chills and fever, headache, and pain in the limbs, the temperature often reaching 104° F. (40° C.). The bloody urine follows in an hour or less and may last four or five hours, or there may be two or three paroxysms in a day. At other times there is no fever or the temperature is even subnormal. Jaundice is associated with some cases, especially toward the end. At times, instead of the expected hæmoglobinuria there is only albuminuria. Ralfe explains this by supposing that the toxic agent has destroyed only a small number of corpuscles, the coloring matter from which is used up in the spleen and liver, while the globulin goes off in the urine. V. Leube especially calls attention to a swelling and tenderness of the liver and spleen, and says he has met with these symptoms in lieu of the expected hæmoglobinuria, in lieu even of albuminuria.

The occasional association of hæmoglobinuria with Raynaud's disease is very interesting. The probability is that in most cases where the two conditions are associated the preliminary hæmoglobinæmia is due to a separation of the hæmoglobin from the red discs in the peripheral asphyxiated part of the nose, ears, and fingers.

As to other causes of paroxysmal form, malaria is undoubtedly one, though perhaps not so often as once supposed. Another cause is excessive muscular exertion especially when associated with cold, while cold itself is perhaps the most frequent of all causes. Mental emotion is sometimes a cause. It must be admitted that for the cases not explainable by toxic agency no satisfactory solution has been presented.*

Prognosis.—The prognosis of the paroxysmal form is commonly favorable, though it may continue to recur for a long time.

Treatment.—This depends upon the cause. If it be malarial the condition is easily curable by quinine. To seek the causes in many cases is to seek the unattainable, and the cases must be treated on general principles. Rest and warmth are essentials. After this the same astrin-

* It is well known that horses are subject to hæmoglobinuria, and that it occurs in them after exposure to cold, especially after having been stabled for several days.

gent remedies as those recommended under hæmaturia may be tried. As cold seasons and cold weather favor it, a residence in a warm climate should be tried where the condition persists. Nitrate of amyl is said to cut short and to prevent an attack.

CHYLURIA.

Definition and Description.—A state of the urine in which the secretion is admixed with fat in a minute state of division, whereby the urine acquires a milky or chylous appearance.

The proportion of fat varies greatly, being at times only enough to impart a mere opalescence, while at others the urine is scarcely distinguishable from milk, while even the characteristic odor and taste of urine are wanting. The fat also often rises to the surface on standing like cream. By the microscope, in addition to this molecular fat and a few oil drops, numerous blood discs are also found. These are sometimes so numerous as to impart a pinkish tinge to the fluid, and at times a spontaneous coagulation takes place with the formation of a slight reddish clot showing the presence also of fibrin.

Etiology.—To produce chyluria there must in some way be brought about a leakage from chyle vessels into the urinary passages somewhere between the kidney and the neck of the bladder. Yet no such communication has ever been found so far as I know, though W. H. Mastin noted the patulous mouths of several opening into the serous sac of a testicle which he laid open for the cure of a chylous hydrocele or lymph scrotum, and having ligated them no recurrence of the hydrocele occurred. Supposing such a communication to exist, how is it brought about? In most cases probably by the blocking of the lymph channels by the prematurely discharged ova or the embryos of the filaria Bancrofti, to be described when treating of animal parasites. Undoubtedly there occurs, on the other hand, what may be termed under the circumstances an idiopathic chyluria, the most searching examination of the blood during life and careful dissection after death of lymph glands and vessels in certain cases failing to discover either ova or embryos. On the other hand, by no means every case of filariasis is attended by chyluria.

Symptoms.—Few symptoms other than those of the chylous urine are present. It is usually intermittent but may be persistent. There is sometimes a loss of strength from the draining off of a nutrient fluid, at other times some pain in the back, and at others again painful urination due to obstruction of the urethra by coagula of fibrin, but in most cases the patient feels well and would not know there was anything the matter with him but for the state of the urine.

Treatment.—No means have been discovered to destroy the filariæ in parasitic chyluria nor to check the leakage in the idiopathic form, which often persists for years and then subsides spontaneously.

See also article on filariasis in section on "Parasites."

THE RELATION OF HEART DISEASE TO KIDNEY DISEASE.

Allusion has more than once been made in the foregoing pages to the relation of kidney disease to heart disease and the association of lesions in the two organs. Of such importance is a proper understanding of this relation, that a few pages devoted to its consideration may be helpful to the student. The association admits of classification.

I. *Renal Disease Associated with Hypertrophy of the Left Ventricle without Valvular Disease.*—Modern studies have made it pretty certain that this form of combined heart and kidney disease may originate in two ways: (*a*) the heart affection may be secondary to the kidney disease as its direct consequence; or (*b*) both conditions may result from one and the same cause, arterial sclerosis.

(*a*) *The Renal Condition Precedes.*—The condition which precedes is commonly the contracted kidney of interstitial nephritis, and its result is hypertrophy of the left ventricle.

How is the cardiac hypertrophy to be accounted for? I will first review the theories which have been from time to time given, for theories alone they must be acknowledged to be. The oldest, which may be termed the "chemical theory," was advanced in its cruder form by Bright himself, whose acute observation had not failed to notice the association of cardiac hypertrophy without valvular disease with the disease so deservedly coupled with his name since 1827. Bright suggested two alternative explanations: "Either that the altered quality of the blood affords irregular and unwonted stimulus to the organ immediately, or that it so affects the minute and capillary circulation as to render greater action of the heart necessary to force the blood through the distant subdivisions of the vascular system." At the present day Sir George Johnson, in England, and Senator, in Germany, still hold this view. At one time Johnson held that the hypertrophied state of the muscular coat was the result of an effort of the vessels to aid the onward movement of the blood. More recently he ascribes the thickening of the muscular coat, which exists not only in the kidneys, but in the systemic arterioles generally, to a constant exercise of the normal stop-cock action whose object is to resist the passage of this abnormal blood; while the hypertrophy of the left ventricle is ascribed to an effort to overcome this resistance. Dr. Johnson still maintains that there is a true hypertrophy of the muscular coat in all the tissues, associated with a thickening of the intima only in the kidneys, but of the adventitia also in the hypertrophied arterioles in other tissues.* Recent histological studies made for Dr. Johnson under the direction of Dr. Halliburton confirm this view. Unfortunately, other recent studies by modern methods, notably those by W. T. Councilman and Arthur V. Meigs, to be again alluded to, do not find the changes in the middle coat early described by Johnson and now again by Halliburton, while experiments which have for their ob-

* "The Pathology of the Contracted Kidney," by Sir George Johnson. London, 1896.

ject charging the blood with urea and allied excrementitious substances have also failed to excite hypertrophy. It should be added, however, that it is not possible by experiment to produce precisely the conditions furnished by diseased kidneys, especially in the matter of duration. It is very generally conceded that uræmia is due to retained excrementitious substances which are in health eliminated by the kidney, but the experimental introduction of these same materials has as yet failed to produce uræmia. Here, too, it is to be remembered that sound kidneys are encountered, which by copious diuresis promptly eliminate the substances introduced. It is to be regretted that a histological question seemingly so simple cannot be settled by the refinements of modern histology.

The so-called "mechanical theory" of cardiac hypertrophy was advanced by Traube, whose researches in 1856 gave a decided impulse to clinical study of the subject. According to Traube, the increased arterial resistance was caused by two erroneously supposed states—the first an over-fulness of the vessels due to the diminished renal secretion; second, a resistance to the admission of arterial blood into the kidney, due to the renal contraction itself. The first hypothesis was erroneous for contracted kidney, where the urinary secretion is really increased, while the second is opposed by the fact that even ligature of the renal arteries fails to increase arterial pressure, because of the ample vascular space elsewhere for the blood thus diverted. Nor did Cohnheim's further elaboration of the mechanical theory, which located the increased resistance more precisely behind the wasted glomerule, give any more permanent life to it.

I incline to the belief that the difficulties are best met by supposing the primary changes in the heart to be *compensatory* in their nature, set up with a view to making up for the gradual loss of renal substance. Such an action is paralleled everywhere in the physiological economy. Nowhere do we meet with loss of function which is not at once met by an attempt of nature to supplement it. The dependence of the urinary secretion upon cardiac pressure is well understood, and an increase of cardiac power is the most reliable means available for stimulating the action of the kidneys, when desired, in therapeutics. The diuresis which is so constant a symptom of the contracted kidney is acknowledged to be the direct result of a supplemental contraction of the left ventricle, which it is reasonable to suppose is induced for the purpose named, and results in hypertrophy.

This view receives confirmation in the subsequent course of the disease. So long as the free secretion which is the result of the compensatory action of the heart is kept up, so long the patient remains tolerably comfortable, and perhaps even for a time unconscious of the presence of disease. But an organ thus overgrown is sure sooner or later to suffer in its nutrition. Especially is this the case if its arteries be the seat of an endarteritis, interfering with the free movement of the blood and producing also fibro-myocarditis. And what are the further consequences? The strong propulsive power of the heart declines, the pulse falls away in tension and power and becomes more frequent

and sometimes irregular. The urine secreted diminishes in quantity and assumes a darker hue. Fortunate is the patient if the specific gravity of the urine rises inversely with its reduced quantity, as it indicates that the normal quantity of solids is kept up. Too frequently, however, this is not the case, and excrementitious substances accumulate in the blood, laying the foundation for uræmia. Headache, nausea, a foul and even urinous breath, may be superadded, and uræmia set in, preceded by drowsiness, or it may be ushered in suddenly with convulsions. Or another set of symptoms may supervene. The patient becomes short of breath, first on slight exertion, and later this very distressing symptom occurs without such exciting cause. This sort of asthma, spoken of as uræmic asthma, has been discussed on page 637. For a time this symptom may be averted by whipping up the heart by cardiac stimulants, and the right ventricle even comes to the rescue for a time and hypertrophies in its effort to overcome the now disturbed compensation. Subsequently this as well as the left ventricle may become dilated, and œdema of the lungs set in, with annoying cough and serous frothy expectoration, sometimes blood-tinged. Nor does general dropsy continue absent, but ensues sooner or later with the growing heart-failure. Our resources are now almost at an end, but are not exhausted, as even these symptoms sometimes subside.

It should be stated that not every case of interstitial nephritis is attended with hypertrophy of the left ventricle. In addition to the cases of contracted kidney from senile endarteritis already referred to, cardiac hypertrophy is apt to be absent in the interstitial nephritis of the weak and cachectic.

(*b*) *Both the Hypertrophy of the Left Ventricle and the Contracted Kidney are the Result of One and the Same Cause, Arterial Sclerosis or Arterio-capillary Fibrosis.*—It should be stated first that a certain school explains all cases of contracted kidney in this way. The changes described in the arteries differ chiefly from those described by Dr. Johnson in that they are held to be degenerative so far as the muscular coat is concerned.

The late Sir William Gull and Thomas Sutton, in a paper which has become historic, announced in 1872 * that the changes in the muscular coat were chiefly of an atrophic character, and, although the methods of these observers have been much criticised, the most recent studies on this subject by W. T. Councilman and Arthur V. Meigs go to confirm their conclusions both as to the seat and the nature of the changes. Councilman † finds atrophic changes in the muscular coat, including greater or less destruction of the muscular fibre-cells and the formation of a homogeneous hyaline tissue invading both coats, but especially the intima, where it produces decided thickening, and encroachment to a varied extent upon the lumen, sometimes amounting to occlusion. The capillary walls are likewise thickened, and sometimes, especially in the

* "Arterio-Capillary Fibrosis," Med. Chir. Trans. London, 1872.

† "On the Relations between Arterial Disease and Tissue Change." Trans. Asso. Amer. Phys., Vol. VI, 1891.

glomerule of the kidney, obliterated. Meigs's * studies also find these changes for the most part confined to the intima, which is decidedly thickened. To a less extent the intima of the veins is similarly involved. The picture of the changes thus briefly described may be obtained from a small artery taken almost indifferently from any tissue or organ of the body—for example, from the muscular substance of the heart, the kidney, or the liver. It is this diffuse form of arteritis rather than the nodular, where the changes are limited to small areas in the aorta and large arteries—atheromatous patches, sometimes calcareous and sometimes fatty, or the so-called senile endarteritis—which is the link between the hypertrophy of the left ventricle and the contracted kidney.

Of its consequent changes, the hypertrophy of the ventricle is most easily explained. The resistance in the blood-vessels stimulates the ventricle to increased effort, and there result increased arterial tension and hypertrophy. The degree of cardiac hypertrophy is sometimes enormous, the organ weighing as much as 850 grams, or 28 ounces, and the average in 27 cases studied by Councilman being over 400 grams, or 13 ounces, as contrasted with the normal, 10 to 12.

The alterations in the kidney, which vary greatly in degree, being sometimes scarcely noticeable and sometimes extreme, are the direct result of an interference with its nutrition. The blood-supply to the renal elements being cut off, these gradually waste and ultimately disappear. The cells and tubules thus destroyed are gradually but irresistibly replaced by fibrous connective tissue, in obedience to the pathological law elaborated by Weigert but which was announced, at least so far as the kidney is concerned, fifty years ago by Dr. Johnson,† that parts destroyed are partially replaced by cicatricial connective tissue. This contracts and reduces the size of the kidney, and perhaps also, in this contraction, further destroys the proper kidney structure and thus augments the atrophy.

It has been said that in this form of combined kidney and heart disease there is no cardiac murmur. Nor is there, as a rule. It is not impossible, however, for the endarteritis of which we are speaking to creep along the walls of the aorta until it reaches the aortic valves and so structurally changes them as to make them rough or incompetent and give rise to murmurs.

The causes of this form of Bright's disease are, therefore, the causes of the endarteritis, which have been discussed on p. 567. To a less degree probably the specific causes of all the infectious diseases must be included in this category—possibly even malaria. The subjects are usually middle-aged men between the ages of forty and fifty-five, but they may be younger. Councilman has found these changes more common in the negro than in the white race.

Treatment.—The treatment of this form of combined kidney and heart disease is not different from that of interstitial nephritis. Even greater rigidity is necessary in eliminating nitrogenous food, and all food

* New York Med. Record, July 7, 1888.

† Medico-Chirurg. Trans., 1847.

should be reduced to a minimum. A diet of milk diluted with water is the safest of all. Bread and butter may, however, be allowed, and even succulent vegetables easy of digestion, such as rice, potatoes, peas, and string beans, while simple fruit-juices, as those of oranges and lemons, are allowable. Mental excitement and immoderate muscular exertion must be avoided, and the heart should not be over-worked in any way.

The medical treatment especially directed to the arterio-sclerosis may be divided into that intended for the cure of the endarteritis and that directed to the relief of symptoms. The only drug from which results may be expected for the former purpose is the iodide of potassium, which should be given a fair trial in doses as large as can be borne without deranging the stomach. For the relief of the symptoms, more especially due to the sclerosis, viz., headache, throbbing, and vertigo, the nitrites are often useful. Nitroglycerine should be given in doses of $\frac{1}{100}$ grain (0.00065 gm.) every four hours or oftener, rapidly increased to $\frac{1}{50}$ grain (0.0013 gm.) if the smaller dose is without effect. The aim should be to produce the physiological effect, which is a sense of fulness or a flushing. The sodium nitrite may be substituted in three to five-grain (0.19 to 0.32 gm.) doses. It has the advantage of being more permanent in its effect, although it is slower in its action.

II. *Valvular Heart Disease Associated with Renal Disease in which the Heart Disease is Primary.*—The heart disease is commonly disease of the mitral valve. The kidney involvement does not occur in connection with aortic valvular disease until mitral insufficiency is superadded. It is well known that in mitral regurgitation as soon as compensation ceases the blood accumulates first in the lungs, then in the right side of the heart, and finally in the venous system, engorging especially the liver, the stomach, and the kidneys. The effects upon the first two were discussed in treating of heart disease. They generally manifest themselves sooner than the renal symptoms. The result in the case of the kidney is the kidney of passive congestion or cyanotic induration, already described on p. 629.

Much more decided are the clinical phenomena resulting from such congestion. It is a well-recognized condition of copious secretion of urine that the blood should move freely through the kidney. A stasis is followed immediately by diminished filtration of water, the twenty-four-hours' quantity being reduced to from 30 to 20 ounces, and even to less. The solids at first at least remain the same, the urine is dark-hued, the specific gravity is high, the reaction is markedly acid, and a copious sediment of urates and uric acid makes its appearance as soon as the urine cools off. There is almost always a small amount of albumin found, but as the congestion increases albumin becomes copious. Casts are sparsely, if at all, present, and are of the hyaline and faintly granular variety. Both red and white blood corpuscles are also sometimes detected, as might be expected. This condition of the kidney and the symptoms are the direct results of the cardiac valvular disease. Yet I often see these cases diagnosticated as Bright's disease as though it were the primary and principal affection. They may also be produced by any

cause producing venous stasis, as pulmonary emphysema, chronic pleurisy, and thrombosis of large veins. Their effect is further to augment the symptoms of the cardiac disease. The circulation, already everywhere obstructed, is further impeded, there is dyspnœa, dropsy increases, the appetite fails, there are nausea and constipation. Sleep, already disturbed by dreams, becomes more so, and a more distressing picture than is presented by such a case is rarely met.

Treatment.—Yet these symptoms are often easily amenable to treatment so long as the heart-muscle remains capable of being influenced by digitalis. I have seen many a patient, apparently *in extremis*, gasping in orthopnœa and with legs heavy and almost bursting with dropsical effusion, completely relieved by a few large doses of this drug. But they must be large doses, not less than eight to ten minims (0.5 c. c. to 0.6 c. c.) or 15 to 20 drops every three hours until an effect is produced. If digitalis fails, the tincture of strophanthus may be given in the same doses, or caffeine citrate in three-grain (0.2 gm.) doses, each every four hours, or sparteine sulphate in doses of ¼ to ½ grain (0.016 to 0.03 gm.). Purgation should not be omitted, but should rather be pushed to the production of watery catharsis. The ingestion of fluids should be restricted, and the Hay's treatment of dry diet with purgatives is sometimes useful. But I have found a restricted milk diet, limited to two ounces every two hours, associated with the drugs above named, more efficient.*

Such a kidney is, of course, liable to become the seat of an acute or a chronic nephritis.

III. *Renal Infarct the Result of Heart Disease.*—The next form of kidney-involvement secondary to disease of the vascular apparatus, commonly heart disease, is more frequently seen on the post-mortem table than recognized in the living subject. It is *embolic infarction*, produced by the lodgment in some branch of the renal artery of an embolus derived from the heart or a blood-vessel. Its most frequent source is a fragment of vegetation or clot from a diseased heart-valve. An embolus may also arise from a thrombus in the heart or an artery, but it may also arise from a thrombus in a vein. If from the latter, it must be carried first to the right heart, and thence through the lungs into the left heart, and thence by the aorta to the kidney, and must, of course, be small.

The effect of the lodgment of an embolus in the kidney is a wedge-shaped hemorrhagic infarct, which at first is dark red in color, standing out above the surface, but which in time whitens, contracts, and is ultimately absorbed, leaving a mere cicatricial mark.

Most frequently a renal infarct occurs without noticeable symptoms. Its occurrence, if looked for by reason of the presence of valvular heart disease, may be suspected if there is the sudden appearance of blood in the urine. A sudden pain in the region of the kidney occurring at the

* See some cases reported by the writer in a paper "On the Management of Obstinate Dropsies," Med. News, June 21, 1890.

same time with hæmaturia would go to confirm the diagnosis. No treatment is indicated, even if the event is recognized.

IV. *Accidental Coincidence of Renal Disease and Heart Disease.*—Finally, kidney disease and disease of the vascular apparatus, and especially cardiac disease, may coincide accidentally, each the result of its own cause, and reacting the one upon the other in various degrees and variously aggravating the symptoms of each, so that it often becomes a very nice question to settle which is the preponderating disease. Fortunately, this difficulty does not always extend to therapeutics, the same remedies which are useful to one affection being commonly indicated for the other. A careful study of each case should, however, be made on its own merits, and due weight assigned to each factor of the disease.

DISEASES OF THE BLADDER.

CYSTITIS.

Synonyms.—*Catarrh of the Bladder; Vesical Catarrh.*

Definition.—Inflammation of the bladder.

Etiology.—The more usual causes of inflammation of the bladder are foreign bodies such as stone, obstruction to the outflow of urine by enlarged prostate or stricture of the urethra, and pathogenic organisms variously introduced, but especially by dirty catheters. The number of cases caused by catheterization has diminished because of the greater precaution taken of late in the care of instruments. Of acknowledged bacterial origin is also gonorrhœal cystitis, which, succeeding an attack of gonorrhœal urethritis, is due to the invasion of the bladder by simple extension by continuity. Among organisms capable of exciting inflammation are the streptococcus pyogenes, staphylococcus pyogenes aureus, diplococcus, bacterium coli commune, tubercle bacilli, and perhaps others. Virulent causes are the bacteria contained in decomposing urine retained in the bladder.

Whether the obstructive causes above referred to are of themselves sufficient, or whether they may simply supply the conditions favorable to the operation of bacteria may be considered unsettled at the present day. Cold was formerly recognized as a cause of cystitis, especially in women and children, but it may be subject to the same conditions as the causes just named. On the other hand, substances introduced in the blood, as cantharides and capsicum, are capable of producing cystitis. Even the ingestion of certain articles of food has been followed by cystitis. Traumatic causes may be classed among predisposing causes rather than exciting, furnishing the conditions favorable to the operation of bacteria.

Morbid Anatomy.—The bladder of cystitis is a varying picture. There may be degrees so slight as to produce scarcely appreciable change

in the appearance. At other times the mucous membrane is hyperæmic and bathed with a mucoid or muco-pyoid secretion of dirty grey color. In many cases only the neck of the bladder and the part of the ureter passing through the prostate is involved. Again, the bladder is "ribbed," a result of straining. During this act the mucous membrane between the muscular trabeculæ yields, producing depressions bounded by the more unyielding muscular bands. On the other hand, in chronic cases permanent thickening of the bladder walls may result. Finally, in the severest forms of inflammation due to pathogenic organisms, such as those associated with putrid urines, the mucous membrane may be covered with patches of false membrane or the wall of the bladder may be infiltrated and undermined with pus, constituting the so-called phlegmonous or diphtheritic cystitis, from which there may result urethral and perineal infiltration. A further extension of the cystitis into the pelvic connective tissue about the bladder is known as paracystitis, which belongs to the province of the surgeon.

Symptoms.—While a division of cystitis into acute and chronic is justified by the suddenness and severity of symptoms in certain cases, as contrasted with their slow development in others, yet the conditions so constantly verge into one another that a separate consideration of the two forms is not necessary. The first symptom of cystitis is usually *frequent desire to pass water.* Such frequency varies greatly in intensity. It may be every few minutes or almost incessant, several times an hour or once in a couple of hours. After the primary frequency of disturbance it usually diminishes somewhat, so as to be once in a couple of hours. Such frequency is often attended by painful straining. In severe cases there is always *tenderness* over the region of the bladder above the pubes and in some cases there is constant *pain.* In these cases tenderness can also be elicited by pressure from the vagina and rectum, while catheterization is especially painful. In calculous cystitis pain is excited or aggravated by motion, especially such as is communicated to one riding in a wagon over a rough road.

As commonly met with, there is not often fever with cystitis, but the severe forms are attended with *moderate fever* and sometimes the diphtheritic variety with *high fever.* Even where there is fever the temperature does not exceed 100° to 102° F. (37.8° to 38.9° C.), though it may be higher. In certain acute diphtheritic cases of great virulence there are *chills*, *sweats*, and *high fever.* In advanced stages there may be *sepsis* due to absorption of retained putrid matters from the bladder.

The *urine* presents striking changes, from which alone the diagnosis may be made. *First*, it contains pus in varying quantities, but it is especially characteristic of the pus of cystitis that it is associated with mucus, imparting a glairy, stringy character to the urine, which increases the difficulty of its discharge from the bladder. The reaction of the urine when passed is commonly either alkaline or faintly acid, and if acid it promptly becomes alkaline. This is due to the formation of ammonium carbonate out of the normal urea, the result of the operation of bacteria. The greater alkalinity thus resulting reacts upon the pus and converts it into a glairy matter similar to mucus, thus further increasing the difficulty

in micturition. The pus under the circumstances is loaded with amorphous phosphates of lime and glistening crystals of ammonio-magnesium phosphate. It is so viscid that it will not rise in the pipette and must be cut with scissors to be manipulated for microscopic study. Blood is an almost constant constituent of the urine in calculous cystitis, and in the grave diphtheritic forms shreds of gangrenous bladder tissue may be discharged.

Diagnosis.—Ordinarily the diagnosis of cystitis is easy. Yet there sometimes occur mild forms which it is difficult to differentiate from mild degrees of interstitial nephritis, while it not very rarely happens that the two conditions are associated. In contracted kidney there are sometimes a good many leucocytes also. The presence of hyaline casts, even when scanty, points to nephritis, while hypertrophy of the left ventricle and increased arterial tension settle the question. Still more emphatic is the diagnosis if there is retinitis albuminurica.

The question as to whether there is pyelitis, separate or associated with cystitis, is still more difficult. I have said elsewhere that I know no distinctive cellular elements which settle this question, though some assert there are. Even spasm of the bladder, commonly regarded as peculiar to cystitis, may be present in pyelitis. Rather must we rely upon tenderness in the neighborhood of the kidney. Marked intermission in the purulent discharge, especially if associated with attacks of nephritic colic, which imply an obstruction of the ureter, point to pelvic involvement.

Calculous cystitis may be suspected when pain in the region of the bladder is excited by motion, as in riding over a rough road, or at the end of the penis after micturition; and when there is blood in the urine or when the stream of urine is suddenly interrupted. These symptoms should immediately suggest the use of the sound, negative results with which must not, however, be accepted without qualification, as the stone may be concealed in a diverticulum.

Prognosis.—The medical treatment of cystitis does not furnish a very satisfactory chapter in therapeutics. It includes such treatment as the physician is called upon to use, supposing the exciting cause, such as a stone in the bladder or obstruction in the urethra, to have been removed wherever possible. I say when possible, because the enlarged prostate which is responsible for so many cases of cystitis is, in the vast majority of cases, not removable even in these days of brilliant surgical results. It may be that castration, lately practised for the cure of enlarged prostate, is the boon so long desired. It must also include the treatment of a certain number of cases in which no removable cause is ascertainable, as well as cases where, as with a long previous gonorrhœa, the cause has long since been removed, but has left a deep-rooted tendency scarcely eradicable. Many cases get well; others are only partially relieved. Some of the most virulent acute cases terminate favorably with rupture into the vagina or rectum if the patient resist the primary attack of the disease.

Treatment.—*Acute Cystitis.*—Of this form the treatment is far more satisfactory, at least so far as the removal of the acute symptoms is concerned, than that of the chronic form. Rest in bed is a primary and essential condition. Leeches to the perineum should be applied more frequently than they are. A poultice to this same region and over the

lower abdominal region is always useful, while a brisk saline cathartic should never be omitted.

As the feverish state which always accompanies cystitis is more or less constantly associated with a scanty urine, concentrated and irritating to the inflamed mucous membrane, it is desirable at once to increase the secretion and thus dilute it. Copious libations of pure water, to which the citrate or acetate of potassium is added, in 15- to 20-grain (one to 1.3 gm.) doses for an adult, should be allowed. The ordinary spirit of nitric ether in drachm (3.4 c. c.) doses every two hours is an admirable adjuvant, and may be combined with the officinal liquor potassii citratis, which contains about 20 grains (1.3 gm.) of citrate of potassium to ½ ounce (15 c. c.). Formerly the mucilage of flaxseed or flaxseed tea was much used as a diluent menstruum for the diuretic alkalies indicated, but it is doubtful whether it is any more efficient than a like quantity of water. Where there is much pain and straining, as is often the case, especially where cantharides are the cause of the inflammation, opium is indispensable, always in the shape of a suppository, ½ grain (0.03 gm.) to one grain (0.065 gm.) of the extract, or a corresponding amount of morphine. Iced-water injections into the rectum, or pieces of ice similarly applied, are very efficient in allaying the pain and irritation where additional measures are needed. I have recently found injections of cocaine into the bladder useful in allaying the intense irritation. Not more than two grains (0.13 gm.) of cocaine should be introduced into the bladder at one time.

Chronic Cystitis.—The successful treatment of chronic cystitis is a much more difficult task, for three evident reasons:—

(1) The constant presence in the bladder of the urine with its irritating qualities, especially to an inflamed mucous membrane.

(2) The difficulty in getting remedies to reach the inflamed surface; and

(3) The pent-up inflammatory products, which in their decomposition often make the urine still more irritating by exciting in it ammoniacal changes.

There is no doubt that, if the urine could be kept from entering the bladder during the existence of an inflammation, the latter would rapidly heal; that cure would be facilitated by obtaining ready escape for the pus and mucous; while happier results might also be reasonably expected if we could secure readier access for remedies to the inflamed areas. None of these indications can be met entirely. They remain, however, the conditions to be fulfilled, and while none can be thoroughly fulfilled, they may be variously approximated.

First, the irritating qualities of the urine may be diminished by the use of diluents, already recommended in the treatment of acute cystitis. Almost any of the negative mineral waters, so highly recommended by their owners, are useful for this purpose. Just as good is pure spring water, and even better is distilled water. From one to two quarts should be taken daily. If the kidneys are equal to their office, a large quantity of light-hued urine, of low specific gravity and relatively weak in solids, will be secreted.

When it is proposed to go further and add to the efficiency of diluents, mistakes are often made. While one can scarcely go astray in adding alkalies to the fluid ingested in acute cystitis, it is very different with the chronic form. In this the urine is often alkaline, or ready to become so on the slightest addition of alkali to the blood. Such alkalinity of urine in turn favors decomposition, the effect of which is to convert the pus, if present, into a tenacious, glairy fluid which the bladder cannot evacuate. Notwithstanding this tendency, liquor potassæ and other alkalies are sometimes administered under precisely these conditions—adding fuel to the flame. The indication under these circumstances is to render the urine acid, if possible, although the means to this end are unsatisfactory. Benzoic acid has the reputation of doing this, and it probably is true of it when administered in sufficient doses. It may be given in the shape of a five-grain capsule, of which at least six must be given in a day to produce any effect. The same property has been assigned to citric acid, but this is a mistake, as all of the vegetable acids, when ingested, are eliminated as alkaline carbonates.

The second indication is to medicate the inflamed surface. Two ways suggest themselves :—

(1) By the internal administration of drugs.

(2) By the injection of medicated liquids into the bladder.

To carry out the first method, an enormous number of infusions, decoctions, and fluid extracts of vegetable substances have been suggested, the vast majority of which are absolutely useless, except as they serve by their quantity to act as diluents. Among the best known of these are buchu, pareira brava, uva ursi, and triticum repens. I have never known any beneficial results from any of them, and have long ago ceased to prescribe them except occasionally as vehicles.

The only class of remedies I have found of service in cystitis through their internal administration are the balsams. Of these, the balsam copaiba is practically unavailable, because not one stomach in a hundred will bear it in sufficient doses or for long enough time to permit it to be of any use. Sandal-wood oil is easier borne and is also an efficient remedy. It is the best administered in capsules containing ten minims. Contrary to the usual custom of giving these and like remedies after meals, I have given them on an empty stomach before meals. They are as well, and even better, borne than when given after food, and they pass into the blood much more quickly. It is desirable to impregnate the blood and impart to the urine a balsam odor. This is scarcely possible with less than eight capsules a day—two before each meal and two at bedtime. Both boric acid and benzoic acid are useful adjuvants to the treatment of chronic cystitis through their antiseptic effect on the urine, each in five-grain (0.32 gm.) doses rapidly increased to ten grains (0.65 gm.). They may be given jointly as in the following prescription: ℞. Sodii Biborat., Ac. Benzoic, āā gr. x (0.65), Infus. Buchu, f℥ij (60 c. c.). Three times a day. Resorcin in five- to ten-grain (0.32 to 0.65 gm.) doses, and naphthalin in two-grain (0.13 gm.) doses are recommended for the same purpose. Salol has become a popular remedy, very large doses being advised, 15 to 30 grains (one to two gm.) every three hours,

but in my experience these doses are not well borne, ten grains being a maximum dose.

The application of remedies to the bladder by injections is best considered in connection with the third indication—the getting rid of inflammatory products, the pus and mucus, and the matters resulting from their decomposition. The latter are not always present, but all who have had much experience with cystitis are familiar with the tenacious, glairy mucoid matter, which will not drop or rise up in a pipette, glistening with large crystals of triple phosphate, and exhaling a stinking, ammoniacal odor which quickly contaminates an entire apartment. There is only one way to get rid of this, and that is to wash out the bladder, and often this is too long deferred. Tepid water should be first used, and the injection made through the soft, flexible catheter. Sir Henry Thompson is very emphatic in his directions that no more than two ounces should be thrown in at a time, and that this should be allowed to run out, a like quantity again injected and allowed to run out, and this repeated until the water comes out as clear as it enters. But double this quantity may be used with entire safety, and with such quantity used a much shorter time is necessary to cleanse the bladder thoroughly; and after the capacity of the bladder has been determined, even more may be thrown in, because it is sometimes useful to distend the viscus a little in order to reach the depressions and inequalities, always present in advanced bladder inflammations. These simple injections, practised once a day, or in severe cases twice a day, often result most happily. After a few injections with plain water some medication may be added. Salicylate of sodium in the proportion of a drachm (four gm.) to the pint (one liter) is one of the best. Its disinfecting qualities are undoubted. Boric acid, in the proportion of a drachm (four gm.) to the pint (one liter), is also very satisfactory. Sir Henry Thompson's soothing solution—of biborate of sodium an ounce (30 c. c.), glycerine two ounces (60 c. c.), water two ounces (60 c. c.), and of this mixture ½ ounce (15 c. c.) to four ounces (120 c. c.) of tepid water—may also be used. Nitrate of silver of the strength of ¼ grain (0.016 gm.) to the ounce (30 c. c.), increased to ½ grain (0.03 gm.), is often very efficient in diminishing pus. The bladder, if previously washed out by boric-acid solutions, should be irrigated with plain warm, sterilized water, in order to avoid chemical reaction between the nitrate of silver and the boric acid.

Alum is an astringent which has been too much overlooked of late in suppurating processes in mucous membranes, and may be substituted for the salicylate with advantage where the pus does not diminish as rapidly as is desired. It should be more cautiously used than the salicylate of sodium. Sufficient of the powdered alum should be first added to a pint of water to give it a distinctly astringent taste, when the bladder should be washed out as described, while a small quantity may be allowed to remain after the last injection.

Where there is a foul odor present, the bichloride of mercury may be used in exceedingly dilute solution, not more than one to 25,000 at first, gradually increasing the strength if it is well borne. Carbolic acid may also be used in weak solution, ¼ to ½ per cent., also peroxide of hy-

drogen, one part to four or five of water. Among other remedies recommended to be used the same way are acetate of lead, one grain (0.06 gm.) to four ounces (120 c. c.); dilute nitric acid, one or two minims (0.06 or 0.12 c. c.) to the ounce (30 c. c.).

Anodynes are indispensable in many cases of cystitis to relieve the patient of extreme pain and the frequent desire to pass water, which are the result of the same cause. Opium and its alkaloids are the most efficient, and they are best introduced by the rectum. There appears to be no absorbing power in the bladder for opium, and there is no use in attempting to administer anodynes by that channel. Cocaine, from which so much might reasonably be expected, has been disappointing. I have injected as much as two ounces (60 c. c.) of a one per cent. solution into the bladder, representing eight grains (0.5 gm.), without effect, except to produce some of the symptoms of cocaine poisoning. Most disappointing, too, has been the use of cocaine to remove the exquisite tenderness of the urethra which sometimes attends this condition, and is a serious drawback to the use of the catheter; yet it may be tried for both purposes.

Where there is greatly enlarged prostate, catheterization is indispensable, and is attended often with the most happy results. It is often too long deferred because of the natural repugnance to the use of the instrument. The patient or his friends should be taught to use the catheter and to wash out the bladder. In these days of refined antisepticism it is scarcely necessary to say that the extremest precautions should be taken to cleanse the catheter after its use, in order to avoid sepsis. There is nothing better for this purpose than the bichloride solutions one to 1000, in which the catheter should be allowed to lie for a short time after being cleansed with boiling hot water.

How much can be accomplished by such treatment as the above described? An absolute and total cure in chronic cystitis is a rare event. On the other hand, a life of suffering may be converted into one of comparative comfort, and I have many times seen it. It occasionally happens, of course, that all treatment of this kind fails, and yet the patient lives to be tortured by the discomfort of the situation. In such cases perineal section may be recommended, and I have had the operation done several times with some relief, although with less than was hoped for. The operation of castration, strongly advocated by J. William White, offers some prospect of relief in cases due to enlarged prostate. It recommends itself by its comparative freedom from danger.

STONE IN THE BLADDER.

All that has been said in a general way of stone formation and the treatment of its tendency when treating of nephrolithiasis may be applied to stone in the bladder.

Symptoms.—The symptoms of stone in the bladder are practically those of cystitis, already described, aggravated by motion, especially riding over rough roads. As distinctive of stone in the bladder is pain at the end of the penis immediately after micturition. The only proof, how-

ever, of the presence of stone is its recognition by the sound, which should be used in every case of cystitis.

Treatment.—For removal of stone in the bladder medical treatment is even less efficient and operative treatment is, if possible, more imperative.

NEUROSES OF THE BLADDER.

Physiology and Its Derangement.—A proper understanding of nervous derangements of the bladder will be facilitated by a review of its physiology. The control of the bladder over the urine is partly reflex and partly voluntary. The reflex centre is the lumbar enlargement of the cord, the voluntary in the cortex of the brain. In the lumbar cord seem, however, to be lodged two reflex centres, one for the *detrusor vesicæ* and the other for the *sphincter*. If the bladder be partly full and at rest, its control is given over to the sphincter, whose striated fibres are reflexly active. As the bladder becomes fuller the unstriped fibres of the detrusor muscle are more and more stimulated, and a time arrives when the sphincter can maintain itself only by the assistance of the will if the subject be in a normal state. In such state, too, under suitable circumstances the will suspends its control over the sphincter and the bladder is emptied.

If the spinal cord is cut above the lumbar enlargement, voluntary power to aid or suspend the action of the sphincter is lost, and the urine escapes more or less continuously or as soon as a sufficient amount accumulates to stimulate the extrusor. This is one of the forms of incontinence.

If, on the other hand, there is paralysis of the detrusor muscle, and the sphincter remains intact, there will be retention of urine. If, however, communication with the brain remains intact, by an act of the will the reflex contraction of the sphincter may be suspended and the bladder partially emptied by a straining effort, at least so far as pressure can be exerted by the abdominal muscles. Should the afferent or sensory nerves of the reflex arc be paralyzed either alone or in conjunction with the efferent to the detrusor, the bladder will become enormously distended; but if the distention continue, a point is reached when the sphincter is paralyzed by over-stretching, when incontinence occurs and the urine dribbles away. The same effect takes place if there is destruction of the cord in its lumbar enlargement. So long as the cord is intact the patient may partially empty the bladder by abdominal pressure. Again, if paralysis of the *sphincter vesicæ* occurs, incontinence succeeds as soon as urine has accumulated sufficiently to overcome the elastic closure of the bladder orifice. It may be also slightly delayed by voluntary innervation of the sphincter, but is unrestrained during sleep. Hence at such time the patient wets the bed. Such incontinence is also manifested when the patient coughs or in any way sudden pressure is brought to bear on the bladder. It is often seen in women who are said to have "weak" bladders. Combined detrusor and sphincter paralysis is followed by *dribbling* away of urine as soon as enough accumulates to overcome the elastic

closure of the urethra, because there is no contraction of the bladder, and the outflow is a mere overflow. See figure, p. 796, introduction to study of Nervous Diseases.

Muscular Spasm of the Bladder.

Symptoms.—In detrusor spasm sudden evacuation of the bladder takes place. This occurs in hyper-irritability of the mucous membrane of the bladder or of the reflex centre in the cord, as soon as a small amount of urine accumulates in the bladder. It may be controlled to a degree by a voluntary impulse to the sphincter, but at other times is irresistible, and is especially prone to occur during sleep. To this class of cases belong many of the instances of incontinence in children.

In spasm of the sphincter, on the other hand, the orifice is kept forcibly closed, though it, too, may be intermittent at times by the will, permitting thus a small quantity of urine to be discharged at a time. As the urine accumulates the discomfort increases still further, when an attempt is often made to empty the bladder by straining efforts. This sometimes reacts on the sphincters, producing further contraction, which may extend to the bulbo-urethral and sphincter ani muscles, causing intense distress. Such spasm, too, forcibly resists the introduction of a catheter. It may be due to hyper-excitability of the sensory reflex centre or irritation applied directly to the neighborhood of the sphincter, such as intense inflammation.

A combination of spasm of the detrusor and sphincter may exist, giving rise in high degrees to intense suffering. It may be caused by a simultaneous irritation of the two reflex centres in the cord or intense irritation of the mucous membrane of the bladder reflected to both sets of muscles.

In addition to the central nervous affections chiefly of the cord which may occasion these symptoms, modifications in the sensibility of the mucous membrane of the bladder, of the deeper urethra, and prostate may also occasion them. These changes may be associated with inflammation or they may be purely neurotic. R. Ultzmann has refined the subject of neuroses of the genito-urinary system to a high degree, in the course of which he refers many symptoms of the kind described to an "exalted reflex excitability" caused by "over-strained physical but especially by exciting mental activity," long kept up. Among these he mentions fright, pain, grief, loss of property, and the like, as well as the "gonorrhœal process," excess in venery, and masturbation, apart from the organic processes of hyperæmia and even inflammation which may be due to gonorrhœa. The pure neurotic representatives of this class are unattended with changes in the urine, which is normal in every particular. These are not very uncommon and they are often extremely difficult to treat successfully.

A comparatively frequent representative of this class is due to a hyperæsthesia of the vesical mucous membrane, as the result of which the presence of the smallest quantity of urine gives rise to a pressing desire to empty the bladder, which is accomplished with spasm, pain, or other discomfort. As the result of this the patient has to empty his bladder

often, several times an hour, but much less frequently, if at all, at night. The urine is as a rule normal, and though sometimes concentrated, with a proportionate specific gravity, is still no more so than that which is commonly retained with perfect comfort. This occurs also sometimes in women.

Occasionally there is absolute loss of sensation in the vesical mucous membrane, apparently also functional, in consequence of which the urine accumulates without exciting the attention of the patient and the bladder becomes thus over-distended.

Treatment.—*Of Incontinence.*—Previous to instituting treatment for these conditions the most careful inquiry must be made as to the cause and its removal sought. This is often impossible, and treatment must then be empirical.

Incontinence most frequently calls for treatment. If due to disease of the cord, it is amenable to treatment so far as such disease is, and in the meantime the patient must be protected by catheterization from the over-distention which is so apt to precede incontinence. Incontinence due to weak sphincters demands that this weakness should be treated by full doses of strychnine, which may be advantageously given gradually ascending. Tincture of nux vomica may be substituted in ascending doses. Electricity has lately been highly commended for this form of incontinence in the shape of faradization, one pole being applied to the lumbar part of the spine and the other in the urethra, vagina, or perineum, the sittings being continued for a few minutes each day or every other day. Cold douches to the perineum are also useful.

If incontinence is due to hyperæsthesia of the mucous membrane or irritability of the bladder belladonna is the accepted remedy. It should be given in ascending doses and toward evening if it be nocturnal incontinence so common in children. The physiological effect of the belladonna should be produced. The bromides may be combined with it or used separately. If there is irritability of the lumbar cord ergot commends itself through its effect of diminishing congestion of the cord. The urine should receive attention, since a high degree of acidity, the presence of sediments of uric acid, and oxalate of lime may become the exciting causes of incontinence.

Habit is sometimes a cause of incontinence in children, and encouragement of a cautious practice of holding the water may gradually correct the evil. General ill health and irregular habits are sometimes responsible, and when these are corrected the patient recovers. Phimosis is sometimes a cause and should be corrected if present.

Of Retention.—An over-full bladder should always be relieved by the catheter, and catheterization should be repeated as often as necessary to prevent recurring distention while the cause is being treated. When it is weakness of the detrusor muscle, strychnine will be of service. Electricity may also be used here, when a pole should be placed behind the pubis and the other to the lumbar region.

If retention is due to spasm the cause of this should be carefully sought. The same irritations referred to as causes of incontinence may act to produce it, and some of the same remedies are useful to relieve it,

as belladonna and the bromides. Warm sitz baths, and full baths, and enemata of warm water may be used at a temperature of 95° F. (35° C.) two or three times a day. In the event of failure with these measures, more powerful anodynes may be used, including opium and morphine. These are best administered in the shape of a suppository containing ½ grain to one grain (0.033 to 0.066 gm.) of extract of opium and ¼ grain (0.0165 gm.) of morphine. Ultzmann recommends in the cases of frequent micturition, due to hyperæsthesia in the neighborhood of the prostate, injections through the prostate by a catheter which just reaches the membranous portion of the urethra with ¼ to ½ per cent. solution of carbolic acid, a ½ per cent. solution of sulphate of zinc, increasing as it is borne to three, four, and five per cent. strengths. These should be used once a day with a syringe holding four ounces (100 gm.), and the whole quantity should be thrown into the bladder through the prostate.

Other forms of spasm must also be treated by sedatives, and strange as it may seem, the passage of a sound will sometimes relieve such.

Unfortunately, the causes of either of these conditions cannot always be ascertained, and a cure must be secured by passing from one remedy to another until the correct one is arrived at.

HEMORRHOIDAL VEINS OF THE BLADDER.

Excluding all other causes of hemorrhage of the bladder heretofore considered, such as villous cancer, stone, and tuberculosis, there remains a cause of hemorrhage which, by exclusion, resolves itself into a hemorrhoidal state of the veins. Its subjects are among older persons, rarely under sixty, it is rather copious and yet rarely fatal,—in my experience never,—though fatal cases are reported.

Great care should be taken in the study of cases of this kind in order to make sure that the hemorrhage is not due to the more serious causes already considered. Otherwise a serious mistake in prognosis as well as diagnosis may occur. The bladder should be carefully explored by the sound and if necessary by the endoscope.

Treatment.—Hemorrhages from this source may occur and not be repeated, and it is this favorable termination in the absence of stone or malignant disease, on which we are sometimes unfortunately compelled to rely, which occasionally determines the diagnosis. Should the hemorrhage continue, astringent solutions, ½ per cent. and upward of alum and sulphate of zinc, may be injected into the bladder, always using the soft catheter. Absolute rest in bed should also be insisted upon. At the same time the astringent drugs and mineral waters recommended under the treatment of hæmaturia may be tried, but it is hardly to be expected that astringent effects can be produced in the bladder through the route of the circulation by medicines administered by the mouth.

MORBID GROWTHS OF THE BLADDER.

The bladder is subject to myoma, myxoma, sarcoma, and carcinoma, especially the variety known as villous cancer or papilloma; also tuberculosis. Carcinoma may be primary, but is commonly secondary. The simplest histioid tumors are not clinically recognizable one from the other.

Carcinoma of the bladder may be suspected if in addition to the usual symptoms of cystitis hemorrhage is copious and persistent, if there is carcinoma elsewhere, and if there is rapidly developed cachexia, and especially if there are other signs of secondary cancer in the vicinity. Occasionally villi of the papillomatous growth are passed in urine and easily recognized. Unless thus fortunate, the only certain means of diagnosis in the male, though not always to be relied on, is the endoscope, which, in the hands of a skilful manipulator, affords valuable assistance. In the case of the female, it is easier and quite as satisfactory to dilate the urethra under ether and explore with the finger, though the endoscope can be used.

The symptoms of tuberculosis of the bladder are those of cystitis, and the recognition of the bacillus of tuberculosis by microscopic examination affords the only sure means of diagnosis between it and other forms of inflammation of the bladder. It is, however, relatively easily found when present, especially if the urinary centrifuge is used.

Treatment.—If the diagnosis of villous cancer can be made early, the life of the patient may be prolonged by scraping the bladder, but in my experience the growth returns sooner or later. In the few instances where I have had surgeons operate for me in true cancer of the bladder the result has been unfortunate—the patient perishing soon after the operation. The palliative treatment is that of cystitis. At the same time the counsel of the surgeon should be promptly sought.

The local treatment of tuberculosis of the bladder is that of cystitis. It demands the same general treatment as tuberculosis elsewhere.

SECTION VIII.

DISEASES OF THE SUPRARENAL CAPSULE.

ADDISON'S DISEASE.

The suprarenal capsule is subject to the following diseases: tuberculosis with fibro-caseous and calcareous degeneration; cystic degeneration; fatty degeneration; simple atrophy; chronic interstitial inflammation which may lead to atrophy; malignant disease, including carcinoma and sarcoma; hemorrhagic extravasations; embolism.

Symptoms.—Some of these morbid states appear to be totally without symptoms, the conditions having first come to light at autopsy. Further, the same symptoms may be produced by any of the different morbid states, and there are none distinctive for any one state. Thus any one of them if sufficiently prolonged may produce *pigmentation* or *bronzing* of the skin, first described by Addison as associated with disease of the suprarenal capsules, an association which, since the publication of his paper in 1855, has been called Addison's disease. The clinical concept thus named is especially a disease of the lower classes, Dr. Greenhow having found nine-tenths of cases among laboring people. It is also a disease of adults, being rare under thirty-five years. The most frequent lesion thus associated is the fibro-caseous tubercular one. But, as stated, the pigmentation may accompany any one of the lesions mentioned, if prolonged, or may be absent in the presence of any one.

As to the *coloration* itself, which is usually the first symptom to attract attention, it varies from a light yellow to a deep brown and even almost black. It is deeper on the more exposed parts of the body, where the normal pigmentation is greater, and therefore is commonly first seen on the face and hands. In rare instances only is it general. It is associated at times with patches of absent pigmentation, leucoderma. It is noticeable also at times on the mucous membrane of the mouth, conjunctiva, and vagina, and very rarely even upon serous membranes in patches.

As stated, there are no other symptoms which are distinctive of any one of the lesions of the suprarenal capsule described, but among those which are more or less constantly associated are *anæmia*, *extreme debility* and *general languor*, *irritability of the stomach*, and quite often *diarrhœa*. The irritability of the stomach is manifested by anorexia, nausea, and vomiting, and may be a very early symptom. The heart's action is feeble, the pulse correspondingly small and rapid, and there is

also often a tendency to fainting. There is *dyspnœa*. At other times there is headache. *Mental hebetude* goes *pari passu* with bodily weakness, while the other symptoms commonly associated with the latter condition are also present, namely, dizziness and ringing in the ears. Ultimately the *asthenia* becomes so profound that the patient cannot rise, but keeps his bed, growing weaker and weaker till he dies of sheer exhaustion. Sometimes there are *convulsions*, possibly due to brain anæmia.

The *urine* is usually normal, although occasionally there is polyuria, and sometimes the urinary pigments have been found increased.

Great pains have been taken to explain this peculiar "bronzing" ascribed by Addison himself to the loss of function of the adrenals. Two chief theories have been suggested:—

(*a*) By experimental evidence it has been sought to show with some reason that these glands furnish some sort of internal secretion essential to normal metabolism. To explain those cases in which the adrenals are diseased and yet there is no pigmentation, it is suggested that accessory adrenals may be present, though none have been demonstrated so far as I know; while the presence of pigmentation in association with healthy adrenals is ascribed to disease of the adjacent semi-lunar ganglia interfering in some way with the blood-vessels or lymphatics of the glands. Such a theory, that a secretion furnished in health by the suprarenal capsules is necessary to normal metabolism, is analogous to that which ascribes myxœdema to the loss of function of the thyroid gland and has acquired some additional support by the recent knowledge acquired upon this subject.

(*b*) According to the second theory the pigmentation is ascribed to disease of the abdominal sympathetic system itself, commonly associated with disease of the adrenals, but also at times caused by other chronic disorders which invade the solar plexus and ganglia. In one of my own cases in which all the symptoms, including pigmentation, were conspicuous, and which came to autopsy, there were found in addition to the advanced tuberculosis of the suprarenal capsules also enlarged semi-lunar ganglion. The latter view would make it a disease of the nervous system and the pigmentation a trophic phenomenon.

Diagnosis.—It is probable that pigmentation alone, at least unless it be very decided and general, is never sufficient to justify a diagnosis of suprarenal disease, since other abdominal affections are known to produce a similar condition. Among these are tuberculosis of the peritoneum, cancer, and lymphoma; pregnancy, uterine, and even hepatic disease. The popular notion that every discoloration of the skin is due to some derangement of the liver (liver spot) has scanty foundation. In the hardening of the liver sometimes associated with diabetes pigmentation has been noted. All of these facts go to show that the nervous system must have some powerful influence and to support the second theory. The same testimony is afforded by the pigmentation which attends exophthalmic goitre. Protracted filthiness also produces discoloration of the body which is not distinguishable *per se* from that of Addison's disease. Deep, general pigmentation has been found associated with melanotic cancer.

Finally, pigmentation is sometimes the result of the prolonged administration of arsenic. It is well, therefore, to seek carefully for other signs and symptoms than pigmentation before a diagnosis is made. In the case of my own referred to, there was pulmonary tuberculosis and tuberculous disease of the spine with pigmentation and asthenia, on which was based the diagnosis of Addison's disease, which was confirmed by autopsy.

Prognosis.—In a well-determined case of Addison's disease, as might be inferred from the nature of the causes, recovery is impossible, though the disease is commonly a prolonged one and improvements take place. In a few cases only the course is rapid. From eighteen months to several years usually cover the duration.

Treatment.—This is principally symptomatic. We aim to restore the condition of the blood, and, of course, above all, iron is indicated. It may be associated with that other tonic so constantly used with iron especially, arsenic. An excellent preparation of arsenic is the solution of the chloride, which is as good as Fowler's solution and mixes nicely with the chloride of iron. The doses are the same as those of Fowler's solution, from three to five minims (0.18 to 3 c. c.).

Or the iron and arsenic may be given in pill form as the carbonate of iron and arsenious acid and to this strychnine may be conveniently added. If very asthenic, the patient should be kept in bed and fed on alcohol with nutritious, easily assimilable food, of which peptonized milk and broths, beef juice, cod-liver oil, and glycerine are the type. The diarrhœa should be treated as other diarrhœas, with bismuth and other remedies. For the nausea the usual gastric sedatives, including ice, carbonic acid water, champagne, milk and lime-water in small doses, koumyss, whey, and the like are suitable.

With the knowledge which has grown out of the treatment of myxœdema with thyroid extract no treatment of the combination of symptoms known as Addison's disease would be complete without the administration of some similar preparation of the adrenal. George Oliver has had prepared an extract in the shape of the tincture, a glycerine extract is made, and the glands are eaten fresh or dried. The equivalent of two a day is recommended. No sufficient results have as yet been published.

SECTION IX.

CONSTITUTIONAL DISEASES.

RHEUMATISM.

Historical.—The term rheumatism was originally used to indicate morbid conditions associated with mucous discharges (Gr. *ῥευμα*, a flux), conditions to which the term catarrh was later applied and is still used. Rheumatism and gout were originally confounded under the name *αρθριτις*, first applied to gout and so used by Hippocrates (B. C. 460-357) and Aretæus (latter half first beginning second century A. C.). Sydenham (1633) was the first to separate the two conditions and describe them intelligently.

Rheumatic Fever.

Synonyms.—*Acute Rheumatism; Acute Articular Rheumatism; Inflammatory Rheumatism.*

Definition.—An acute febrile, infectious, but non-contagious disease, characterized by multiple arthritis.*

Etiology.—While no distinctive bacterium has as yet been isolated, Hermann Sahli found in the diseased joints in which there was no suppuration a bacterium closely resembling the staphylococcus citreus, and Leyden a diplococcus differing from that of pneumonia. An exciting cause seems, however, to be necessary in the majority of cases, and exposure to cold is the most common, although epidemics of acute rheumatism occur quite independently of such exposure. While sudden changes in temperature, also, often afford the needed conditions, the continued action of moderate degrees of cold, especially when accompanied by moisture, is almost as frequently responsible. If to these be added a lowered vitality due to insufficient food, fatigue, over-work, or all of these combined, we include most predisposing causes. The winter and spring, being the seasons in which the conditions of temperature and moisture operate most strongly, are those in which the disease is most prevalent. For a like reason it is more common in the temperate zones, the extreme north as well as the extreme south being for the most part exempt. In my own experience, the late spring finds many cases due to the cold and

* I am aware that in defining rheumatism as an infectious disease, it would have been more strictly consistent to treat it among diseases of the infectious class, but it still possesses so much in common with diseases of the class in which I place it that I shall retain it here for the present.

dampness of houses where fires have been prematurely dispensed with. It is a disease especially of young adults, being rare before fifteen and after fifty; while the exposing occupations, including those of driver, servant, and laborer, are more subject to it.

It may be still in place, in connection with the newer etiology, to allude to two of the older theories of acute rheumatism. According to the *metabolic* theory, a morbid material is developed in the economy as the result of defective assimilation. Prout early named lactic acid as the peccant material, and more recently P. W. Latham has suggested combination of lactic acid with other substances. The *nervous* theory was suggested by the late John K. Mitchell in 1831. According to it the nerve centres are affected by cold and the local lesions are trophic in character, or defects of metabolism result from the primary nervous lesion, whence arises lactic acid, which accumulates in the blood.

Acute rheumatism is a disease simulated by other affections not infrequently called rheumatic. Thus, scarlet fever is often accompanied by a painful swelling of the joints due to the specific cause of that disease and called rheumatic, when it should be spoken of as scarlatinal synovitis. The same is true of the so-called gonorrhœal rheumatism, which is not a rheumatism, but a gonorrhœal synovitis due to a specific cause or a remote consequence of it.

Morbid Anatomy.—There is little to be added to what will be described in treating of symptoms, and to what is furnished by the complications whose morbid anatomy will also be considered in connection with the diseases which constitute them. The synovial membrane is hyperæmic and swollen, and in some cases the fluid in the joints is increased, is turbid, and contains flakes of lymph, rarely pus. There may be slight erosion of the cartilages. The fibrin of the blood is usually increased.

Symptoms.—While rheumatic fever is *seldom* ushered in by a *chill*, there is more frequently a short *prodrome* of a day or two, during which the patient feels uncomfortable or has an unpleasant aching feeling in his joints. More often, however, the *pain in the joints*, which is the first symptom to attract attention, develops rapidly, coming on in a single day or night, or seemingly in a much shorter time, making locomotion at once difficult or impossible.

The joint-affection has some peculiarities. In the first place, the involvement is almost always multiple, and generally includes the larger joints, such as the knee, ankle, elbow, wrist, shoulder, and hip, although none are exempt, and the phalangeal and metacarpo-phalangeal articulations also suffer. The toe-joints escape most frequently. It rarely happens that a single joint is involved, but its occasional occurrence must be admitted. More rarely, if ever, does it happen that all are affected, although even the vertebral articulations must sometimes be included. The joint-inflammation is further characterized by a tendency to fly from one joint to another. Now it will be the elbow, then the wrist; again, the knee, and then the ankle or shoulder or hip, either on the same side or the other; but while there will be a reduction in the degree of the inflammation and correspondingly of pain in the relieved joints,

the relief will not be total. On another day, again, the pain will have returned to the joint which had been temporarily relieved.

While the joint-affection always includes a synovitis, the process is by no means confined to the synovial membrane. The adjacent structures, including the capsular and lateral ligaments, and the tendons, with their sheaths, coursing over the joint, and even muscles, are all the seat of involvement, contributing to the swelling and to the pain by the exudation pervading them. Comparing two hands, one of which is involved and the other not, one can often see the depressions between the metacarpal bones in the former obliterated by swelling, while they maintain their usual distinctness in the latter. It is for such reasons that I prefer the name "acute rheumatism" to that of acute "articular" rheumatism, which would limit the process to the joints.

The pain is almost always extremely severe, making all motion an agony, while jarring of the bed, the pressure of bed clothing, and even the motion of the floor produced by some one walking over it may cause the patient to cry out with pain. To diminish the tension which aggravates the pain, the patient is disposed to lie with all the limbs semi-flexed.

From the beginning there is *fever*, but being seldom high at this stage, it is not commonly the first symptom to attract attention. Later, it usually increases proportionately to the extent of joint involvement, but only in the meningeal form is it extremely high. Nor does it pursue a course at all distinctive. In a case now under treatment, for example, the temperature remained at 102° F. (38.8° C.) and a fraction night and morning and throughout the day for a number of days. More rarely it rises to 104° F. (39.9° C.). Occasionally, however, there is intense hyperpyrexia, when the temperature rises rapidly from 104° to 110° F. (39.9° C. to 44.3° C.) and even higher. With this is associated cerebral symptoms of an alarming and dangerous kind, intense headache, and delirium, symptoms otherwise rather unusual in acute rheumatism. To these are often added unconsciousness, pulselessness, and cyanosis, rapidly followed by death, unless the temperature is promptly reduced. The sudden onset of these symptoms adds to their alarming character. This combination of severe symptoms is known as the meningeal form, or *rheumatism of the brain.*

The *pulse* in rheumatic fever is rapid, often disproportionately so to the fever, probably because of the nervous demoralization caused by the acute suffering.

Next to the fever and joint-inflammation, the most distinctive symptom of acute rheumatism is the *sweating*, which is copious and usually acid in reaction, sometimes even to such extent as to impart an acid odor to the air of the room. *Sudamina* are a frequent consequence of such profuse sweating.

Discolorations of the skin, varying in intensity and character, make their appearance in certain cases. There may be a simple diffuse erythematous redness, or it may be papular or tuberculated or marginate. There may be true *urticaria*, or there may be extravasations of blood, *purpuric* patches of such extent and depth as to result in sloughing of the tissues, hemorrhages from the mucous membranes, and hæmaturia.

In one case under my observation there ensued permanent blindness from extravasation into the retina. These cases of *peliosis rheumatica* are not acknowledged by all to be truly rheumatic in their origin, the joint-affection being declared to be of a different nature, analogous to what occurs in scorbutus and hæmophilia.

The *urine* is also somewhat characteristic. It is scanty, of high specific gravity, very acid in reaction, and deposits a copious sediment of pink-hued mixed urates.

Very interesting and characteristic are certain *subcutaneous nodules* attached to tendons and fasciæ, which have long been observed as occasional events in connection with acute rheumatism and have recently been studied by Barlow and Warner. They vary in size from a shot to a pea and may be very numerous or but few. They occur on the fingers, hands, and wrists, the elbows, knees, scapula, spines of the vertebræ, and more particularly after the acuteness has passed away. They may last a few days or for months, and are more common in children than in adults.

Disposition to recurrence must be mentioned as a characteristic feature of acute rheumatism. Quite rarely does a person who has had one attack escape another, and it is these successive attacks which, augmenting previous cardiac lesions, finally cripple the heart until its work is greatly hampered. The intervals between successive attacks are various—from a year to four or five years—and they are the more frequent and more liable to occur the younger the subject.

Complications.—Very interesting in connection with acute rheumatism is the frequent involvement of the *serous membranes* other than those of the joints, such as the pleural membranes and the peritoneum. The involvement of the former simulates pleurisy and the latter peritonitis, and I well remember a case of my own, a girl of eight years, in whom for days I thought I was dealing with peritonitis, when a few doses of salicylate of sodium relieved my anxiety by promptly arresting the disease. In rheumatism of the pleura, the absence of physical signs aids in the diagnosis. These phenomena are easily explained with the modern views of the etiology of rheumatic fever, since we have only to suppose the infectious material circulating in the blood to lodge upon the serous membranes instead of the joint tissues.

Of the same class is a much more common complication, *cardiac disease*, including endocarditis and pericarditis, the former being by far the more frequent, and confined almost exclusively to the left heart. Again, the mitral leaflets are much more frequently attacked than the aortic. While the cardiac involvement bears some relation to the severity of the disease, the mildest cases may become complicated as well as the severest. Hence the heart should be daily examined, and for the further reason that the approach of the disease is often exceedingly insidious. On the other hand, cardiac oppression and palpitation may occur without actual structural change, and even a functional murmur may occur in acute rheumatism, and this, too, not only at the base, but also at the apex of the heart, an unusual site for a functional murmur.

The proportion of cases in which cardiac complications occur,

though difficult to estimate precisely, is not less than 25 to 33 per cent. for endocarditis with ten per cent. more for pericarditis, making in all 35 to 43 per cent., while some estimate even a larger proportion. Young subjects are more vulnerable than adults, and Fagge mentions an interesting difference in the sexes after adult life, which is, that *pericarditis* is more frequent in men above twenty-five than in women of the same age, probably because at this age men work much harder than women.

The variety of endocarditis is usually the verrucose, or warty, ulceration, laceration or perforation of the valve flaps being very rare. The malignant form of endocarditis does, however, occur. While the endocardial murmurs in the endocarditis of acute rheumatism are commonly soft, the pericardial murmurs are often loud, rough, and rasping, and the vibration resulting from the friction may even be communicated to the hand laid upon the præcordium. Both conditions may result in complete recovery, but the former more commonly is the beginning of a chronic valvular defect.

Acute myocarditis is a fatal but fortunately rare complication of rheumatic fever, occurring alone or in association with endocarditis and pericarditis. It is commonly first discovered at the autopsy, though severe epigastric or præcordial pain, embarrassed respiration, and cynosis may suggest it.

Other complications of rheumatism are probably also the direct result of the poison. They include the inflammations of the serous membranes mentioned, bronchitis, and, more rarely, pneumonia. Convalescence from the latter is said to be slow.

The *sequelæ* directly traceable to acute rheumatism are also few. Chorea, acute nephritis, and exophthalmic goitre are among those so regarded. The nephritis is, perhaps, better considered a complication resulting from the same cause, just as are the endocarditis, pleurisy, and peritonitis.

Diagnosis.—The diagnosis of acute rheumatism is seldom difficult, the multiple painful involvement of the joints, the fever, and sweating seldom mean anything else; but pyæmia and scarlatinal and gonorrhœal arthritis must be remembered as possible events. It is the monarticular variety which demands most discrimination in its determination. Traumatic synovitis, tuberculosis or white swelling, and the so-called nervous arthropathies are to be eliminated.

It is not always easy at a first visit to distinguish gout from acute rheumatism, but the most serious possible error in diagnosis is to mistake a pyæmic arthritis for a rheumatic arthritis. This is not an uncommon mistake where there is no evident surgical lesion to suggest it. Osteomyelitis is said to be the most common cause of such pyæmias; but other bone-diseases, puerperal sepsis, and gonorrhœa are also causes.

Prognosis.—The course of acute rheumatism is characterized by many fluctuations independent of treatment, and its duration is various. Sooner or later recovery generally takes place, although it may be with a crippled heart and a susceptibility to return. More rarely the attack passes over into a subacute condition which makes the patient a sufferer for a long time, while still more rarely true chronic rheumatism is the

result. It used to be said the cure for inflammatory rheumatism is "six weeks," and though this is not true of every case many are prolonged to quite this length.

Subacute Rheumatism.

This term is applied to forms in which all the symptoms are less marked and more prolonged. The fever is not so high, ranging from 99° to 101° F. (37.2° to 38.3° C.). The inflammation of joints is not so intense and the joints involved are less numerous. It exhibits the same "flying" tendency. It may also be associated with the cardiac complications, especially in children. It may pass into the chronic form.

Treatment.—Whatever may be the drawbacks to a successful treatment of acute rheumatism, and they are many, it is certain that most of those who had to treat this disease a quarter of a century ago now attack it with much more confidence than they did in that day. The drug which is responsible for this feeling is salicylic acid, and very few physicians think of any other at the outset of a typical case. The introduction of salicylic acid as a remedy for acute rheumatism is commonly ascribed to Buss, of Basle, some time prior to 1876, but attention was first prominently drawn to it in the latter year by Dr. Stricker, of Traube's clinic in Berlin.

Salicylic acid and salicylate of sodium are equally efficient, but the former has been largely superseded by the latter, because of its less irritating nature and easier administration. Whichever of the two is used, there is one necessary condition of its efficiency, and that is its constitutional impression. The aim in the administration is, of course, to relieve the patient, but this effect is seldom obtained, or, if obtained, is of fleeting character, until the peculiar ringing in the ears is secured. To do this in the adult 1½ to two drachms (5.8 to 7.7 gm.) of salicylic acid and from two to three drachms (7.7 to 11.6 gm.) of the sodium salicylate in the first twenty-four hours are required. If the salicylic acid is given, it should be in capsules or compressed pills containing 7½ to ten grains (0.49 to 0.65 gm.) every two hours, followed by a little water or milk. The salicylate of sodium may be given in doses of ten to 15 grains (0.65 to one gm.) in solution every two hours, or every hour if the pain is severe, until relief comes, after which it should be kept up until the toxic effect is produced, when the dose should be diminished, but the drug continued; or the interval may also be prolonged. Others would give the salicylate of sodium, one to 1½ drachms (5.8 to 7.7 gm.), in a single dose, but in my experience few stomachs will submit to such quantities. The doses laid down may be pushed more rapidly if the suffering is extreme, but I have seldom found it necessary. Under this treatment the pain fades away, the swelling diminishes, and the anxious expression of the patient is changed to one of comfort in from twenty-four to forty-eight hours. Those who object to the salicylate treatment do so on the ground that the relief is not permanent, and it must be admitted that relapses do occur. I am confident, however, that this is often due to the fact that the remedy is too soon discontinued. As stated, the drug, while it should be cut down with the

appearance of relief and toxic effect, must be continued for some time after relief is obtained.

Salicin, first used by T. J. Maclagen, appears to be about as efficient as salicylic acid, given in 20-grain (1.33 gm.) doses every two hours, in suspension or dissolved in warm water. It is much less irritating than salicylic acid, but has not superseded it on this account.

I do not, however, rely wholly upon the treatment by salicylates. Warmth is commonly a useful adjuvant, and to this end the joints and limbs should be kept surrounded by warm flannels or carded wool or cotton. The opposite plan, treatment by cold, is also recommended by some.

Sometimes the salicylates cannot be tolerated by the stomach, even in the smallest doses likely to be useful. They may then be given by injection as follows: The rectum is washed out with warm water, and after a short rest, 20 to 40 grains (1.3 to 2.6 gm.) or more of sodium salicylate in solution injected well up into the bowel. This may be done once in six hours with the happiest result, as I can attest from personal experience.

But the salicylate treatment is not always successful, and sometimes the drug is not well borne in any shape. Then the oil of wintergreen should be tried, in doses of ten to 15 minims (0.6 to one c. c.) every two hours, in capsules or in emulsion. Or it may be alternated with the salicylate, if it is a question of tolerance of the latter, the gaultheria being usually better borne for a time by the stomach. I say for a time, because, however pleasant wintergreen is at first, its continued use is apt also to excite disgust.

The alkaline treatment of acute rheumatism, most relied upon before the salicylic treatment came into vogue, is a treatment which is by no means worthless. This, originally instituted by Sir A. Garrod, received an additional impulse from the late Dr. H. W. Fuller, who insisted upon the administration of such doses as secured and maintained an alkaline reaction of the urine. This is accomplished by sufficient doses of almost any of the alkaline salts, as potassium citrate, potassium acetate, sodium carbonate, or *liquor potassæ* may be used. Twenty grains (1.33 gm.) every two hours of the first three are generally sufficient, or 20 minims (1.3 c. c.) of the last. The dose may then be reduced, but enough should be given to maintain the alkalinity of the urine.

Failing for any cause in the treatment with salicylic acid, the alkaline treatment, or what is called the "mixed" treatment, may be employed. By this is meant the alternate use of the salicylates and alkalies. This may be tried, for example, where sufficient doses of the salicylates are not well borne by the stomach, when they may be supplemented by alkalies.

While using the alkaline treatment before the salicylates came into use it was quite usual to combine with it the "flying" blister, one of small size,—say an inch square,—and applying it now to one joint and then to another. That this practice was efficient in relieving pain there can be no doubt. That it had any effect in cutting short the disease is more uncertain, and since the introduction of the salicylate treatment I

use it less. In the subacute and chronic stages of the disease counter-irritation by blisters or iodine is more imperatively called for.

For the relief of pain opium is sometimes necessary, but less frequently than before the introduction of salicylates. Here, again, the hypodermic injection of morphine, ¼ grain (0.016 gm.), is most comforting, but the Dover's powder in ten-grain (0.6 gm.) doses is often efficient. Phenacetin, acetanilid, and exalgin may be used for milder degrees. It is soothing to have the joints enveloped in cotton or wool.

The *treatment* of the *hyperpyrexia* of acute rheumatism must be prompt and energetic, as the danger to life is imminent, the extraordinary high temperatures thus encountered being inevitably fatal in a few hours. There is but one treatment. It is the application of cold. The bath is to be preferred, although in its absence affusions of ice-cold water and rubbing the head and body with ice may be substituted. As soon as the temperature begins to mount rapidly above 105° F. (40.5° C.) it should be used, and if delirium or unconsciousness is associated with such temperature it is even more imperative. Where time permits, the application of cold may be more gradual. Thus the patient may be put in the bath at 70° F. (21° C.) and the temperature further reduced, if necessary, by the addition of ice or colder water. As stated, there seems now to be no doubt about the propriety of this treatment. Numerous cases of recovery have been reported, some even where the temperature had reached 107°, 108°, and even 109° F. (41.6°, 42.2°, 42.7° C.) and a fraction. With the reduction of temperature, the cerebral symptoms gradually disappear.

As the disease becomes more subacute or chronic, the necessity for more active local associated with tonic treatment becomes urgent. It would seem that at such stage the pathogenic cause has exhausted itself, and the disease has become more a local one, maintained by the dyscrasic state of the blood, itself brought about by the long suffering. Hence roborant treatment with iron, arsenic, cod-liver oil, wine, and good food becomes necessary. Indeed, the patient with acute rheumatism should be well fed throughout. Counter-irritation by iodine or by blisters should be kept up, with appropriate intermissions, although the results are often slow in appearing. Massage is especially valuable, and often surprisingly soothing ultimately, even although at first somewhat painful, while the mobility of the joints is gradually restored. There results sometimes in the muscles of the neighborhood of the joint, and especially in the case of the shoulder, a paretic state, which is also benefited by massage, especially when associated with electricity.

Some allusion needs to be made to remedies now more or less obsolete which have had some reputation in the treatment of acute rheumatism. Nitrate of potassium was among the most popular of the older remedies. As much as two drachms (eight gm.) of the latter were given by Brocklesby three and four times a day. It was revived by Basham, who used it also locally to the inflamed joints. It is diuretic and diaphoretic. Guaiac is also one of the older remedies still used in chronic rheumatism, which see. The bromide of ammonium has the endorsement of J. M. Da Costa in the quantity of one to 1½ drachms (four to six gm.) in twenty-four hours.

It should be mentioned also that no less eminent authorities than Sir Alfred Garrod and the late Austin Flint, Sr., thought acute rheumatism was self-limiting, and that it terminated about as quickly without medicines as with them.

Muscular Rheumatism.

Synonyms.—*Rheumatic Myositis; Myalgia.*

Definition.—A painful condition of voluntary muscles and their aponeurotic coverings, especially aggravated by motion and pressure. It affects especially large muscles, such as those of the neck, the shoulders, the arms, the intercostal muscles, the back, thighs, and calves of the leg.

Etiology and Pathology.—Exposure to cold and especially to draughts of cool air, as from an open door or window, are the most frequent causes. The acute form, at least, does not fly about, but persists in the muscles primarily attacked until relieved.

Its true nature is unknown, and whether it is an affection of muscular substance, or of the intermuscular connective tissue, or of the minute branches of sensory nerves distributed throughout the muscles is also unknown. Certain forms of muscular rheumatism, especially of the back, are ascribed to gout. An infectious origin has been suggested. It is sometimes associated with articular rheumatism, but has probably a different etiology, though similar exciting causes operate to produce it. Similar pain often succeeds muscular strain, but it is doubtful if this should be called muscular rheumatism.

The division of muscular rheumatism into acute and chronic is based upon the duration of the pain and disposition to recurrence. The term chronic is justified for those forms which recur with changes in the weather, and are either excited or relieved by them. It, too, is less localized than the acute. On the other hand, it is not inaptly at times called wandering. It is more frequent in men than in women, because of their more frequent exposure to its cause.

Symptoms.—The only invariable symptom is *pain*, aggravated by motion or pressure. Sometimes there is *swelling*. It is usually rather sudden in its onset, requiring at most but a few hours and often less to develop it. It is never accompanied by marked constitutional disturbance. The *pulse* may be somewhat accelerated and the *temperature* approach 100° F. (37.8° C.), but more often there is no fever at all.

Muscular rheumatism is specially named according as it involves special muscles. Thus *lumbago* is a painful affection of the lumbar muscles and their tendinous attachments. The attacks come on under the conditions already named, but sometimes suddenly without discoverable cause. The suddenness of its occurrence under these circumstances, as, for example, subsequent to a simple twist or stooping, has given rise to the term "kink in the back," or among the Germans to the word *Hexenschuss*, or "witches' shot." It has seemed to me that this sudden pain was really a "cramp," being entirely analogous to the cramps which seize the muscles in other localities. The strain in most cases is altogether too insignificant to cause laceration. As a consequence, the body

is at times immovable as in a vice, so excruciating is the pain caused by motion.

Stiff neck, or *torticollis*, is an affection of the side and back of the neck, forcing the patient to hold his head to one side as the situation of least discomfort, and when he desires to turn his head he is forced to turn the whole body. Sometimes it becomes chronic and is rather difficult to cure. It is more frequently met with in children and young adults. *Omalgia* is a similar condition of the muscles of the shoulder and upper arm making motion exquisitely painful. Anchylosis of the shoulder joint may be caused by delayed motion. *Pleurodynia* affects the intercostal muscles and makes breathing and coughing very painful, while a deep breath becomes impossible and sneezing an agony. The pectoral and serratus muscles may also be involved when the pain is higher up. It is more frequent on the left side.

Cephalodynia, or rheumatism of the muscles of the scalp, *scapulodynia*, and *dorsodynia* are all forms of muscular rheumatism which explain themselves. It also affects the *abdominal muscles*, and a most interesting instance of this form simulating peritonitis was published by myself in the Philadelphia Medical Times, Vol. x, 1880.

The duration of the acute form is brief, seldom lasting for more than a few days, though there may be a tendency to relapse. The chronic forms are indefinite in duration.

Diagnosis.—This is easy for the coarser acute forms of omalgia, stiff neck, and lumbago. Muscular rheumatism may, however, be confounded with neuritis and neuralgia. In muscular rheumatism the pain is more diffuse, in neuritis there is pain and tenderness more localized and along the course of large nerve trunks. Muscular rheumatism and neuritis are distinctively worse on motion, neuralgia less so. Rheumatism is commonly relieved by the warmth of the bed, neuritis may be aggravated, while neuralgia is indifferent, though increased by cold winds. Pleurodynia is sometimes difficult to distinguish from intercostal neuralgia, but attention to the points named will prevent mistakes. Neuritis of the brachial nerve trunks resembles omalgia, but the former is early followed by atrophy, while muscular rheumatism is not. From pleurisy, pleurodynia is easily distinguished by the absence of fever and the absence of physical signs. The lancinating pains of locomotor ataxia and the pains of incipient disease of the vertebræ resemble at first those of lumbago, but the special symptoms of these diseases are soon superadded.

Treatment.—The acute form of muscular rheumatism is occasionally amenable to treatment by the salicylates and salicin. Some phenomenally prompt results sometimes follow the use of these remedies. They are, however, inconstant, and if, after a fair trial of the drug, they are not attained they should be discontinued. If efficient, the same rules as to their continued use in reduced doses after relief has been obtained apply as in acute articular rheumatism. The group of muscles treated must be placed at absolute rest, and in the case of the thorax this is best accomplished by strapping the side with adhesive plaster. Rest may, however, be overdone, and in the case of muscles like those of the shoulder atrophy may result from too prolonged a rest. Another

measure of great value is dry heat applied by means of a hot-water bag covered with flannel or by a warm flat-iron. To use a popular expression, a muscular rheumatism may thus be sometimes "ironed out." A flannel cloth should be interposed. With these measures may be associated also massage. Sometimes a single efficient treatment by massage is enough to "rub out" such a rheumatism. Of less permanent utility are hot poultices, although they allay pain at least. The same effect is accomplished by moist hot air or vapor (steam) baths, which in special establishments can be localized.

The chronic form is also treated by massage, passive motion, and electricity, either the induced or direct current. Counter-irritation by liniments, such as those made with chloroform, ammonium hydrate, or turpentine, have long enjoyed a reputation, but at the present day it is beginning to be questioned as to whether, after all, it is not the friction rather than the liniment itself which produces the good effect. Some efficiency in the liniment itself I think must still be admitted and would advise its use as determined by circumstances. Acupuncture, consisting in the puncture by needles deeply thrust into the skin, is a measure which has some advocates, especially in the treatment of lumbago. My experience with it is limited. Hydrotherapy is more likely to be useful, and here the warm or cold pack is the better method of application. Dry cupping is also often of service. General treatment should not be neglected, cod-liver oil, iron, strychnine, quinine, and good food are necessary measures where the patient is run down. Among diseases which need good feeding, chronic muscular rheumatism is preëminent.

Chronic Rheumatism.

Definition.—A term often vaguely applied which should be restricted to chronic inflammatory swelling involving the soft tissues of the joint, not due to sepsis or traumatism.

Etiology.—Occasionally a sequel of acute rheumatism, it is more frequently of independent origin in those subject to prolonged exposure to cold and dampness or changes in the weather. It is often a sequel of the subacute form, as the subacute is of the acute. On the other hand, it sometimes precedes the acute and subacute forms. It is a disease usually of middle life and is more frequent among the poor and working classes.

Morbid Anatomy.—This superficially is similar to that of acute rheumatism, but the internal joint changes are more marked. The joint is enlarged chiefly because the capsule and tendons are thickened, as are also the sheaths of the tendons, explaining the difficulty of motion and stiffness. The cartilages may be involved and eroded in chronic cases. On the other hand, the joint-tissues may be unaltered. In protracted cases, especially where there is neuritis, atrophy of the muscles covering the joints takes place, producing marked deformity. In these cases, too, there may be anchylosis. At other times there is distention of the joint with effusion, the pressure of which upon the muscles

themselves or the vessels leading to them may be responsible for the atrophy.

Symptoms.—These are chiefly *stiffness* and *pain in the joints*, including their muscular and fibrous coverings. The latter pain is characteristically increased on motion, while the stiffness is often diminished by exercise. There may be little swelling, but marked *tenderness* to the touch, though not always. All the symptoms are aggravated by cool and damp weather and often by other unknown meteorological influences. They are almost invariably better in warm weather. There is rarely any elevation of temperature worth noticing, nor is the pulse much altered. The stiffness may pass into actual immobility due to anchylosis and there may be distortions of the joints, as described under morbid anatomy. Cardiac lesions as the direct result of the cause do not occur, but gradually the valves may be sclerosed and fibroid changes take place in the muscle.

Diagnosis.—This is not usually difficult. The age of the patient, the family history, the changing amount of the pain, the multiple joint affection and the large size of the joints involved, the effect of motion, and the slight fever are sufficient to distinguish it from gout and surgical monarthritis.

Prognosis.—This, as to cure, is unfavorable, unless the patient can be removed to a warm climate. Generally, in spite of treatment, the symptoms gradually grow worse as the patient ages, though his life is rarely shortened. Yet unaccountable improvement sometimes takes place.

Treatment.—The remedies useful in acute rheumatism are of no avail in the chronic form. Occasionally acute exacerbations are relieved by the salicylates. Of drugs sometimes serviceable the iodide of potassium and bichloride of mercury may be named. Cod-liver oil, iron, and tonics are often useful by building up the strength of the patient. Guaiacum is a drug which still maintains some reputation in the treatment of chronic rheumatism. It is usually given in the shape of the tincture or ammoniated tincture, 5 to 30 minims (0.3 to 4 c. c.), preferably in milk.

Local treatment is more important than internal medicine. Counter-irritation by iodine or blisters persistently kept up is sometimes useful. Encasing the joints in red-flannel bandages occasionally gives relief and is a convenient adjuvant. Massage is undoubtedly an efficient measure, though its effects are not always permanent. It is especially useful where there is atrophy of muscles, when it may be combined with electricity and passive motion.

Hot baths, which should always be taken at night, give temporary relief, but in my experience it is not permanent. Even the systematic baths at the various hot springs, as those of Virginia, Arkansas, St. Catharine's, and Banf, in Canada and elsewhere, which are undoubtedly serviceable, are apt to be followed by relapses. More satisfactory is permanent residence in a warm climate for those who can afford it.

See also treatment of rheumatoid arthritis.

JOINT-AFFECTIONS SIMULATING RHEUMATISM.

These include numerous joint inflammations of septic origin, such as occur in septicæmia, scarlet fever, diphtheria, and the like. They have all been appropriately referred to except gonorrhœal arthritis, erroneously called gonorrhœal rheumatism.

Gonorrhœal Arthritis.

Definition.—Gonorrhœal arthritis is a septic arthritis due to the gonococcus.

Morbid Anatomy and Pathology.—We do not often have an opportunity to study the morbid changes in the joints in this affection, since patients never die of this disease alone. Reasonably, however, we may expect a primary hyperæmia, and later the phenomena peculiar to inflammation in similar structures, viz., exudation into the joint cavity, including the out-wandering of white blood corpuscles, which are, however, rarely so numerous as to constitute pus. The periarthritic tissues are invaded by the exudate, and pus has been found in the sheaths of the tendons. There may be not only change in the shape but impairment in the motility of the joints. They may become stiff and swollen, as in chronic rheumatism.

Now as to the nature of this disease. Gonorrhœal rheumatism is an affection in which symptoms identical with those of rheumatism are more or less closely associated with gonorrhœa. These rheumatoid symptoms usually appear six to ten days after the discharge is seen. They may appear, however, much later—as much as four or five months or even a year after the discharge sets in, or during a chronic gleet. A lately married woman may acquire it from a husband who has gleet. There seems to be no relation between the severity of the symptoms and that of the original disease. The discharge generally continues with the onset of the joint symptoms, although it often abates, and may even cease altogether for a time. It may even recur with the disappearance of the rheumatic symptoms. It cannot be said that the true relation between these two very definite conditions is exactly known. The term gonorrhœal rheumatism is very generally recognized in all languages, and some English and American physicians do not hesitate to speak of it as a species of rheumatism. This is certainly erroneous. It is doubtless caused by the gonococcus, which, when once present, seems to be ineradicable. Witness its power to cause mycotic endocarditis. The simple non-purulent synovitis and arthritis may be the result of absorption of ptomaïnes furnished by the urethral pus.

These views are confirmed by modern bacteriological studies, which have found gonococci in the pus, in the tendinous sheaths, and more rarely in the non-purulent exudate.

Although the gonorrhœal poison is quite sufficient to produce the arthritis *de ipso*, it frequently happens that cold coöperates as an exciting or predisposing cause.

Symptoms.—A study of these admits a classification as made by the late R. P. Howard, of Montreal, into seven subdivisions :—

(1) The purely *arthralgic* form, *i. e.*, cases characterized by pain, but not much other evidence of local inflammation. Fever is also absent, although the condition is apt to be polyarthritic, wandering from joint to joint.

(2) *Rheumatic gonorrhœal* arthritis, resembling more than all other forms acute inflammatory rheumatism. In this division fever is added to the local symptoms of rheumatism, and polyarthritic involvement is also common. The fever, however, is less severe than would be expected from the severity of the other symptoms. The maximum temperature may be 102° F. (39° C.), more frequently it is less than this.

(3) *Acute gonorrhœal monarthritis*, in which one joint only is involved, with severe pain and swelling and moderate fever. It is the knee-joint which is most commonly attacked in this monarthritic variety. Next in order follow the ankle, shoulder, elbow, and wrist; any one of these is liable to be the seat of the trouble. Suppuration is rare.

(4) *Chronic gonorrhœal arthritis* without or with effusion (chronic hydro-arthrosis). Suppuration, though rare, does take place and pus is found in the joint cavity. In these cases, too, there is generally slight elevation of temperature.

(5) The *periarthritic* variety, including cases in which the periarthritic tissues are involved, including the capsule, ligaments, tendons, and adjacent fibrous structures. The periosteum is included among these, but the joint cavity itself is not affected.

(6) A variety which invades *fibrous tissue not connected with joints*, as the plantar fascia, the sclerotic coat of the eye and iris, the pericardium and endocardium.

(7) The *septicæmic form*, where in addition to the arthritis there is general sepsis and endocarditis. In this event there are the usual signs of blood invasion, high fever with or without chills and sweats.

Complications.—Isolated and even collected cases of endocarditis associated with gonorrhœal rheumatism have been reported by German and French physicians during the past thirty-eight years. Comparatively recently the studies of Gluzinski (1888) and R. L. MacDonnell * have settled the question in favor of a causal relation, the latter having found endocarditis present in four out of 27 cases of gonorrhœal arthritis, while Gluzinski collected 31 cases. They may reasonably be attributed to the action of the microörganisms on the valves. Malignant endocarditis may be thus caused.

Pericarditis and pleurisy similarly caused may complicate the disease, as may also iritis and sclerotitis.

Treatment.—This is not always satisfactory. Iodide of potassium is perhaps the drug most commonly found useful and its effect is increased when combined with the bichloride of mercury. It must, however, be associated with rest and local treatment. The former is sometimes better accomplished by the use of splints, the latter by blisters or iodine.

* "Gonorrheal Rheumatism," Amer. Jour. of the Med. Sciences, Jan., 1891.

General roborant treatment by tonics and good food may also be necessary. The ammoniated tincture of guaiac may also be used as in chronic rheumatism.

ARTHRITIS DEFORMANS.

SYNONYMS.—*Chronic Rheumatic Arthritis; Rheumatoid Arthritis; Osteo-arthritis; Rheumatic Joint.*

Definition.—A deforming disease of the joints regarded by most authorities as distinct from gout and rheumatism and characterized by destructive changes in the synovial membranes, cartilages and bone, and by bony outgrowths restricting the motion of the joint.

Historical.—Sydenham (1633), Musgrave (1763), Haller (1764), and de Sauvages (1768) allude to the characteristic changes in the bone due to arthritis deformans, but the first correct description was read by Landré Beauvais before the Paris Academy of Medicine, August 3, 1800, under the name "*Goutte Asthénique Primitive.*" Heberden, of England, in 1804 was, however, the first to recognize its true clinical position as something distinct from gout and rheumatism on the terminal finger joints, since known as Heberden's nodosities. Haygarth's paper on "Nodosity of the Joints," in 1805, describes the disease clinically, and he remarked upon the peculiar liability of the female sex. Robert Smith (1835), R. Adams, and Charcot (1868) contributed to its morbid anatomy; Trastour (1853), Vidal (1853), and Charcot to its clinical aspects. Charcot and Trastour regarded it as a variety of chronic rheumatism, as do Mitchell Bruce, of England, and Kahler, of Vienna, at the present day. H. W. Fuller (1852) and A. B. Garrod (1859) described the disease accurately as something distinct from gout and rheumatism, basing their views on the absence of the visceral complications of these two diseases. Jonathan Hutchinson and others at the present day still hold that the disease is the product of a blending of gout and rheumatism. J. K. Mitchell suggested a nervous origin for the multiple form, and this idea has been developed by Senator (1877) and R. Wichmann (1890) in Germany, W. M. Ord (1884) and Dyce Duckworth (1884) in England, L. Weber (1884) in America, and others, until at the present day this neuro-trophic view seems to be the most popular. Arbuthnot Lane (1891) attaches much importance to mechanical wear and tear in the production of the lesions. The most recent paper is the exhaustive one of Archibald E. Garrod, in Vol. II of the "Twentieth Century Practice of Medicine," 1895, from which the facts of this historical sketch are mainly drawn.

Etiology.—Although in the clinical features of its incipiency arthritis sometimes closely resembles the mild form of acute rheumatism, its independence of rheumatism and gout and their causes is generally conceded. Heredity, however, plays a likely, if not an important, rôle. Shock, worry, and grief have a closer relation. Females are much more liable to the disease than males, especially sterile women and those who have had uterine or ovarian disease. A. E. Garrod collected 500 cases, of which 411 were females and only 89 males. It is a disease said to be as common in the rich as the poor, though I have seen

many more of the latter, perhaps because of hospital practice. It more usually begins between the ages of twenty and thirty, but it may occur in children under twelve and as late as fifty. The beginning of the menstrual function in women is a favorite time for its incipiency.

Traumatism, often assigned by the subjects of the disease as a cause, has often no well-sustained relation, but must be allowed as a factor in monarthritic cases. Exposure, cold, and dietetic errors are ruled out at the present day as exciting causes, yet sometimes it seems impossible to exclude them. Certainly insufficient food seems to favor the disease. Uterine disease, worry, care, and actual illness have been mentioned.

Nature of the Disease.—The neuro-trophic theory of the disease suggested by the late J. K. Mitchell, and additionally supported by Charcot's studies of the arthropathies, certainly explains the phenomena better than any other. According to this view there must be allowed a lesion of the spinal cord, either primary or secondary to peripheral irritation the result of uterine or traumatic disease. The reasons in favor of it are, briefly, the symmetrical distribution of the articular dystrophies in the multiple form, the primary invasion of peripheral joints, and the centripetal extension of the disease; its etiology, the atrophy of muscles, the contractures, the occasional skin dystrophies manifested by pigmentation and local sweating. All of these are, however, offset by the absence of anatomical evidence of spinal-cord lesion, without which evidence the theory must still be regarded as not proven. The reflex origin of the cord lesion is especially supported by W. M. Ord.*

Morbid Anatomy.—All three of the structures which enter into the formation of the joint share in the process, but the changes probably begin in the cartilages. These consist in a proliferation of the cartilage cells, succeeded by fibrillation of the intercellular substance, which subsequently undergoes mucous degeneration, liquefaction, and absorption. Thus, the bone ends are laid bare. These subsequently become atrophied, smooth, and eburnated. The bone ends and joint cavities are alike distorted; concavities may become convexities, and convexities concavities. The edges of the cartilages where overlapped by synovial membranes thicken and form outgrowths, which subsequently ossify and become the osteophytes which contribute to the deformity of the bone, sometimes also forming rims or lips. The effect of the latter is to impair motion without producing actual anchylosis, except in very rare instances, which may include even vertebræ. The synovial membranes also become thickened and the fringes hypertrophied. Effusion is also sometimes present in the joints and in the bursæ. Fragments of cartilage may be attached to the tufts, or becoming detached they may lie loose in the joint. Muscular atrophy also makes a conspicuous part of the morbid changes.

Symptoms.—If the joint lesions be made the criterion of the presence of arthritis deformans any remaining difference in symptoms depends mainly upon the grouping and extent of these lesions. Hence it is more convenient to subdivide them into clinical varieties. Two such are easily made:—

* Trans. of the Clinical Society, 1879, XIII.

(1) Multiple arthritis deformans, including (*a*) Heberden's nodosities and (*b*) the progressive form, in which successive large joints are invaded in an acute or chronic manner.

(2) The monarthritis or partial form, in which one or two joints are alone attacked.

I. Multiple Arthritis Deformans.

(*a*) Heberden's Nodosities.—These are prominences or nodules which develop gradually on the sides and ends of the distal phalanges, especially of the fingers and sometimes also of the toes. Women are the most frequent subjects, and the development begins usually between the thirtieth and fortieth years and gradually increases with age, but varies also at times and seasons independently of this gradual increase. The pain and tenderness also vary, being usually worse when the hands become cold, especially when accidentally struck. At other times they are insensitive. These same nodosities occur in gout and by the laity are especially attributed to gout, but this is an error. They may be considered of probable gouty nature when they are aggravated by errors of diet. They are quite independent of the tophaceous deposits of gout, which are altogether absent in arthritis deformans. Persons in whom they are permanently present rarely have the large joints invaded, and, indeed, are said to have promise of long life. Subcutaneous nodules similar to those characteristic of acute rheumatism are also found in rheumatoid arthritis.

Fig. 56.—"Seal Fin" Deflexion of the Hand in a Case of Protracted Arthritis Deformans.

From a photograph with the Roentgen ray, showing also erosion and dislocation of joints.

(*b*) The Progressive Form.—This may be acute or chronic.

The *acute* form simulates in its beginning a mild form of rheumatic fever in young women of from twenty to thirty years, but it may occur also in children. There is swelling of the joints, tenderness, and fever. This may continue without material change for weeks, or may abate to recur with increased severity; on the whole, however, growing worse until the permanently enlarged and distorted state to be described is established.

In the *chronic* form the same changes develop more slowly, maintaining with remarkable constancy a symmetrical order of development,

the order of frequency being the hands, knees, feet, ankles, wrists, elbows, shoulders, jaws, cervical spine, hips, and dorsal spine. The most striking changes are seen in the knees, which become enlarged and so set that the legs are constantly flexed on the thighs and the thighs on the trunk. These *flexions* may be contributed to by *contractures*, which may, however, arise secondarily, subsequent to the flexion, or form *pari passu* with it. They are seen in the upper extremity as well as the lower, producing the "seal-fin" deflexion. The actual enlargement is exaggerated in appearance because of wasting of the adjacent muscles and thickening of the capsular ligament. Its surface becomes hard and shining. There may also be some effusion in the joint, though the condition has been called by the French *arthrite séche*. Motion grows more and more difficult until the joint is almost locked and grating and crackling attends attempt at motion. *Pain* varies greatly, at times very severe, at others it is quiescent, but is always excited by attempt at motion. *Weather* has its influences, diet rarely. *Tingling*, *numbness* of the hands and feet, and *local sweating* and *skin pigmentations* are not uncommon among the early symptoms and are regarded as trophic in origin. Day by day the patient becomes more helpless and, in the absence of fresh air, wan, weary, and anæmic. Fortunately, in many cases the fingers are unencumbered and the patient may be able to occupy himself or herself in some handiwork, such occupation serving to make more bearable a life of virtual imprisonment. The condition is singularly free from complications of all kinds.

II. The Partial or Monarthritic Form.

This affects men more frequently than women, and of these elderly men at the hip-joint, constituting the *morbus coxæ*, or chronic rheumatic arthritis of Adams, whose studies of this subject have added so much to our knowledge of the morbid anatomy. The other joints affected are the knee, the shoulder, and the vertebral articulations. It is often traced, rightly or wrongly, to injury. It is not always unassociated with disease in other joints, as there may be Heberden's nodosities or the disease may involve in a less degree the corresponding joint on the other side. *Wasting* of the muscles of the buttock and thigh is a conspicuous associated symptom. The *knee-jerk* is commonly increased on the affected side. An interesting though unusual symptom when the hip is affected is the presence of a large *cyst* at some distance from the joint, ascribed by W. Morrant Baker * to an outflow of the serum from an over-filled joint to a point at which it is restrained by the muscular and other tissues of the part.

Diagnosis.—This is rarely difficult. Arthritis deformans differs widely from gout in the total absence of tophaceous deposits, and from acute rheumatism in the absence of fever, though in the incipiency of the progressive multarticular form there is a certain resemblance to acute rheumatism, while in the more advanced stage, a stage in which the joint

* "St. Bartholomew's Hospital Reports," 1877, xiii, 1885, xxi.

lesions are conspicuous, it resembles also chronic articular rheumatism. This will be appreciated when it is remembered that some regard it only as a further development of chronic rheumatism. Time may be necessary to determine which it is. The atrophied shoulder of omo-neuritis also somewhat resembles the monarticular form, but the greater tenderness and painfulness as well as acuteness of this affection distinguish it. The arthropathies attending locomotor ataxia and syringomyelia are distinguished by the symptoms peculiar to them and the absence of osteophytes.

Treatment.—This is not generally promising and the usual remedies for rheumatism are of little avail. Yet treatment is by no means unavailing, especially if instituted early, and we may always hold out to the patient the hope of arrest at some stage. The principle of treatment consists in efforts to improve nutrition by means of good food and tonics, of which cod-liver oil, iron, iodine, and arsenic are the most efficient. A systematic course of these remedies, continuous except so far as judicious intermission may be necessary, will sometimes accomplish surprising results if instituted early and continued perseveringly. The iodide, either in the shape of the pill or syrup, is the best preparation of iron. A grain (0.066 gm.) of the former and 15 minims (one c. c.) of the latter are suitable doses three times a day. Massage is perhaps the single measure calculated to be of most use, and if cod-liver oil and iron be used in connection with it further benefit may be expected. Disappointing as the treatment often is, in a few cases surprising results may be obtained. One of the most serious drawbacks in certain cases is the difficulty in securing outdoor life and the advantage of exercise. One of the objects of massage must be to substitute the latter, while every possible effort should be made to have the patient in the open air as much as possible, and where his means will permit it to take advantage of residence in warm but dry climates. It is very important to avoid the use of anodynes altogether, if possible. The relief afforded by them is but temporary, they militate against the effort at securing an improved nutrition, and, above all, there is a danger of the morphine habit. Simple support by splints is sometimes a comfort to patients. The treatment by hydrotherapy, as carried out at Aix-les-Bains and Aix-la-Chapelle, in Europe, undoubtedly affords temporary relief. The same may be said of the treatment at the Hot Springs of Arkansas and Virginia in this country and at St. Catharine's in Canada. While general steam baths are contraindicated by reason of their debilitating effect, local vapor baths applied to separate limbs or portions of limbs by a specially constructed apparatus are sometimes useful. Some mention should be made of the electric bath. Like so many remedies, it is often of service at first, but I have yet to see any permanent results. It may, however, be tried in cases which have resisted other treatment. The bath should be given under intelligent supervision and the current should be weak at first.

The bowels should receive close attention, the body should be frequently bathed, preferably in warm water, and all measures desirable to secure the most perfect personal hygiene practised. This is another of the few diseases in which an abundance of good, nourishing food is neces-

sary. This is the more important when we remember that many cases originate among the poor and badly fed. The use of wine, porter, stout, and ale should be encouraged.

Having a highly intelligent friend afflicted for many years with rheumatoid arthritis whose circumstances permitted him to seek every available means of relief, I asked him to give me the benefit of his experience. He replied in a letter so sensible, and which appeared to me to contain the truth so well condensed, that I append it with the belief that it will be useful:—

"I received no benefit at any of the following places: Aix-les-Bains,* Bourboule, Royat, Wiesbaden, Homburg,* Carlsbad,* Gastein,* Wiltbad,* Acqui,* Montesumano, Richfield, N. Y., Green Sulphur, Fla. In all the places marked with an * there were people to all appearances affected similarly to myself who did receive benefit, and at the same time there were others also so afflicted who were made worse. In short, I am convinced from my experience that no physician can definitely settle what spring will be of the greatest service to any patient, and that the only chance of finding this out will be for him to select those which he thinks will be of the most service and let the patient try them in turn. In this way the patient will perhaps find one which will either materially alleviate or absolutely cure him. I found that the mud baths of Battaglia, Italy, did me the most good, but they, at the same time, nearly cost me my life. I am also convinced that rheumatoid arthritis cannot be successfully treated by giving the same remedies to different people suffering with it. There must be variations in our general make-up as there are in our features. In my judgment, therefore, each patient must be a law to himself. He must find out for himself by judicious experiments the food and drink that he can best assimilate and confine himself to them, even if the usual practice would indicate that they ought to injure him. I also think that no patient ought to take any hot baths anywhere without ample nourishment of some kind, for a depleted diet and terrific perspiration have done me great harm, for it was like 'burning my candle at both ends.' My experience has convinced me that careful attention to diet, out-door exercise, freedom from anxiety, and good digestion are much more serviceable than any springs in the world."

GOUT.

SYNONYM.—*Podagra.*

Definition.—An acute and chronic constitutional affection, due to an abnormal accumulation of uric acid in the blood and tissues, causing various symptoms, of which arthritis is the most distinctive and significant.

Historical.—With the exception of the classic work of Thomas Sydenham, "*Tractatus de podogrâ et hydrope*," published in 1683, and based upon his own case—he having been himself a victim for forty years—the history of our knowledge of gout is the history of the development of its pathology, which begins with Wollaston's discovery in 1797 that

the deposits in the joints and vicinity in this disease are largely composed of uric acid. Previous to this time a humoral view of the nature of gout was accepted, though without tangible foundation. In 1784 Cullen promulgated the nervous theory of gout which was adopted from G. E. Stahl. Cullen's theory did not entirely exclude the humoralistic, since he allowed that a morbid substance appeared in the blood of some gouty persons after a time, but as the effect of the disease rather than its cause. (See also Pathology.)

Etiology.—The tendency to gout is more frequently inherited, but is also acquired. Between 50 and 60 per cent. of all cases of gout can be traced to ancestry, parents or grandparents. More men are gouty than women, and it is the male line through which the tendency is most frequently transmitted. It is not usually manifested until after forty years of age, sometimes later, but the signs which are almost sure to eventuate in gout may show themselves before the twelfth year. While over-eating, especially of meats, and intemperate drinking, associated with the luxurious habits which grow out of the possession of wealth, are the most frequent causes of acquired gout, these last are by no means essential. Sir Dyce Duckworth's studies of gout in what is probably the richest field in the world, London, go to show that many of the peasantry of Ireland, among whom gout is unknown, became gouty after having lived for a time in London. On the other hand, not every person who inherits a tendency to gout becomes gouty, since the fostering causes above mentioned may be wanting. In others this tendency is so great as not even to require the favoring condition.

While alcohol is an acknowledged cause of gout, it has been observed that something depends on the shape in which it is presented. Malt liquors, especially the "heavy" English ales and beers, strong in alcohol, are more active in the production of gout than the lighter beers consumed in Germany and this country. The same is true of the strong and the sweet wines of which port and sherry are the type, while pure whiskey is less harmful. The glycogenic substances in the malts and sweet wines are probably responsible as sources of acid fermentation products in the stomach which, after absorption, reduce the alkalinity of the blood and impair the solvent power of uric acid. An interesting exciting cause of gout is lead poisoning. This is met particularly in England and especially in London, as pointed out by Sir Alfred Garrod in 1854. It is, however, rare in other parts of Great Britain and Ireland, and is growing less in London. For in 1870, according to Garrod, 33 per cent. of people who suffered from gout had been poisoned by lead, while Sir Dyce Duckworth, up to 1890, found only 18 per cent. in hospital cases. It is a rare cause also in France and Germany. It may be, as suggested by Alexander Haig, that the effect of plumbism is to diminish the alkalinity of the blood. In this country the combination is comparatively rare.

Pathology.—It cannot be claimed that the pathology of gout is thoroughly established, although so many facts bearing on it are well determined. In this respect it resembles its sometimes congener, diabetes mellitus. It is commonly admitted that uric acid is in some way causative. Whether, however, the uric acid thus responsible is the result of

increased formation or diminished excretion, or both, is not so generally acknowledged. As early as 1797 Wollaston showed that the deposits in the joints and vicinity were a compound of uric acid. About 1838 Sir Henry Holland suggested that the peccant material was uric acid in the blood. This was, indeed, suspected by others, but it was not until 1848 that Sir Alfred Garrod furnished the first demonstrative evidence of such accumulation by his well-known thread test.*

Alexander Haig † claims that there is "almost never" an excessive formation of uric acid at any time, and that its accumulation in the blood and body is generally due to retention or failure of excretion; that uric acid is, on the whole, continuously formed in the proportion of one to 33 of urea. In certain states of the blood, consisting essentially in increased alkalinity, uric acid is held in solution in larger quantity, constituting uric-acidæmia. At such times, too, it is eliminated in increased quantity in the urine, by which it is also readily held in solution because of the alkalinity of this secretion. In opposite states of the blood the uric acid is driven out of this fluid and deposited in the tissues of the joints. Haig holds, also, that these opposite conditions, which are fluctuations in secretion only, can be artificially produced by drugs, food, temperature, and other conditions influencing the reaction of the blood. Thus, alkalies, alkaline foods, and warm weather favor the former, *i. e.*, solution, while acids and cold weather favor the latter, and it is under influences like these that uric acid in the shape of urates is stored up in the body. He further says that the blood never becomes loaded with uric acid except as the result of previous imperfect excretion, and such imperfect excretion or retention is sufficient to account for the largest quantities he has ever seen in the human body, and that it is not necessary to suppose excessive formation in explanation. Further, that he does not assert that such formation never occurs, only that he has never met any conclusive proof of its occurrence, while all the phenomena of disease can be explained without postulating the excessive formation of a single grain.

The ultimate result is, however, the same. Whether it be from diminished excretion or increased formation, or both, there is an accumulation of uric acid in the blood, which is responsible, first, for certain premonitory symptoms of gout, and, second, for certain local symptoms. The latter are of an inflammatory nature, and consist essentially in pain, swelling, and redness of the joints, preferably of the smaller ones, and especially of the metatarso-phalangeal articulations of the great toe; more frequently, perhaps, of the left great toe.

As to the relation of the uric-acid compounds to the local inflammation, it is scarcely necessary to say that uric acid does not exist as such in the blood, even in pathological conditions. The normal urates, as originally shown by Bence Jones, and recently confirmed by Sir William

* This is done as follows: To two drachms (eight gm.) of serum from a blister add five or six minims (0.33 to 0.396 gm.) of acetic acid in a watch glass. A thread immersed in this will show in a few hours an incrustation of uric acid. This reaction is not confined to gout. It occurs in chlorosis and leucæmia.

† "Uric Acid as a Factor in the Causation of Disease," by Alexander Haig. London, 1892.

Roberts, are quadri-urates. Such is the composition of the urate-sediments in urine. In the pathological state these are converted into the less soluble biurates which make up the local deposits. It has all along been considered that these deposits are the direct cause of the gouty inflammations. Haig, as the result of his recent researches, reasserts this view in the following graphic language :—*

"Then I also noticed that in curing a headache by giving an acid to diminish the excretion of uric acid, I always produced a certain amount of pricking and shooting pain in my joints (generally in those which had been most used on the day in question), and it naturally occurred to me that the uric acid was held back in these joints and produced the pains. The uric acid which had failed to appear in the urine must have gone somewhere. What more natural than to suppose that it had been retained in the joints (where in gout it is found), and that the pricking pains were the evidence of its presence? Then, on turning to Sir A. Garrod, I find that he had described precisely similar joint pains as occurring in gouty subjects immediately after the ingestion of beer or wine, and a very little investigation sufficed to prove that all wines and beers are strongly acid, so that a very simple explanation could be given of the facts."

E. Pfeiffer holds that both gout and gravel are due to the fact that in these conditions uric acid is produced and excreted in a form difficult of solution. As such patients excrete on an average less uric acid, the blood becomes overcharged with it and the uric acid salts are deposited in different parts of the body unnoticed and without symptoms of irritation. Then, according to Pfeiffer, the attacks of gout occur when from any cause the alkalinity of the blood becomes so great as to dissolve the deposited urates, which then give rise to irritation and inflammation. Not the precipitated uric acid, therefore, but the dissolved uric acid, according to this view, must be regarded as the irritating agent; while an acute attack is the result of a re-solution in the blood of previously-deposited uric acid, the cause of which is an increased alkalescence of the blood and body juices, while a deposit is the result of diminished alkalescence. Pfeiffer, in further support of this view, calls attention to the fact that the most recent chemical analyses by Lecorché, Ebstein, and himself show that the excretion of uric acid *during* an attack of gout is increased and not diminished, as taught by Garrod. Pfeiffer further cites in support of this view the fact that acids, especially salicylic, in large doses promptly relieve the pain in gout, while it is increased by the administration of alkalies.

The most recent studies on this subject—those of Sir William Roberts †—reaffirm the older view, that the "mechanical theory offers a natural and complete explanation. The crystalline urates precipitated in the cartilaginous and fibrous structures of the joints necessarily act as foreign bodies; they excite irritation, clog the lymph-channels, exercise pressure on the tissue elements and impede their nutritive operations.

* *Op. cit.*, p. 2.

† "Uric Acid, Gravel, and Gout," 1893, p. 111.

These effects sufficiently account for the inflammation, pain, and swelling which ensue, and explain the remoter degenerative changes which follow." While admitting that old tophaceous deposits are not always painful, my experience is that of Sir William Roberts and I believe of most clinicians, that coincident with a fresh deposit of sodium biurate there is always pain, while the salicylates or alkalies are, in my experience, the promptest means to relieve the pain of gout.

Of modern authorities, Ebstein* is the most eminent who ascribes the excess of uric acid in the blood to an abnormal formation of this substance in the body, in parts also which do not normally produce it, as bone, marrow, and cartilage. The lymph and blood becoming surcharged, a balance is commonly restored by increased renal excretion or perhaps by decomposition of the uric acid. When, however, from any cause the lymph moves slowly, the so-called premonitory *symptoms of gout occur, viz., a "tired feeling," vague pains, etc. If an actual stasis* occurs, the acute attack appears. This concentrated solution of uric acid acts like a chemical poison on certain tissues, giving rise to necrobiotic changes, and when these reach a certain stage characterized by an acid reaction the uric acid is deposited in them as sodium biurate. If this stage is not reached, the local symptoms disappear and the joints return to their natural state. So much of Ebstein's view as makes a previously degenerated state of the tissues a necessary condition of the deposit of uric acid is commonly admitted by modern authorities as represented by Ord and Dyce Duckworth.

The nervous theory of gout was promulgated by Cullen in 1873 as a mixed neuro-humeral rather than purely neural. Of modern authors, Sir Dyce Duckworth has taken the most pains to elaborate and establish the view that gout is a neuro-humeral disease, the sum of which may be said to be :—

That the diseased states recognized as of undoubted gouty nature are primarily dependent on a functional disorder of the nervous system, and that thus gout is a primary neurosis. That this, like others, may be inherited or acquired, modified, or even repressed, and may be associated with other neuroses. A large part of the symptoms known as gouty is due to perverted relations of uric acid and sodium salts caused by the neurosis.

Morbid Anatomy.—As will be further evident in treating the symptomatology of gout, there is scarcely a tissue which may not be affected by it, but the morbid conditions which are more distinctive are, first, the characteristic inflamed great toe of acute gout, the true podagra. The angry, swollen, dark red, or mottled appearance of such a toe once seen is not forgotten. Similar though less striking changes are sometimes seen in the metacarpo-phalangeal articulation of the thumb.

The superficial changes in chronic gout are less distinctive and are often not different from those of chronic rheumatism. But wherever uratic deposits are present in the tissues, there, by universal consent, is gout. They are found mostly in joints and the parts around them :

* "*Beiträge zur Lehre von der Harnruhr-Diathese*," 1891.

first, the cartilages of the movable joints, then the ligaments, tendons, bursæ, and finally the connective tissue and skin, this being the order of feebleness in vascularity and nutritive activity. Frequent situations are the digital joints and cartilages of the ear, more rare the cartilages of the nose, the vocal cords, the cornea, kidneys, marrow of bone, and expectoration. Cartilages impregnated with urates present the appearance of being smeared with whitewash or white paint, and when preserved in pure alcohol maintain it for a long time. Minutely examined, cartilages are infiltrated on the peripheral surface, more rarely beneath, with acicular crystals of sodium urate. Rarely they are found in the bone under the cartilage. The cartilage cells are for the most part free, and after the urates are dissolved out the tissue appears natural or slightly granular. The tophaceous deposits are the best known and most characteristic lesion of gout. About the digital joints, especially the knuckles of the hands, they sometimes ulcerate through the skin, making the oft-repeated story of the gouty subject who chalked his games of whist with his knuckles a not unlikely one. The deposits are often associated with deflection of the fingers to the ulnar side, "seal-fin" type of hands, and of the toes outward, a late symptom not confined to gout—in fact, more common in rheumatoid arthritis. It is due to stronger action of the abductor muscles.

The nodular gouty tophi are not to be confounded with Heberden's nodosities, also characteristic of rheumatoid arthritis. Indeed, Heberden himself denied their gouty nature, though they do occur in gout also. Their gouty nature must be determined by other symptoms more essential to gout. They are irritative nodular outgrowths on the distal ends of the phalangeal bones. They vary in prominence and tenderness at different times, being worse especially after errors of diet, but on the whole they slowly increase with the age of the patient and the persistence of other symptoms.

Of undoubted gouty nature are regarded the little vesicles called "crab's-eye" cysts over the nodosities. To these are to be added certain exostoses and ecchondroses, or "lippings," beneath the synovial membrane, at the edges of the cartilages, and round the heads of the phalanges and even larger bones, like the femur, patella, and tibia.

Changes in the internal organs are mostly confined to the kidneys and vascular system. Uratic deposits have been alluded to. They are not constant and are found usually in the interlobular tissue toward the apices, but also more rarely in the tubules. Ultimately the gouty subject acquires an interstitial nephritis, the well-known gouty kidney. Arterio-sclerosis is almost always present. The heart is hypertrophied in its left ventricle. There may be deposits of urate of sodium on its valves. Changes in the lungs are mainly confined to emphysema, which is found in many cases of advanced standing.

Symptoms.—*Of Typical Acute Gout.*—Persons subject to attacks of gout sometimes have premonitory symptoms suggesting the approach of an attack. These vary with the individual and are significant only in each case. They may be headache, neuralgia, any one of the numerous manifestations of deranged digestion, irregularity of the heart's

action, palpitation, high tension of the pulse, depression of spirit, drowsiness, a disposition to yawn, a tired feeling—in fact, any symptom which the patient learns to associate with the attack. Attacks are apparently also invited or determined by anything which lowers the vitality of the patient. On the other hand, a supper with wine or a single glass of champagne will often produce an attack.

The first true symptom of the typical attack is *articular pain, commonly in the great toe*, at the metacarpo-phalangeal joint, and with its appearance the premonitory symptoms usually pass away. The pain is extreme, sharp, shooting, and sudden, often awakening in the middle of the night a patient who has gone to bed in apparent good health and least expecting an attack. With this pain are the *swelling*, *heat*, and *discoloration* already described under morbid anatomy. Rarely the attack begins with a *slight chill*. On the other hand, there may be pain without heat, redness, or swelling, and all the typical local anatomical features of an attack without pain. In some instances the attack develops more slowly. At times the first attack is so slightly distinctive that it is assumed to be something much more trifling, as rheumatism or the result of some slight injury, while the personal peculiarities, natural or acquired, always more or less influence the symptoms. After the outburst at night the extreme pain diminishes as morning advances, but it may repeat itself the next night, and this goes on for four, five, or six days, when the attack terminates.

Some fever usually accompanies the onset of acute gout. The temperature promptly rises to 100° F. (37.8° C.), but does not far exceed it, 102° F. (38.9° C.) being the usual maximum attained. As in other acute diseases, the temperature is higher in the evening. The local temperature, notwithstanding the sensation of heat, is five or six degrees below that of the axilla at the same time. The attack terminates with desquamation of the epidermis over the inflamed joint.

Changes in the urine are almost distinctive. It is scanty, acid, high-colored, and of high specific gravity. It deposits urates on standing and often contains a small quantity of albumin. It contains sometimes sugar in small and even decided quantity, but I am certain that at others the reaction of uric acid on copper oxide is mistaken for that of glucose. During or preceding an attack the uric acid excreted may be diminished, later it may be increased, but as elsewhere stated a relative increase in uric acid is often mistaken for an absolute increase in the twenty-four hours' amount. The probabilities are that these changes are inconstant.

A recognized symptom of acute gout, and sometimes the only one, is *pharyngitis*, and now the term "gouty sore throat" is one in common use, though it is doubtless also often carelessly used. There seems no way of distinguishing it locally from other forms of sore throat in which there is no decided swelling.

Gout is said to be *retrocedent* or *metastatic* when it disappears suddenly from its external site and there are substituted for the external symptoms derangements of some internal organ, especially the heart or stomach or brain or urinary bladder. In the first there appear cardiac symptoms of varying severity, including pain, shortness of breath, and

irregularity in the heart's action; in the second, gastro-intestinal pain, a sinking sensation, vomiting or diarrhœa, often associated with intense mental excitement or depression; in the third, meningeal symptoms, and in the fourth, cystitis. More rare events are gouty orchitis, parotitis, and urticaria, or other fugitive skin affections. Metastasis is more prone to occur in atonic cases. Sudden death has supervened in some instances, but post-mortem lesions of a definite kind seem to be wanting, at least lesions which can be held responsible for the symptoms.

Of Irregular or Atypical Gout.—This includes a set of symptoms not so distinctive in themselves as peculiar in this, that they occur only in persons who have been gouty or have a decided hereditary tendency thereto. These conditions being fulfilled, there is scarcely any superficial or visceral symptom which may not be of gouty origin. But among them may be named cutaneous eruptions, gastro-intestinal disorders, various forms of headache and neuralgia, hot and itching palms and soles, especially at night, a similar condition of the eyeballs, lumbago and other muscular pains, arterio-capillary fibrosis and its consequences, iritis, bronchitis, pericarditis, cystitis with hemorrhage into the bladder, and others. All that was said of the general physical and chemical characters of urine in acute and chronic gout may be true of it in irregular gout. Retrocedent gout, already described, is really a form of irregular gout.

Of Chronic Gout.—As repeated attacks of gout occur and the patient grows older, there gradually accumulate the morbid changes described under morbid anatomy as more or less characteristic—the joints deformed by tophaceous and other deposits, the lipping, the seal-fin hand, the renal and arterio-vascular changes, and the interstitial nephritis, etc. The urine now is increased, lighter hued, contains albumin, and a few hyaline and granular casts.

Treatment.

The treatment of gout easily divides itself into two parts: first, that of the paroxysm or acute attack; second, that of the gouty diathesis or tendency which eventuates in the acute attack. Taking up the second first, it is plain that if uric acid is its cause, whatever diminishes the amount of uric acid in the economy must tend to relieve gout. It is plain, also, that we may diminish uric acid in two ways: first, by confining the gouty person to such food as produces a minimum of uric acid; second, by administering such medicines as will promote its solution and elimination. The first of these constitutes, in the main, the *dietetic* treatment, the second the *medicinal.*

(1) *The Dietetic Treatment.*—This is by far the most efficient of the treatments of gout, without which all else is only palliation. It consists essentially in the elimination as far as possible from the dietary of all nitrogenous or albuminous principles, being those whose complete combustion results in urea and incomplete in uric acid. I say as far as possible, for it is practically impossible to eliminate them altogether. The foods which are the type of this class should, however, be altogether omitted.

Such are the meats of the butcher-shops, the albumen of eggs, and the cheeses. The first include beef, veal, mutton, lamb, and pork, whether salt or fresh, and for the most part fish. As to cheeses, as a half-pound of cheese contains almost as much nitrogenous matter as a pound of beef,—27 per cent. when made of the whole milk, and 28 per cent. when made of skim-milk,—it is plainly contraindicated. If we consider only the edible parts of beef,—*i. e.*, meat deprived of the refuse represented by bones, skin, and shells,—it contains, according to its source, 17 to 23 per cent. of proteids; mutton from 15 to 18 per cent. Of fish, flounder contains 13.8 per cent.; mackerel, 18 per cent.; halibut, 15 per cent., and salmon, 21 per cent., or quite as much as beef and more than mutton. Salt codfish contains 15 per cent.; smoked herring, 20 per cent., and canned sardines, 24 per cent. Poultry contains 14 to 15 per cent. of albuminates, and game 22 per cent. The hen's egg, including albumin and fat, contains 13.7 per cent. of protein, whence it is plain that the yellow of eggs contains a very small quantity and becomes a suitable food.

On the other hand, milk contains but three per cent. to four per cent. of protein; butter, one per cent.; and oleomargarine, 0.6 per cent. The fat oyster contains eight per cent., the lean, 4.2 per cent., and the lobster, 5.5 per cent.; other fish than the above-mentioned, five to ten per cent.

Of vegetable foods, wheat bread contains 8.9 per cent. of protein; wheat flour, 11 per cent., and Graham flour, 11.7 per cent.; rye bread, 6.7 per cent.; buckwheat flour, the same; corn (maize), nine per cent.; rice, 7.4 per cent.; sugar, 0.3 per cent.; potatoes, two per cent.; sweet potatoes, 1.5 per cent.; turnips and carrots, one per cent.; cabbage, 1.9 per cent.; melons, one per cent.; apples and pears, 0.4 per cent., and bananas, two per cent. Again, beans contain 23.2 per cent. and oatmeal 12 to 15 per cent., large proportions of proteids.

Thus, the typical foods permissible from the standpoint of composition are milk, butter, fruits, and the succulent vegetables, except beans and oatmeal. To these oysters and lobster may be added, moderately; fish, except those named as containing a large amount of protein, and, where extreme rigidity is not required, poultry in moderate amount; but all butcher's meat should be strictly forbidden.

It is usual, also, to interdict the use of carbo-hydrates,—*i. e.*, starches and sugars,—as well as the hydrocarbons,—fats and oils,—but I have never been able to see any reason for this. There is absolutely none from the standpoint of chemical composition, since they are totally without nitrogen, and, so far as my own experience goes, no cause from the clinical standpoint. Only in the event of their producing indigestion and fermentation, with the generation of acids, can they become a cause of gout, and then only, I should say, an exciting cause. I am in the habit, therefore, of permitting the use of rice, potatoes, and other farinacea, and, to a reasonable extent, sugar. I am glad to be able to say that I am sustained in this view by Sir William Roberts, who, in the brochure quoted on p. 737, says also: "The most trustworthy experiments indicate that fat, starch, and sugar have not the least direct influence on the

production of uric acid; but as the free consumption of these articles naturally operates to restrict the intake of nitrogenous food, their use has indirectly the effect of diminishing the average production of uric acid." Basing his conclusions upon experiments with solutions of blood-serum impregnated with common salt (0.1 per cent.), in which he found the precipitation of crystalline biurate always appreciably hastened, Sir William Roberts for some years past has directed the gouty to restrict as far as possible the use of common salt with meals. On the other hand, he recommends that the subjects of uric-acid gravel should be advised to take habitually with their meals as much culinary salt as their palates will tolerate.

There are, however, other sorts of ingesta, also entirely or almost free from nitrogen, acknowledged to be both a predisposing and exciting cause of gout,—namely, malt liquors and wines. These are composed of water, alcohol, carbohydrates, and a trace of mineral matters, but no nitrogen. It is not easy at first thought to understand why these substances should be harmful. Experience, however, shows that the stronger wines, such as port, Madeira, and sherry, by their continued use, are very likely to produce gout, while the lighter wines,—the clarets, hocks, and Moselle wines,—if taken in moderation, rarely produce it. After these, stout, porter, and the strong ales induce gout. Even lager beer, which contains but three per cent. of alcohol, is capable of acting similarly, and I know many men who have been forced to give up this beverage because of this effect. Cider and perry least of all beverages predispose to gout. On the other hand, distilled spirits, especially whiskey, are almost entirely without effect in producing gout. Why is this? Apparently, the amount of alcohol is not the measure of the effect, for whiskey, gin, brandy, and rum all contain more alcohol than any of the wines alluded to. If reference is made to the wines most apt to produce gout, it will be found that they are those which contain a considerable quantity of both sugar and alcohol. Such are port, sherry, and Madeira, all of which contain more than 15 per cent. of alcohol and much sugar; also sweet champagnes containing 11 per cent. of alcohol. On the other hand, some very sweet wines, as Tokay, Malaga, and the higher Sauternes, which contain much sugar, produce gout less rapidly. It would seem that those liquors which contain alcohol in combination with other substances, especially sugar, are potent gout-producers, especially where they excite indigestion, probably by restricting elimination rather than producing more uric acid.

As to the acidity of alcoholic drinks, their influence is pretty clearly as exciting causes. In this way act the beers, in which both alcohol and sugar are present in small amount, but which are highly acid. An explanation of this fact is less ready from the standpoint that the acute attack of gout is due to a resorption of the deposited uric acid by an alkaline blood, than on the supposition that the attack is due to the irritative effect of uric acid deposited in the joints, because of the diminished alkalinity of the blood induced by the absorbed acid. Whatever be the explanation, few facts in the clinical history of gout are better established than that the ingestion of acid is an exciting cause of attacks.

In the same way act acid fruits, such as strawberries, acid oranges, and lemons. On the other hand, to such influence I have known the most divergent response. Thus, a gouty patient of my own could bring on an attack by drinking a single glass of lemonade, while a gouty friend would drink a pitcher of lemonade at dinner without any effect whatever. It is to be remembered that the otherwise harmful effects of the strong distilled spirits, such as are well borne in gout, are no less serious in gouty subjects than in others, and are often induced by the careless prescription of whiskey as less harmful than wines in gout.

A most valuable adjuvant to the dietetic treatment are the natural mineral waters. The waters which have heretofore received almost universal approval are the alkaline waters, although those possessing purgative properties also enjoy much reputation. In America, however, few alkaline waters are native, while those which are, are so far inferior to the foreign waters that it is practically impossible to fulfil the requirements with them, while the costliness of the foreign waters contributes a very serious obstacle to their general use. Most of the native waters which have been employed and highly vaunted by their owners are of the kind known as negative waters; that is, they have no mineral ingredients in any quantity to justify their classification in any of the four principal varieties of mineral waters, viz., the alkaline, the saline, the purgative, or sulphurous, or on which to base any therapeutic results except by their diluent effect. At the same time it has been noted that these waters are not without effect in relieving gouty symptoms. Reasoning from these facts, we may prescribe the native negative waters, such as are accessible to the patient, or distilled water, with this end in view—the simple diluent and solvent effect which comes from an increased proportion of water. The further propriety of such a course is found in the fact that gouty and lithæmic patients are often small water drinkers, never drinking water between meals and very little at meals. To such, eight ounces of water ordered on rising, between meals, and at bedtime will often clear off a dark-hued urine of high specific gravity and substitute a light-hued, clear urine, without any sediment.

The actual mineral waters which have acquired the greatest reputation in the treatment of gout are those of which sodium bicarbonate is the chief ingredient, to which the calcium bicarbonate is regarded a valuable adjuvant. Such are the alkaline waters of Vals and Vichy in France, Evian-les-Bains in Switzerland, Neuenahr and Fachingen in Prussia, Contrexville and Vittel in the Vosges, France, and Dax in France. Other waters possessed of reputation in the treatment of gout, in which the quantity of alkaline bicarbonate is smaller, owe it to their combined alkaline and aperient properties, chiefly due to sodium sulphate and magnesium sulphate, and belong to the second category of remedies for the treatment of gout. Such are the alkaline and saline waters of Carlsbad and Marienbad in Bohemia, Kronthal in Nassau, and Brides-les-Bains in Savoy. Then there are the saline waters represented by Baden-Baden, Ems, Homburg, Kissingen, Wiesbaden, and our own Saratoga waters. In saline waters we are much more fortunate in this country, the Saratoga waters furnishing all that can be desired. Finally, there are the

bitter acidulated and purgative waters—Hunyadi Jaños, Friedrichshalle and Rakoczy in Hungary, Pülna in Bohemia, and Rubinat in Spain—rarely resorted to for gout, but useful as eliminating agents. Among the weaker aperient waters are those of Bedford Springs, Pa., in this country.

The use of these mineral waters is especially indicated in a continuous manner between the attacks with a view to averting them. Especially useful are the thermal waters in the chronic arthritic complications, in which their internal use is combined with bathing. In this connection may be mentioned Carlsbad and Marienbad, where also the mud-baths are employed, Baden-Baden, Ems, Wiesbaden, Hammòn R'Irha in Algeria, available in winter, Plombières in the Vosges, and Dax in France. Homburg and Kissingen are also resorted to for their baths, although the waters are cold.

Sulphurous waters also have some reputation in gout. Especially is this the case with the waters of Aix-la-Chapelle in Rhenish Prussia and Aix-les-Bains in Savoy, Harrogate in England, Richfield Springs, Sharon, and St. Catharine's in America. In all these places the bath treatment is an important adjuvant. America is also more fortunate in sulphur waters.

The second category of remedies—the aperients—are decidedly useful in gout, both as eliminators and to prepare the way for the absorption and prompt action of the alkaline bicarbonates. They are not, however, used at the present day as freely as a century ago, and they are commonly reserved for the acute attack.

Hygienic measures are also of importance in the treatment of gout. The patient should bathe daily, using the cool bath in the morning or the warm in the evening on retiring as experience may determine to be the best. The skin should be thoroughly groomed and daily exercise should be practised, an open-air, out-door life being desirable whenever possible.

(2) *The Medicinal Treatment and the Treatment of the Acute Attack.*—As a rule, the use of medicines is reserved for the acute attack. From the earliest history of the disease practice has recognized two classes of remedies in the treatment of gout,—alkalies and purgatives,—the object of both being to eliminate the offender, the first by producing soluble combinations which pass off readily by the kidneys, the second to carry it off by the bowels. It is plain that a combination of the two principles might be expected to be more efficient than either one alone.

First, as to alkalies and alkaline combinations. My experience places the salicylate of sodium easily at the top, and while it is not so rapid in its effect in relieving the pain of an acute attack of gout as it is in rheumatism, it is nevertheless an invaluable remedy, excelling all others. During an attack it should be given in doses as large as can be borne. As a rule, adult men easily bear 15 grains (one gm.) four times a day, or ten grains (0.65 gm.) may be administered every two hours. Even larger doses may be given with advantage if borne by the stomach. With relief to the acute symptoms, the dose should be reduced; but, as in rheumatism, the remedy should not be discontinued, and between at-

tacks smaller doses should be kept up for some time. These, however, may be substituted by the natural mineral waters to be presently alluded to.

Among the eliminating remedies is the time-honored colchicum, a drug which is of undoubted value in gout, but which, in my experience, must yield the palm to salicylic acid. For a long time its action was inexplicable, and it came to be spoken of as a specific in gout, as quinine in chills and mercury in syphilis. Modern studies have, apparently, solved this problem. Professor Rutherford has shown that it is one of the most powerful cholagogues known. This, taken in connection with what we now know of the office of the liver in urea formation, simplifies very much the solution of the problem. This explains, too, why colchicum produces its sedative and anæsthetic effect without necessarily producing purgation. Indeed, some, as Sir Alfred Garrod, consider that its effects are best attained without purgation, and Garrod says that if cathartic action is required, it is better to combine some aperient with the colchicum, as when much purging and vomiting result from colchicum, nervous and vascular depression follow. I confess I like to secure a little action on the bowels by increasing the dose gradually, and it is not necessary to produce either violent purging or vomiting. Whatever its mode of action it sometimes acts in the most magical manner in relieving pain. The preparation commonly used is the wine. In this country the wine of the seeds is no longer officinal, so that if the wine is ordered that of the root is dispensed. This is more powerful than the wine of the seeds. The dose of the latter is from ½ to one drachm or 1 ½ drachms (two c.c. to four c.c. or six c. c.) every three hours during the attack, but of the root 15 to 30 minims (one to two c. c.), reducing the dose when nausea or purgation ensues. The acetic extract of colchicum was a favorite preparation of the older physicians, especially Scudamore, who introduced it, and who considered its action milder than any other form. It is still sometimes used, and has the advantage that it may be put into pill form. Its dose is one to two grains (0.065 to 0.13 gm.). Colchicene, the active principle of colchicum, is also employed. Its dose is $\frac{1}{50}$ grain (0.0013 gm.). The same dose may be employed hypodermically. The fluid extract of colchicum may be administered in doses of two to six minims (0.12 to 0.30 c.c.). The aperients commonly used in gout are the salines, of which the magnesium sulphate is the favorite. Sodium sulphate is also used, and it is the constituent of the most actively purgative mineral waters—the Hunyadi Jaños, Rakoczy, and Friedrichshalle mineral waters—already mentioned, now largely used instead. A favorite combination of the older physicians was magnesium sulphate two drachms, magnesium carbonate a scruple, suspended in an ounce of cinnamon water, two or three times a day until active purgation results. These two substances may be combined with colchicum, and with it make one of the forms of Scudamore's mixture, a popular gout medicine.

Colocynth is also employed as an aperient in gout, and advantage has been taken of this fact in the preparation of the secret remedy known as Laville's tincture, which is very largely used by the laity, and which undoubtedly has a very prompt effect in many cases of acute gout.

The following has been published * as the composition of Laville's remedy, as determined by analysis :—

Quinine,	5	parts.
Cinchonine,	5	"
Colocynthin,	2.5	"
Lime Salts,	5	"
Water,	82.5	"
Alcohol,	100	"
Port Wine,	800	"

After the salicylates, the alkaline carbonates have always held a high position in the treatment of gout. Half an ounce (16 gm.) a day in divided doses should be the initial treatment, continued, but in smaller doses, when relief comes to the acute symptoms. It may be combined with a little lemon-juice to improve the flavor, or the citrate of potassium may be given in the same doses.

The lithium compounds—the carbonate and citrate—have not proved so useful as to cause me to prefer them to salicylic acid. Indeed, the early results of Garrod with them cannot be said to have been realized in modern therapeutics. Sir Dyce Duckworth says of lithia that it is a remedy better adapted to the chronic than the acute phases of gout, and so I have been using it. Five grains (0.3 gm.) four times a day, freely diluted, is the dose usually administered, and with this the potassium salts are sometimes combined.

A remedy which has recently been introduced as efficient in the treatment of gout is *piperazin*. I regret to say that I have been disappointed in it. In my early trials I thought it useful, but soon learned that it was less efficient than the salicylates and colchicum. While an acknowledged solvent for uric acid when dissolved in water, it seems to be incapable of dissolving uric acid in the system. It still has some strong adherents and may be tried. From 15 to 30 grains (one to two gm.) daily are advised, dissolved in water or in some one of the numerous mineral waters.

For the relief of the acute attack of gout, leeches, blisters, and cold have all been discontinued of late years, not only because they are useless, but also because their use has been followed by fatal attacks of the so-called internal gout. Warmth and moisture do, however, have a mollifying effect, which is increased if the liquid preparations of opium be associated. Cocaine, which might be expected to be useful, operates only through open surfaces. Should the latter be present, a five per cent. solution may be applied on lint.

It constantly happens that the pain in a paroxysm of gout is so severe that it is impossible to wait until the effect of the above remedies is secured, and a hypodermic injection of morphine is absolutely necessary to relieve the sufferings of the patient. I must remind the reader, however, that as many old subjects of gout have contracted kidneys the use of morphine under these circumstances is attended with some danger and the drug should be used with great caution.

All pressure by boots on joints disposed to gout should be carefully

* Druggist's Circular, October, 1889.

avoided, as well as injuries, as such influences undoubtedly act as predisposing causes. Muscular and mental fatigue are existing causes of acute attacks and should be avoided by the gouty.

Treatment of Retrocedent Gout.—The true nature of a metastatic attack having been determined, it must be relieved symptomatically, while efforts to simulate a true external attack may be made by hot mustard foot-baths, sinapisms, and the like. It has even been suggested that a pint of champagne may be advised, this being the wine most frequently responsible for acute attacks.

LITHÆMIA.

Synonyms.—*Uric Acidæmia; Uricæmia; American Gout.*

Definition.—A condition of imperfectly determined anatomical and chemical nature, but probably the result of an accumulation in the blood of partly oxidized products of food metamorphosis, of which uric acid is the type. It differs from gout chiefly in the absence of joint deposits and joint inflammation. In England the name of Murchison is inseparably associated with lithæmia, in this country that of J. M. Da Costa. The former ascribed the accumulation to inactivity of the liver.

Etiology.—The accumulation referred to is the result of intemperate eating and drinking, especially if associated with the want of exercise sufficient to oxidize the food ingested. In either event the income is greater than the output, accumulation results, and morbid phenomena follow. Heredity also plays a part at times.

Symptoms.—Among symptoms tolerably constant are manifestations of *indigestion*, including fulness and discomfort after meals, an unpleasant taste, sometimes nausea, at others acidity; a tendency to constipation and absence of bile from the discharges; also a tendency to aphthous ulcers in the cheeks and lips with punctiform ulcers on the end and sides of the tongue. Extreme *nervous irritability* is also often associated or may be the most striking symptom, while *vertigo* and *headache* are among the most conspicuous and annoying. While vertigo is often associated with a fulness and throbbing, the vertigo and severe headache are rarely associated. A further characteristic is the *slow pulse*, which may beat at the rate of but fifty or sixty times a minute and even less, and exhibits a correspondingly increased tension, with sharp accentuation of the aortic second sound. On the other hand, if the patient be an alcoholic or addicted to tobacco, the first sound of the heart may be feeble. Curious *paræsthesias* are also often present, of which *tingling* and a *sense of numbness* are conspicuous. There may be *anæsthesia*. In contrast with this *muscular pain*, shooting or aching may occur anywhere in the body. Last, but by no means least, is *depression of spirits*, most inveterate and unpleasant, the patient imagining he is the subject of every known disease, while suicide is sometimes sought for relief.

There is almost invariably *alteration* in the *quality* and *quantity* of the *urine*. It is *scanty*, *high-colored*, *of high specific gravity*, depositing on standing, especially at a slightly lower temperature, a large bulk of

sediment composed of mixed urates or uric acid or both. In this sediment also are sometimes included oxalate of lime crystals. Yet, though almost invariably present, this state of the urine cannot be regarded as essential, and cases occur with all the ensemble of symptoms to be named while the urine is in a natural condition.

Diagnosis.—This hinges very largely upon the condition of the urine as described and the habits of the patient. It has already been mentioned that lithæmia differs from gout in the absence of joint-symptoms, but it cannot be denied that the two conditions often run together. Less frequently, if at all, is lithæmia the result of heredity, and it demands as an essential condition the over-eating and drinking with defective oxidation referred to, which, while exciting causes of gout, are by no means always necessary to its production, especially where there is a decided hereditary tendency to the same. As intimated in the definition, uric acidæmia is rather a condition inferred than actually demonstrated—inferred from the disproportionate amount of uric acid excreted as compared with urea.

Prognosis.—It may be said, too, of lithæmia, as distinguished from irregular gout and gout, that the prognosis is on the whole more favorable, and a cure may be generally promised the patient if he comply with the physician's instructions.

Treatment.—The indications are evident. The over-eating and over-drinking must be reduced and active out-door exercise must be practised in order to oxidize the remnant of unburned tissue. The restriction which would be required depends somewhat upon the severity of the case, but in all instances may be covered by the injunction to the patient to become a vegetarian rather than carnivore. The juicy, green vegetables, such as peas, beans, spinach, asparagus, cauliflower, celery, onions, cabbage, lettuce, and an abundance of milk are allowed. The free use of water, preferably the alkaline mineral waters referred to in treating of gout, and the omission of all alcoholic drinks should be enjoined. A quart (liter) of Vichy or Vals daily, or in their absence any one of the indifferent mineral waters with the addition of lithium or sodium carbonate, should be drunk. Where urgency is required a diet of dilute milk or milk and Vichy may be insisted upon until such symptoms pass away.

Of meats which may be permitted in moderate quantity along with vegetable food are oysters, fish, the white meat of chicken, and game. The question of the carbo-hydrates is a mooted one. I have never seen such as rice and potato do harm, nor bread if of good quality and not too freshly baked. Sugar is better restricted, because of its tendency to produce acid fermentations and acidity. There is no chemical contra-indication. Hydro-carbons, on the other hand, are badly borne, and all fats should be forbidden, including butter, as well as the fat of meat.

As to medicines, the most important are the alkalies, which may be added to the negative mineral waters, if they are used instead of the more truly alkaline waters. Aperients, and of these again the salines, are especially indicated, and above all the natural mineral purgative waters, such as Saratoga waters in this country, Hunyadi water, Friedrichshalle, and the like. Phosphate of sodium is a favorite aperient in these condi-

tions on account of its supposed action upon the liver. The usual dose is a drachm (four gm.) in the morning on rising dissolved in hot water. The lithium salts may be used, and while I have not been able to trace any very direct results to their action, they serve as an excuse for the administration of liquids, since they are usually given dissolved in water, say five grains (0.32 gm.) of the carbonate or citrate in a glass of water before meals. Pleasant effervescing tablets containing these doses are now made by many manufacturing chemists. The stomachics and bitter tonics, of which nux vomica and strychnine are the types, may form useful adjuvants, and pepsin with hydrochloric acid after meals is often useful. It is in such cases, too, that hot water slowly sipped before meals is sometimes of service. The salicylates, if well borne, are useful remedies to hold in solution the uric acid and favor its elimination. Extremely painful attacks may require the use of opiates, but the doses should be as small as possible, and their use should be discouraged under all circumstances excepting extreme urgency.

DIABETES MELLITUS.

Definition.—A condition characterized by copious secretion of a urine charged with glucose and due to some as yet imperfectly understood derangement of the glycogenic and glyco-destructive function of the organism.

Historical.—Diabetes is one of the oldest known diseases, being referred to by the Roman Celsus and the Greek Aretæus, both of whom lived in the first century of the Christian era; also by the early East Indian physicians as a condition characterized by copious secretion of urine, extreme thirst, and emaciation. Little, if anything, was, however, added to the subject until the latter part of the seventeenth century, when Thomas Willis (1622–75) in England first inferred from its sweet taste the presence of sugar in the urine. Moreover, it was not until another century later, 1775, that Matthew Dobson, also an Englishman, actually obtained sugar from urine. Among other early students of this subject were Cowley (1788), Frank (1794), John Rollo (1797), and W. Prout (1825) in England and Bouchardat and Mialhe in France. Inseparably associated with the subject is Claude Bernard, who first discovered that glycosuria could be produced by puncturing the floor of the fourth ventricle. Since that time there is perhaps no subject in medicine to which has been contributed so much knowledge from an experimental side as this very one, and yet no subject as to the true pathology and etiology of which we possess proportionately less accurate information.

Other names associated with the clinical and experimental investigation of diabetes are Brücke, Cantani, Dickinson, Pavy, Ebstein, Frerichs, Külz, Lécorché, v. Mering, Minkowski, Naunyn, Seegen, C. C. von Voiht, F. Voit, and Carl v. Noorden.

Etiology.—Diabetes is not a very common disease anywhere, and it is variously frequent in different countries and races. Thus it is less common in the United States than in Europe, where there are said to be

five to nine cases per 100,000 inhabitants, while according to the last U. S. Census there are but 2.8 per 100,000. According to Dickinson the disease is more prevalent in the agricultural counties of England than the cities. It is common in Sweden on the one hand, and in Southern Italy and India, especially in Ceylon, on the other, while especially rare statistically in Holland, Russia, and Brazil.

It is much more frequent among Hebrews than Christians in the experience of almost every one, yet for what reason I have been unable to discover. It is rare in the negro race, though I have met several cases. It is a disease especially frequent among the rich and well-to-do, though the poor are not exempt. It is also a disease of adults, yet it has occurred in infants at the breast. In the reports of the Registrar-General of England for the year 1851–60, ten deaths are registered under the age of one year and 32 under the age of three. The youngest patient I ever had was a little girl, aged twenty-two months, who was delivered prematurely because of nephritis in the mother, though she was healthy up to one year. Albuminuria was associated with the diabetes. The disease is most frequent between the ages of thirty and sixty. It is more serious in the young, recovery in very young subjects being almost unknown. It is very much more frequent in males than females, in the proportion of nearly three to one, though Senator's * statistics show that under the age of twenty more females are affected than males. This has been my own experience. Little is known of the effect of occupation, though it is thought that occupations taxing the mind favor it. It has occurred to me to treat a number of physicians and farmers. Heredity has in my experience been less conspicuous than European writers find it. From ten to 25 per cent. are thus traced by different Continental observers.

Pathology and Pathogenesis.—The etiology of diabetes is so intimately united with its pathology that it is scarcely possible to separate their consideration. What is known, therefore, of its immediate causation will be developed in connection with the pathology, while its more remote causes will be briefly considered in the ensuing paragraph. Inseparably connected, also, with the pathology of diabetes are the phenomena of sugar formation in the economy. A brief statement of the latter seems, therefore, justified. During life there is constantly being produced and stored in the liver of man and the lower animals an *amyloid* substance, which was named by its discoverer, Claude Bernard, *glycogen*.† Its formula is $C_6H_{10}O_5$, that of starch, and the term *zoamylin*, or animal starch, was at one time suggested for it. The glycogen formation takes place whether animal or vegetable food is taken, but it is much larger upon a vegetable diet. It is commonly held that it does not occur at all with a diet of pure fats, but Salomon ‡ claims that it is produced in the livers of rabbits fed on olive oil. C. von Noorden also considers that fat

* See Senator's article on "Diabetes Mellitus" in "Ziemssen's Cyclopædia of Medicine," Vol. XVI, p. 866, *ad fin.*

† Bernard, "*Nov. Fonc. du Foie.*" Paris, 1853.

‡ *Virchow's Archiv*, Vol. 61, Part 3, 1874, 18.

is converted into sugar in the liver.* All physiologists agree that this amyloid substance is derived mainly from the starchy and saccharine principles of food, but partly also by a splitting up and rearrangement of the elements of nitrogenous food. This possibly also takes place in the liver, resulting in urea and glycogen, the latter being stored in the liver-cells. The muscles are also favorite reservoirs for glycogen storage. The most important property of glycogen is its ready convertibility at the temperature of the body into glucose, or grape-sugar. For this is probably also required a glycolytic ferment. By means of these storage reservoirs the blood is kept supplied with 0.12 to 0.18 per cent. of grape-sugar, which in health supplies all the metabolism and force production.

In diabetes mellitus some derangement of this balance takes place, as the result of which there is an under-consumption of grape-sugar rather than an over-production, which is also, however, a possible factor, while it is not impossible for the two to be associated. Whichever of these it is, the excess of sugar thus produced in the blood is eliminated by the kidneys, and thus glycosuria becomes an essential symptom of diabetes mellitus.

What causes diabetes? I have already said there is no disease concerning which so much accurate knowledge has been arrived at and of the true pathology of which we are so thoroughly in the dark. It is not a kidney-disease, as was once supposed in its early history, although this impression still prevails among the laity, and naturally so, because the essential evidence of its existence is found in the urine. What more do we know? We know, further, that diabetes occurs under very different circumstances. We can produce diabetes in an animal by irritating the floor of the 4th ventricle, as originally done by Claude Bernard in his celebrated piqûre experiment. There are, however, also other parts of the nervous system the irritation of which will produce, though less promptly, diabetes, from the cerebellum down to the point of emergence of the sympathetic system to the viscera. It is commonly admitted that this experimental glycosuria is caused by a centrifugal stimulus from the nervous centres to the liver, either through the vaso-motor system or direct stimulus to the liver-cells. We know also that tumors impinging on the floor of the 4th ventricle, injuries of this part of the brain, and abscesses are attended by diabetes mellitus; also injuries to the spinal cord; also diseases of the pancreas. On the other hand, we know that autopsies in a large number of the gravest forms of diabetes have failed to discover any lesion whatever; in fact, the most unmanageable and serious cases are those in which we find no lesion. Therefore, while we must admit that the nervous system has something to do with the production of diabetes, sometimes directly and in other instances indirectly, we are not able to trace a nervous lesion in every case. It is further likely that the sympathetic nerve is an important channel for nervous influence, regulating as it does the opening and the closing of the blood-vessels.

There are found, also, other interesting lesions in connection with

* Article, "Diabetes Mellitus," in "Twentieth Century Practice of Medicine." New York, 1895, Vol. II, p. 42.

diabetes in distant organs, and one of the most important organs involved is the pancreas. This is proved from the experimental as well as from the clinical side, and I have myself, in a few cases at autopsies, found a pancreatic lesion. Hanseman claims pancreatic lesions in 50 per cent. of cases. From the experimental side it is found by von Mehring and Minkowski that extirpation of the pancreas is immediately followed by diabetes; and, although there are some differences in results, it is one of the best determined facts that glycosuria follows such extirpation. Sometimes such a diabetes has been transient, but then it has been found also that a fragment of the pancreas was left behind. But while we must say that the pancreas has something to do with a large number of cases of diabetes, we cannot say it of all, as there are some in which no lesion is found. The rationale of this relation is not settled.

It has been alleged that the absence of the pancreatic secretion has something to do with this. But it has been shown that simply cutting off the secretion from the intestine does not cause diabetes. It has been suggested that extirpation of the pancreas really operates by disturbing the sympathetic nerves in the vicinity. But this has also been experimentally refuted, and with this the view of Klebs, also accepted by Senator, that the coexistence of diabetes mellitus and disease of the pancreas is due to lesions of the cœliac plexus primarily or secondarily. The researches of Lepine, which have yet, however, to be confirmed, ascribe it to the absence of a glycolytic ferment furnished in health by the pancreas to the blood. This explanation certainly accounts for the facts. But it is impossible to explain all cases of diabetes from any one standpoint to the exclusion of another. There is no doubt that the pancreas has something to do with it, but it has not all to do with it. The truth is that a certain number of cases are dependent on derangements of the nervous system, either directly or reflexly, a certain number are produced by an interference with the proper function of the pancreatic secretion, while others still are inexplicable.

Morbid Anatomy.—Diabetes can hardly be said to have an essential morbid anatomy. For, except in the instances alluded to, the morbid lesions which are found after post-mortem examination to have been associated with diabetes are, in the main, such as are the consequence of the continued presence of the condition, rather than such as cause the symptoms. Sometimes there are absolutely no alterations discoverable either by the naked eye or the microscope.

To begin with the organ which is apparently immediately responsible for the symptoms, the liver, it frequently presents the appearances of a hyperæmic organ; that is, it is darker and harder than the normal organ, while it is also enlarged, sometimes considerably, at others only slightly. Corresponding to this, the microscope, by very moderate amplification, shows enlarged and distinct acini, with capillaries dilated and distended with blood in various degree. Higher magnifying powers—300 to 400 diameters—show the liver-cells to be enlarged, distinctly nucleated, rounded, and disposed to fuse. If a weak solution of iodine is added, they strike a wine-red color, which, according to Rindfleisch, is confined to the nucleus; but, according to Senator, may extend

to the whole of the cell. Klebs ascribes this reaction to post-mortem changes in the glycogenic substance. The minute changes described are said by Rindfleisch to be more striking in the peripheral zone of the lobule than of the portal vein, while the intermediate zone, or that of the hepatic artery, is fatty, and the central part, including the rootlets of the hepatic vein, is nearly normal. Incidental morbid states are hypertrophic cirrhosis and atrophic cirrhosis. An interesting fact in this connection is that the most serious organic disease of the liver appears never to cause glycosuria.

As to the pancreas, it may be well to mention the changes found at different times. They include calculi found in the pancreas of a diabetic as early as 1778 by Th. Cowley, cancer by Bright at a comparatively early date, and atrophy by Griesinger, who had found the pancreas atrophied in one of the five diabetics whose bodies he examined after death, yet believed that this lesion was of no significance whatever. But the observations which have been published in great numbers (Hartsen, Fles, v. Recklinghausen, Frerichs, Klebs, Harnock, Kuelz, Schaper, Lancereaux, Senator, and others) allow us to assume that diseases of the pancreas are present in about one-half of all the cases of diabetes. The statement of Senator * that this organ "is found diseased with surprising frequency, in particular either atrophied or, in addition, degenerated," is in the main correct. Among nine cases Frerichs saw atrophy or fatty degeneration of the gland five times, and in the Vienna dead-house the pancreas was found strikingly small, soft, and anæmic in 13 out of 30 diabetics (Seegen). Other coincident diseases of the pancreas already noted are cancer and impacted calculus.

The kidneys, primarily unaffected, are in many cases sooner or later influenced by the constant hyperæmia to which they are subject in eliminating the sugar. The appearances commonly met are those of hyperæmia and overgrowth of epithelium—in a word, those of catarrhal nephritis. Occasionally the changes are more advanced, and the epithelium is fatty. More rarely granular contracted kidney is present, contributing a more serious significance to the albuminuria. These changes need not necessarily be attended by albuminuria previous to death. In other cases nephritis may be an accidental coincidence. There occurs also sometimes a vesicular swelling of the epithelium in the straight tubes, hyaline changes in the descending limb of Henle's loop, as well as a hyaline change in the vessels of the Malpighian tubes. As to the proportion of cases in which the kidneys reveal morbid alterations, it is a decided majority.

The lungs are often the seat of tubercular deposits and cavities resulting from their softening; also of broncho-pneumonia and croupous pneumonia, which may terminate in gangrene. The heart is sometimes hypertrophied.

Symptoms.—Almost invariably the earliest symptoms noted by the diabetic are *thirst* and *frequent micturition.* One or the other of the two may be noticed first, or the patient's attention may be called to both

* Senator, *loc. cit.*, p. 887.

simultaneously. It occasionally happens that a *dryness of the fauces* and a glutinous, *viscid* character of the *saliva* attracts attention before any other symptom. Sometimes it is observed that a drop of urine falling upon the boots or clothing and there evaporating leaves a persistent white spot, which is sugar. A *dryness and harshness of the skin*, due to absence of perspiration, soon make their appearance and early attract the attention of those who ordinarily perspire easily and occasion varying amounts of discomfort. *Itching* of the skin is also sometimes present. The *temperature* of the body is not increased, at this stage scarcely altered, although later in the disease it is decidedly lowered. If the further progress of the disease is not arrested a *voracious appetite* becomes the next symptom, notwithstanding which the patient observes that he slowly *loses in weight* and grows daily *weaker*. The rapidity with which these symptoms succeed each other varies. Sometimes it is with great rapidity, constituting an acute form, at others the successive stages are exceedingly slow in developing chronic diabetes.

The above category includes all the symptoms which present themselves in the milder form of the disease. But unless averted, all of these symptoms become intensified. The patient complains of constant burning thirst, is constantly urinating, and as constantly drinking water to quench his thirst, and, while often eating enormously, grows emaciated, although at the onset of the disease he may have been a robust, vigorous man.

Dyspeptic Symptoms may appear at various stages, seldom very early. Acid eructations, flatulence, and epigastric pain, or an indescribable sensation spoken of as "sinking" of the epigastrium, are among them. *Constipation* is sometimes a very troublesome symptom, and adds in my experience to the seriousness of the case; on the other hand, diarrhœa sometimes is present. As the disease advances there is a peculiar vinous or *acetous odor* of the breath, which has been compared to that of stale beer, and by Sir Thomas Watson to the odor of a place in which apples are kept. This is believed to be due to acetone and diacetic acid, both of which exist in the blood of profound diabetics during life.

Later *cough* often sets in, owing to bronchitis and tubercular phthisis, and, with the copious expectoration incident to them, adds to the debilitating agencies already at work. Roberts thinks phthisis occurs in one-half the cases. I am sure not so many die of it in this country. C. van Noorden says one-fourth of all diabetic subjects in Germany have the disease, and Lancereaux that victims of pancreatic diabetes are especially prone to tuberculosis. The consumption thus induced sometimes rapidly hastens the fatal termination, while at others it appears to have but a trifling influence in this respect. The other symptoms characteristic of pulmonary consumption are also present, not even excepting hectic sweats. The perspiration thus arising may contain sugar.

Boils and *carbuncles* in the skin are also of frequent occurrence, favored by the malnutrition growing out of diabetes, and the former are occasionally the first symptoms recognized. The latter never occur early, but when present are frequently the immediate cause of death.

Gangrene of various parts of the body is another of this class of

symptoms. It is sometimes spontaneous, but more frequently is immediately caused by some trifling injury, which under other circumstances would be without result. It has been known to start from a blister or the simple cutting of a corn. Beginning most frequently in those parts of the body most remote from the centre of the circulation, as the toes, its progress and appearances are like those of senile gangrene. Sometimes, however, the gangrene is moist.

Eczema, with itching and burning of the labia and vicinity, is a frequent and troublesome symptom in women which is incident to the extremely frequent micturition. In the male, the meatus urinarius is sometimes the seat of a similar irritation.

The early loss of sexual desire is characteristic.

In advanced stages the temperature of the body, unless influenced by intermittent febrile disease, is almost invariably lowered from 1° to 2½° F. (0.55° to 1.6° C.).

The above-recorded symptoms occur sooner or later in most cases, the thirst and saccharine polyuria being essential. There are many others which occur more or less frequently, but not constantly. Among the rarer symptoms is *cataract*, the association of which with diabetes was long ago noted by Prout. It develops rapidly and is nearly always symmetrical, involving both eyes simultaneously, but not to the same degree. It is sometimes a very nice point to determine whether cataract is due to diabetes or to the usual causes. The earlier the age at which it occurs the more likely it is due to diabetes.

The ophthalmoscope may reveal dilatation of the *retinal* vessels. The late Albert G. Heyl* described a condition which he called intraocular lipæmia, in which the light salmon color of the blood contained in the branches of the retinal vein and artery contrasted with the cinnabar-red of the vein and yellow-red of the artery, also by the greater width of these vessels and the lighter yellow of the fundus. Finally, atrophy of the retina and hemorrhagic and inflammatory affections of the *eye* have been described. Among other derangements of *vision* said to occur are amblyopia, presbyopia, and loss of accommodating power from defect of the ciliary muscle. Occasionally total blindness has occurred from atrophy of the retina.† *Derangements* of other *special senses* said to attend diabetes are impairment of hearing, roaring in the ears, and derangements of smell and taste.

A *spongy* state of the *gums* with recession and excavation is an occasional symptom, resulting in extreme cases in absorption of the alveolar processes and falling out of the teeth.

Severe *neuritis* in the brachial and crural nerves is not infrequent. In severe cases the *tendon reflexes* are diminished or absent. *Unilateral sweating* has been observed. Senator refers to three cases, two of the left half of the face and one of the right. *Œdema* sometimes appears late in the disease, not usually the result of the renal involvement.

---

* "Lipæmia and Fat Embolism in Diabetes Mellitus," New York Medical Record, Vol. XVII, 1880, p. 477.

† Dufresne, "*De l'Amblyopie Diabétique*," Gaz. Heb., November, 1861.

Diabetic coma, first described by Kussmaul in 1874, is a form of coma which often comes on in advanced stages of diabetes and almost as often terminates in death. The condition is one of suddenly or gradually supervening unconsciousness, with or without previous irritability or uneasiness, anxiety, vertigo, or symptoms resembling alcoholic intoxication. Sometimes it is preceded by obstinate constipation or intestinal catarrh or severe colicky and muscular pain. Convulsions do not occur, but the eyes are half open, the pupils dilated, and the eyeballs wandering. In addition to coma there are frequent and feeble pulse, deep inspiration, with short expiration, more or less frequent than in health, and gradually invading cyanosis. The temperature, at first slightly elevated, is subsequently subnormal. The condition lasts from twenty-four to forty-eight hours, when death usually supervenes. Over one-half of all deaths in diabetes are ascribed to diabetic coma by Frerichs, but others assign a much smaller number to it. The odor of acetone may issue with the breath. It has been variously ascribed to acetone and diacetic acid, to oxybutyric acid, and by Professor Saunders and D. J. Hamilton * to slow carbonic-acid poisoning due to fat embolism of the pulmonary vessels, the result of lipæmia. All views are speculative. Acetone and diacetic acid are very often present for a long time, and yet no diabetic coma supervenes. There would seem to be better reason for ascribing the condition to "acid intoxication," as held by Stadelmann and Minkowski, since the continued presence of oxybutyric acid is always followed sooner or later by coma unless the patient die from some other cause. Diabetic coma must not be confounded with other forms of coma which may occur in diabetes, as true apoplexy and uræmia.

Alterations in the Blood.—It has already been mentioned that in diabetes the blood becomes highly charged with glucose, which increases from a normal of 0.05 to 0.15 per cent. to 0.2 and in extreme cases to 0.57 per cent., and from this glycæmia comes glycosuria. From the presence of the first, we should naturally expect a higher specific gravity of the blood serum, which has been found as high as 1033, as contrasted with the normal 1028. On the other hand, the serum has been found thinner than normal, containing, according to different analyses, from 80.2 to 84.8 of water instead of the normal 78 to 79 per cent. The red blood discs are often diminished, and the alkalinity of the blood is also. As such diminution is at a maximum when oxybutyric acid is being excreted, it has been ascribed to this substance.

An abnormal amount of fat in the blood, producing the technical lipæmia, was observed by the earliest students of diabetes, and is attested by many analyses, as well as by the milky appearance of the serum and the intraocular appearances described by Albert G. Heyl. The analyses of Simon show from two to 2.4 per cent. instead of the normal 1.6 to 1.9 per cent.

Changes in the Urine.—The most noticeable peculiarity of diabetic urine to the patient is its enormous quantity, which has been known to exceed 70 pounds (31.78 kilograms) in twenty-four hours, while apocry-

* Edinburgh Med. Jour., July, 1879.

phal accounts of larger amounts are extant. Frank records 52 pounds (23.6 kilograms); Bardsley* 36 pints (20.4 liters) and 32 pints (18.16 liters); Bence Jones found 56 pints (31.78 liters); Sir Thomas Watson and Dr. Dickinson 26 pints (14.77 liters), and Dr. Pavy 32 pints (18.16 liters). From 70 to 100 ounces (2100 to 3000 c. c.) are frequent quantities. The quantity of urine passed is limited by the amount of fluid ingested. For while it is possible that the amount of urine secreted may exceed for a very short period that ingested, it is evident that this cannot continue for any length of time, and, in point of fact, it is found to be almost invariably a little less, the remainder being removed by the lungs, skin, and bowels. On the other hand, it was early observed by Th. Cowley† (1778) that the quantity of water occasionally is not at all or but slightly increased. To this condition Frank,‡ another old author, gave the name of *diabetes decipiens*. It is well known, also, that intercurrent disease, especially febrile affections, sometimes diminish the quantity of urine as well as the amount of sugar excreted; while the same diminution of urine and sugar also occasionally occurs toward the fatal termination of the disease.

But the most important change is, of course, the presence of *glucose*. Of this the quantity varies greatly in different cases and at different times in the same case. Every case of trifling and temporary glycosuria should not, however, be considered a case of diabetes. The sugar should be easily recognizable by the ordinary tests and should be constant. From what may be indicated as "evident traces" the proportion of sugar may reach, it is said, as much as 15 per cent. I have never met more than ten per cent., though I often hear larger quantities erroneously reported. The twenty-four hours' quantity varies similarly. The maximum quantity secreted in this time appears to be that reported by Dickinson, wherein a man twenty-five years of age voided 50 ounces, or 1500 gm., of glucose in twenty-four hours. But the more usual quantity is from ten to 80 milligrams to the c. c., or from 20 to 25 gm. in twenty-four hours; this corresponds nearly to from five to 30 grains to the fluid ounce of the English system, or from 300 to 3800 grains (20 to 253 gm.) in the twenty-four hours.

The effect of muscular exercise in diminishing the quantity of sugar in the urine of diabetics was early confirmed by Kuelz and others, while it is scarcely necessary to say that accidental as well as intended changes in diet are followed by consequent variations. So, too, urine passed after fasting, as on rising in the morning, contains generally less sugar than that passed after a meal, and in the clinical study of cases of diabetes, where a part of the twenty-four hours' urine is not obtained, it is important to bear this in mind. As the difficulties in obtaining a part of the twenty-four hours' urine regularly are very great, it is preferable, in my experience, to take for examination two samples, one passed in the morning

* Bardsley, article on "Diabetes" in the "Cyclopædia of Practical Medicine," Philadelphia, 1845, p. 607.

† Cowley, Th., London Medical Journal, 1788.

‡ Frank, J. P., "*De Curandis Hom. Morbis Epitome.*" Lib. v, De Profluviis, Pars. i. Manheimii, 1794.

on rising, and representing the fasting urine, and another on going to bed, representing the day urine.

Consistently with the increased solid matter thus added, the *specific gravity* of diabetic urine is, as a rule, high, 1040 being very common, while Bouchardat found it as high as 1074 in one instance. The well-known disposition of diabetic urine to become *frothy* on shaking, and to maintain this frothy condition, is a natural physical result of its increased density. Urine may, however, have a low specific gravity and yet contain sugar. I have found it as low as 1010. Such low specific gravities of glucose if present in any decided degree must depend on the low proportion of other normal ingredients. Sugar sometimes disappears very rapidly from urine by fermentation, thus reducing also the specific gravity.

Concurrent with the increase in quantity of urine is a *paleness*, which amounts in extreme degrees to an almost absolute absence of color, so that the urine is almost as colorless as spring-water. Almost all diabetic urine, sooner or later after exposure at a moderate temperature, becomes *cloudy* from the development of fungi coincident with fermentation. The *odor* of the urine is usually normal when first passed, but sooner or later, in consequence of fermentation setting up, it may acquire an acetous odor. The latter change also increases the degree of the normal acid reaction and maintains it much longer after exposure to the air than is the case with normal urine. This acetous odor is ascribed to acetone and diacetic acid. The urine may have a sweetish odor when passed, which has been compared to "sweet brier." Diabetic urine is sometimes quite free of *sediment*. At others there is a copious sediment of uric acid. In the sediment may also be included the *penicilium* fungus, common to acid urine, as well as the more characteristic yeast or sugar fungus, the *torula cerevisiæ*. This also sometimes appears as a *mould* on the surface of the urine.

Of the normal chemical constituents of the urine, *urea* is almost invariably increased. This is contributed to by two causes. The first is the ingestion of large amounts of nitrogenous food, whether to appease the appetite or by the physician's advice. The second cause is the decomposition of the tissues themselves which characterizes the severest cases in the last stages in spite of the enormous food-consumption. In such event, the nitrogenous tissues are split up into urea and sugar.

As regards *uric acid*, it is either normal or slightly increased. Of the other constituents of the urine, creatinin is increased; sulphuric acid is subject to its normal variations; chlorine, phosphoric acid, lime, and magnesia are said to be increased, phosphoric acid and lime especially so. Ammonia is largely increased.

Of *abnormal* constituents, albumin is often present,—perhaps in one-third of all cases; some make it a larger proportion, some less. It is not generally large, and in my experience is not often a serious symptom. Albuminuria does not necessarily imply renal change. It is scarcely necessary to say that the urine may become albuminous from any of the causes of albuminuria independent of diabetes, as pus from pyelitis, cystitis, etc.

Inosit, or muscle-sugar, occasionally replaces the grape-sugar in diabetes, but more frequently accompanies it. Gallois* found it in five out of 35 diabetics.

Finally, *acetone, diacetic acid, and beta-oxybutyric acid* are all frequently met in diabetic urine. It is now conceded that the source of these substances is albumin, aceto-acetic acid probably being the first formed and rapidly transformed into acetone. When but little diacetic acid is formed, it is all converted into acetone; when much is formed, both substances appear in the urine. The conversion takes place mainly in the urine, but doubtless also in the tissues or the blood, since acetone appears in the expired air. To acetone is ascribed the vinous odor sometimes present in the urine. Acetone is produced in health in a slight amount in the normal decomposition of albumin, also in other diseases than diabetes, to a large extent. According to van Noorden these substances are formed in the decomposition of the albumin of the body and not of the food; in a word, when the patient is "consuming his own proteids."

Beta-oxybutyric acid is believed by many to be the first stage in the formation of diacetic acid. Van Noorden also thinks this possible, but he claims for it a certain "clinical independence," and regards it probable that oxybutyric acid on the one hand and aceto-acetic acid on the other arise from qualitatively different decomposition processes. While acetone in the urine does not add to the seriousness of diabetes, the presence of oxybutyric acid is of the gravest prognostic significance, never disappearing permanently after being once present, and almost always followed in a few days or weeks by diabetic coma and death. Diacetic acid has an intermediate significance between acetone and beta-oxybutyric acid.

Of the other secretions, the *perspiration* when present, frequently although not invariably, contains sugar, sometimes a notable amount, as much as 6½ grains (0.42 gm.) having been extracted by Fletcher from a piece of flannel three inches square which had lain upon the skin forty-eight hours. The *saliva* has rarely been found to contain sugar independently of that which it acquires from the food. Whether the *gastric juice* ever contains it under similar conditions is disputed, but it has been found in effusions and exudations, as might be expected.

Duration.—Though the course of a few cases of diabetes is so rapid as to justify the name acute, the number of these cases is not sufficient to justify a classification into acute and chronic. In such rapid cases death has taken place at periods ranging from two days to six weeks. Yet in no instance can it be averred that the disease was of as short duration as it seemed, since it may have existed some time before it was discovered, while in several it was evidently longer. It is true, therefore, that diabetes mellitus is a disease almost invariably of long duration. Cases of fifteen, eighteen, and twenty years' duration are reported. The younger the subject the shorter the duration and the more promptly fatal the result, while after middle age, under treatment the duration may be indefinite.

* Gallois, "*Comtes Rendus*," 1863, I, p. 533; also "*De l'Inosurie*." Paris, 1864.

Diabetes mellitus is sometimes distinctly intermittent for a time, regardless of treatment. I was for a long time incredulous on this subject, but recent experience has taught me that such a form of diabetes occurs, in which both polyuria and glycosuria may disappear without treatment, to recur again. Such cases are, however, easily controlled by treatment when discovered, while they are also certain to pass over into the permanent form if neglected.

Complications.—The diabetic is characteristically subject to complications which may be accounted for in a word by his diminished power of resistance against all disease-causing agencies and the toxic influences incident to the disease. Most of these have already been considered among symptoms. Such are the numerous skin affections, gastro-intestinal disturbances, pancreatic and renal affections, functional cardiac and nervous symptoms, including neuritis and diabetic coma. Tuberculosis has also been mentioned. It is especially apt to attack young subjects and to cause their death. Arterio-sclerosis is prone to occur in diabetics with its full train of consequences and to appear earlier in life among them. Jaundice sometimes occurs, and having presented itself twice in the history of a case under my observation, can hardly be considered accidental, although I am at a loss how to account for it. Senator says that, when not an accidental complication due to a catarrh of the duodenum, it may result from compression of the biliary capillaries by the over-loaded blood-vessels or enlarged gland cells of the liver.*

Diagnosis.—The diagnosis of diabetes mellitus is very easy. Unnatural thirst and copious diuresis should always suggest a chemical examination of the urine. And although there are sources of error in testing for small quantities of sugar in urine, the quantities thus overlooked are not usually of clinical significance. In fact, in my observations, glucose is more apt to be declared present by inexpert examiners when absent than the reverse. Almost any one of the tests, therefore, which are found in the various manuals for the examination of urine, applied with ordinary care, will respond readily to quantities of clinical significance.

For provisional purposes Trommer's method of using the copper test answers very well. Its ingredients are easily attainable, and there is no risk of error from changes from keeping, to which Fehling's and Pavy's solutions are subject.

Trommer's test may be applied as follows :—

To a small quantity of urine, say five c. c. (80 minims), add half as much liquor potassæ or sodæ, then drop by drop from a five or ten per cent. solution of cupric sulphate. On first adding the copper, a blue precipitate of hydrated cupric protoxide takes place, *which, if sugar is present, is redissolved on shaking*, producing a clear blue liquid. The copper solution should be thus added until the precipitate is no longer dissolved on shaking. Then heat the mixture to boiling, and if sugar is present a copious yellow precipitate of hydrated cuprous oxide or of red cuprous oxide occurs. Either is conclusive evidence. Occasionally the precipi-

* Senator, *loc. cit.*, p. 912.

tate of earthy phosphates is so copious as decidedly to obscure the reaction and by beginners is sometimes mistaken for the suboxide. In this event they may be removed by filtration after *slightly* warming the mixture. The reaction should take place as soon as the boiling point is reached—indeed, sometimes before this point is reached. Prolonged boiling should be avoided.

If Fehling's solution is used, a rough quantitative estimation may be made at the same time by the clinical method :—

A given quantity, say one c. c., of Fehling's solution is placed in a test-tube, diluted with about four times its bulk of water, and boiled for a few seconds. If the solution remains clear, add immediately the suspected urine drop by drop. If sugar is abundant, the first few drops will usually cause the red or yellow precipitate, but if the reaction does not occur the dropping may be continued, followed each time by heating until an equal volume has been added. If no red or yellow precipitate occurs, sugar is absent. Now, Fehling's solution * is so composed that if an equal bulk is exactly reduced by an equal bulk of urine, that urine contains half of one per cent. of glucose ; if by half bulk, one per cent. ; if twice the bulk, ¼ per cent., and so on, whence one can easily estimate roughly the percentage. Should the urine contain more than one per cent. of sugar it should be diluted one to ten and the result multiplied by ten.

If a reduction takes place on boiling the test fluid alone, a new supply may be obtained, or a little more soda or potash may be added, the fluid filtered, and it is again ready for use. Such spontaneous reduction of the cuprous-oxide often occurs when Fehling's solution is kept for some time.

In judging of the progress of a case of diabetes under treatment, it is not sufficient to test the urine qualitatively, but a quantitative determination of sugar must be made. This may be done by the clinical method just described or volumetric processes described in the manuals for the examination of urine, but the simplest method is the fermentation method of Dr. Roberts. In this the specific gravity of the urine is taken before and after fermentation, and the difference in the specific gravity indicates the number of grains of sugar in each fluidounce of urine. Then suppose the specific gravity before fermentation to be 1045, and after fermentation 1035, the quantity of sugar per 30 c. c. (one fluidounce) is 0.65 gm. (ten grains). This can be reduced to percentage by multiplying the difference by 0.23.

Tests for Acetone, Diacetic Acid, and Oxybutyric Acid.—Of the numerous tests for acetone, most of which require the distillate for their

---

* *Fehling's Solution.*—Dissolve 34.652 gm. pure crystallized sulphate of copper in 200 gm. distilled water; 175 gm. chemically pure crystallized neutral sodic tartrate in 480 gm. solution of caustic soda of specific gravity 1.14, and into this basic solution the copper solution is poured, a little at a time. The clear, mixed fluid is diluted to one liter, or 1000 c. c.

Ten c. c. of this solution will be reduced by 0.05 gm., or 50 milligrams, of diabetic sugar. If the Fehling's solution is to be kept some time, it is absolutely essential that it should be placed in smaller bottles holding 40 to 80 gm., sealed and kept in the cellar.

Still greater security may be obtained by dissolving the cupric sulphate in 500 c. c. and the tartrate salt and potash in 500 c. c., keeping the two solutions separate in rubber-stoppered bottles. Equal volumes of the two solutions are united when needed for use.

successful application, Legal's nitroprusside-of-sodium test is the most satisfactory for the practitioner because it does not require the distillate.

Legal's Test.—A fresh, rather strong solution of sodium nitroprusside is made by dissolving a few fragments in a little water in a test-tube. To three or four c. c. of the suspected urine add enough liquor sodæ or potassæ to secure a distinct alkaline reaction. To the mixture then add a few drops of the nitroprusside solution, when promptly the whole assumes a red color, whether acetone is present or not, said to be produced by creatinin even more rapidly than by acetone. In any event, the red color disappears, but if acetone is present, the addition of a few drops of concentrated acetic acid causes a purple or violet red. If there is no acetone, this final change does not occur, while the purple color also fades in a little while, even if caused by acetone.

To *test for diacetic acid* add a few drops of a solution of ferric chloride to a small quantity of the urine, when a beautiful Burgundy-red reaction occurs. A precipitate of phosphates succeeds the adding of the first few drops, but this is redissolved by a further addition of the chloride. A more brilliant reaction is obtained if the urine be first treated with a solution of acetate of lead, filtering out the white precipitate and testing the filtrate. Urine passed after the administration of salicylic acid, antipyrin, carbolic acid, salol, phenocol, kairin, and other drugs furnishes a similar reaction.

The reaction for diacetic acid being obtained, it is scarcely necessary to *test for beta-oxybutyric acid*, as the significance is the same. The test is a complicated one, but beta-oxybutyric acid is presumably present when a quantitative estimation indicates a larger quantity of glucose than polarization, since beta-oxybutyric acid rotates polarized light to the left as contrasted with the dextro-rotatory power of grape-sugar. Again, if after complete fermentation with yeast or precipitation with basic acetate of lead and ammonia the urine is found levo-rotatory, beta-oxybutyric acid is presumably present.

Prognosis.—The prognosis of diabetes varies with the age at which the disease makes its appearance, the time which has been allowed to elapse before treatment is instituted, and the treatment itself. Once thoroughly established early in life, or before twenty-five years of age, recovery is rarely possible, but even at this age, if treatment is instituted sufficiently early, much may often be done to avert the end. Diabetes is a disease in which the expectant plan of treatment is disastrous. It is a disease which never gets well of itself, and always gets worse if not properly treated. At the same time the mild cases amenable to treatment are in a decided majority. When the disease appears after middle life in obese persons or those disposed to gout, is early recognized and promptly treated, it is usually easily controlled; and although it is almost never safe to declare a case of diabetes absolutely cured, it does occasionally happen that recovery is so complete that the patient may be left to his own mode of living. As a rule, however, even those who have apparently recovered have to keep a watch upon their diet, and should at intervals have their urine examined with a view to sounding, as it were, their condition. We are entirely justified in saying to a diabetic patient, "As long as your

urine remains free of sugar you are practically as well as if you had no tendency to diabetes." On the other hand, for spare, nervous, and hard-worked persons, especially mentally over-worked, under forty, there is a much more unfavorable outlook. Even here, if the coöperation of the patient can be secured, much may be done. Every variety of intermediate grade may occur. When diabetes depends upon recognized nervous lesions the prognosis is altogether that of the lesion itself. The cause of death is very frequently some intercurrent or consequent disease, as consumption or diabetic coma. The syphilitic origin of the disease and obesity are favorable prognostic factors, a spare habit and constipation are unfavorable.

Treatment.—This resolves itself easily into the dietetic, the hygienic, and medicinal.

I. *Dietetic Treatment.*—This is by far the most efficient, and no permanent results have ever been obtained without it. It consists essentially in the elimination from the diet of such articles as are readily convertible into glucose, viz., the carbohydrates. It is acknowledged that in the early stage of the disease all the glucose in the urine is derived from saccharine and amylaceous alimentary principles. Hence, if these be excluded from the diet and their place supplied by other assimilable articles, the symptom disappears, and the disappearance of this symptom seems to be, for the time being at least, the cure of the disease.

If it were necessary to select a diet absolutely free from sugar and starch, it would indeed be restricted, as there are comparatively few articles of food thus constituted. Such are, however, meats of every kind, fresh or salted, including tripe, tongue, ham, bacon, and sausage; soups made from meat without flour; game, poultry, fish, oysters, lobsters, crabs, eggs in every shape; butter and new cheese, oils and fats. Happily, however, it is not necessary to use articles absolutely free from the two baneful principles, and in this manner quite a variety of palatable articles may be added to the dietary. Among these are cream, curds, milk and buttermilk, all green vegetables, including spinach, endive, the green leaves of lettuce, dandelion, cabbage, as coleslaw, Brussels sprouts, cauliflower, broccoli, string beans, tomato, watercress, celery tops, asparagus tops, turnip tops, young onions, cucumbers, pickles, and olives. To these may be added unsweetened jellies (preparations of gelatin) and especially a variety of nuts, including almonds, walnuts, butternuts, filberts, pecan nuts, Brazil nuts, but not chestnuts; also, all acid fruits, as apples, lemons, strawberries, etc. Tea and coffee, with cream and without sugar, cocoa-nibs, but not chocolate, are permitted; also, all wines which contain little or no sugar, including claret, Burgundy, Rhine, and still Moselle wines, together with very dry sherry, unsweetened brandy and whiskey, and gin when required. The carbonated waters, natural or artificial (the so-called soda-water of the shops), are preëminently suitable. Water is to be allowed *ad libitum*, for water is the medium by which the sugar is carried out of the blood and tissues. Its supply should therefore be liberal, and with the diminished sugar-formation comes diminished thirst. Better still are the alkaline mineral waters, especially those of Vals, Vichy, and the Saratoga Vichy.

Beer, ale, porter, cider, and the fermented liquors generally are not allowable because of the sugar carbohydrates they contain. They are less objectionable when fermentation is carried to a high degree, resulting in a more complete destruction of the sugar. This is the case with certain bottled lager beers and English ales.

It is not simply the small quantity of sugar and starch contained in them which renders the vegetable substances named admissible, for many of them contain a good deal of sugar; but these sugars, unlike grape-sugar, are assimilable. Such are preëminently mannite, the sugar of manna; lactin, or sugar of milk; lævulose, or fruit-sugar, and probably, also, inosit, or the sugar of muscle. Such is also inulin, a hydrocarbon and starchy principle found in the *inula helenium*, or elecampane, but especially in Iceland moss. Hence, too, the impunity with which milk can often be taken by diabetics, although it contains from three to six per cent. of lactin. On this account, too, lævulose may be used for sweetening tea and coffee in mild cases. Glycerine is also sometimes substituted for sugar, but though less objectionable, both theory and experience go to show that it is not a safe substitute. Lævulose and even mannite are much to be preferred to glycerine, both for sweetening and to substitute the part of sugar in force-production.

It will be noted that not only all saccharine substances of animal or vegetable origin and all vegetables largely composed of starch, as potatoes, rice, and corn, are omitted from the category of admissible articles, but that *bread*, and all preparations made of wheat, rye, rice, or corn-flour, are conspicuous by their absence. This is found to be a very important omission from the dietary of most persons, and very numerous have been the attempts to devise substitutes for it, with varying success. The best known and most popular of these is gluten, bread made of the so-called gluten flour, whence the starch is partially removed by washing. Unfortunately, the gluten flour made in this country contains nearly as much starch as the white flours, with perhaps a single exception,—a meal made by the Battle Creek Sanitarium Co., of Battle Creek, Mich.* In England and France patients are much more fortunate, gluten flours of sufficient purity being there obtainable. Flour of the soya bean (soya hispida), containing only four per cent. starch and a large amount of nitrogenous matter and oil, is also used for making biscuit, —a very poor substitute for bread, becoming rapidly rancid from decomposition of oil.

Another substitute is the bran-flour or unbolted wheat, which contains relatively less starch. The pure bran itself is not wholly innutritious. Dr. Prout very early recommended, as a substitute for bread, a compound of bran, milk, and eggs, which he declared not unpalatable.†

---

* This is known as gluten meal No. 1. A biscuit is made of this known as No. 1 gluten biscuit. No. 2 meal and biscuit contain more starch.

† The following are Dr. Camplin's directions for making biscuit of the bran-flour: To one-quarter of a pound of flour add three or four fresh eggs, one and a half ounces of butter, and half a pint of milk; mix the eggs with a little of the milk, and warm the butter with the other portion, then stir the whole together well; add a little nutmeg or ginger or other agreeable flavoring and bake in small forms or pattypans. The cake when baked should be about the thick-

Still another substitute for wheaten bread is the almond food suggested by Dr. Pavy. The almond is composed of 54 per cent. of oil, 24 per cent. of nitrogenized matter known as emulsin, six per cent. of sugar, three per cent. of gum, and *no starch*. Chemically speaking, it is therefore admirably adapted for diabetic food, and when the sugar and gum are extracted leaves nothing to be desired. The sugar and gum are removed by treating the powdered almonds with boiling water slightly acidulated with tartaric acid, or soaking the almonds in a boiling acidulated liquid, which may form part of the process for blanching. The boiling and acid fluid are necessary in order to precipitate the *emulsin*, which would otherwise emulsify the oil of the almond. Biscuits made of almond-flour * and eggs are palatable and may be eaten with a little dry sherry or whiskey and water.

Biscuits made of *inulin*, the starchy principle already referred to on page 765, were suggested by Kuelz.† Lichenin, or moss-starch, abundant in Iceland moss, is a variety of inulin and would be the material used for the purpose. Being very cheap, it is suitable on this account. Though a starch, it is, according to Kuelz, one of the assimilable starches already alluded to, of which small quantities, at least, do not increase the excretion of sugar. The biscuits are made with the addition of milk, eggs, and salt

The best of these substitutes is unsatisfactory, as patients soon tire of them and want the real bread. Aleuronat ‡ bread, mentioned by van Noorden as the only one at all satisfactory though containing some carbohydrate, since it retains the bread taste, I have lately commenced to use for my patients, and it promises to be fairly satisfactory.

In mild cases the dietetic measures above indicated are usually followed by the most prompt and decided results, occasionally by the permanent removal of all symptoms, in others by a continued absence of

---

ness of an ordinary captain's biscuit. The pans must be well buttered. Bake in rather a quick oven for half an hour.

These cakes or biscuits may be eaten by the diabetic with meat or cheese for breakfast, dinner, or supper; at tea they require rather a free allowance of butter, or they may be eaten with curd or any of the soft cheeses.

* Seegen recommends an almond food made as follows: Beat a quarter of a pound of blanched sweet almonds in a stone mortar for about three-quarters of an hour, as fine as possible; put the flour thus produced into a linen bag, which is then immersed for an hour and a quarter in boiling water acidulated with a few drops of vinegar. The mass is then thoroughly mixed with three ounces of butter and two eggs; the yolks of three eggs and a little salt are added, and the whole is to be stirred briskly for a long time. A fine froth is to be made by beating the white of the three eggs and added. The whole paste is now put into a form, smeared with melted butter, and baked by a gentle fire.

† Kuelz, "*Beiträge zur Path. und Therapie des Diabetes Mellitus*," Marburg, 1874, Bd. 1, p. 145.

‡ Aleuronat bread is made as follows by R. Williamson: Mix two ounces (62 gm.) of desiccated cocoa-nut powder with a little water containing a small quantity of German yeast. Make the mass into a sort of paste, and put in a warm place for half an hour or longer. The small amount of sugar contained in the cocoa-nut is almost entirely decomposed by the fermentation produced by the yeast, and the cocoa-nut paste becomes spongy. Add two ounces (62 gm.) of aleuronat, one egg beaten, and a small quantity of water, in which a little saccharin has been dissolved, and mix well until a dough is formed. Divide into cakes and bake in a moderate oven for twenty or thirty minutes. *Aleuronat* is a yellowish powder containing 80 to 90 per cent. of vegetable albumin and only seven per cent. of carbohydrates. I have found much difficulty in securing properly desiccated cocoa-nut powder.

them so long as a watchfulness over diet is maintained. In a more advanced stage of the disease, in which a more rapid emaciation and loss of strength show themselves, such a regimen is followed by a decided diminution in the quantity of sugar excreted, but it fails to disappear altogether, and a more rigid elimination of saccharine and amylaceous articles must be attempted. Sooner or later, however, a stage is reached when not only albuminous food breaks up into urea and sugar, urea and water, but the albumin of the tissues undergoes the same metabolism and excretion, and emaciation, starting first with the disappearance of fats, invades the muscular tissues. Fatty foods longest resist this breaking up, but ultimately, in progressive cases, even they increase the elimination of sugar.

In conclusion, the following classified summary of articles admissible for diabetics will be found convenient for reference :—

Shell-fish.—Oysters and clams, raw or cooked in any way, *without* the addition of flour.

Fish of all kinds, fresh or salted, including lobsters, crabs, sardines, and other fish in oil.

Meats of every variety except livers, including beef, mutton, chipped dried beef, tripe, ham, tongue, bacon, and sausages. Also poultry and game of all kinds, with which, however, sweetened jellies and sauces should not be used.

Soups.—Clear bouillon and other soups, beef tea and broth made without flour, rice, vermicelli, or other starchy substances and without the vegetables named below as inadmissible.

Vegetables.—Cabbage, cauliflower, Brussels sprouts, broccoli, green string-beans, the green ends of asparagus, spinach, dandelion, mushrooms, tomatoes, lettuce, endive, coleslaw, olives, cucumber fresh or pickled, radishes, young onions, watercresses, mustard and cress, turnip tops, celery tops, or any other green vegetables.

Bread and cakes made of gluten, bran, aleuronat, or almond flour, or inulin, with or without eggs and butter. Griddle-cakes, pan-cakes, biscuit, porridges, etc., made of these flours. No pastry permitted unless made of the admitted flours and without sugar.

Eggs in any quantity and prepared in all possible ways, without sugar or ordinary flours.

Nuts.—All except chestnuts, including almonds, walnuts, Brazil nuts, hazelnuts, filberts, pecan nuts, butternuts, cocoanuts.

Condiments.—Salt, vinegar, and pepper in moderate quantity.

Fruits.—Oranges, lemons, cranberries, cherries, strawberries, all in *very* moderate quantity.

Jellies.—None except those unsweetened. They may be made of calf's foot or gelatine and flavored with wine.

Drinks.—Coffee, tea, and cocoa-nibs with milk or cream, but without sugar. Also milk, cream, soda (carbonated) water, and all mineral waters freely; acid wines, including claret, Rhine, and still Moselle wines, very dry sherry. Unsweetened brandy, whiskey, and gin. No malt liquors except those ales and beers which have been long bottled and in which the sugar has all been converted into carbonic acid and alcohol.

Vegetables to be Especially Avoided.—Potatoes, white and sweet, rice, beets, carrots, turnips, parsnips, peas, and beans; all vegetables containing starch or sugar in any quantity.

Each case should be thoroughly studied as to its own peculiarities and demands. My plan is to place the person at the outset on a strict nitrogenous diet of broths, meat, and eggs, with a view to determining what can be accomplished. This done, successive articles of food are added and their effect upon the urine watched. Too great stringency must not be insisted upon and the presence of one per cent. or a maximum of two per cent. of glucose in urine may be permitted for a time, but semi-occasionally, say once a month, a return should be made to the strict diet with a view to take soundings, and if it is found that all the glucose goes out we may be encouraged to permit for a time a more liberal diet.

II. *Hygienic Treatment.*—Next in importance to the dietetic is the hygienic treatment of diabetes. This consists in providing perfect ventilation, bathing, and attention to the skin, together with muscular exercise.

The diabetic should breathe the freshest and purest air. While the cases are not numerous in which embarrassed respiration results in glycosuria, there are undoubted instances in which this has occurred, as in croup and whooping-cough; and it is well known that asphyxiated lower animals are apt to have glycosuria. And although the glycosuria thus resulting is probably reflex, it can hardly be expected that the diabetic should improve under unfavorable respiratory conditions. He should not, therefore, live, work, or sleep in a confined atmosphere, but secure the most perfect ventilation, spending much of his time out of doors, and sleeping in large, well-ventilated chambers, with windows open, etc. Especially should he avoid exposure to irrespirable gases. Attention to the skin, or skin-culture, is most important to the diabetic. He should bathe at least twice a week in tepid or hot water on going to bed in winter, and on rising daily take a cool sponge bath. In summer he may take a cool bath on rising and retiring. He should groom his skin thoroughly daily, either after the bath or independent of it on the days on which he does not bathe. A couple of tablespoonfuls of sodium carbonate to an ordinary bath is a suitable addition to the latter, softening the skin and facilitating its action by removing the effete epithelium.

Attention to other secretions, particularly the bowels, is of the greatest importance. Diabetics who are constipated are always more difficult to relieve. It is probably partly on account of their action in this respect that the alkaline and alkaline-saline aperient waters, as those of Vichy, Vals, and Carlsbad, are so useful. To those who visit these springs, a part of the benefit is ascribable to the other favorable hygienic influences, such as rest, fresh air, and exercise, by which they are surrounded. Independently of these influences, however, there is reason to believe that the alkaline waters are of service to diabetics; and where their cost is not a consideration, a pint to a quart of Vichy or Vals and half as much Carlsbad may be taken during the day, beginning before breakfast. The Vichy is a more alkaline water, containing 35 grains (2.3 gm.) of carbonates to a pint (0.5 liter), while Carlsbad

contains but 11 (0.51 gm.), but twice the proportion of chlorides, eight grains (0.7 gm.) to a pint (0.5 liter), and nearly ten times as much sodium sulphate, or 19 grains (1.25 gm.); hence its more purgative quality. Since Carlsbad has the highest reputation, it is more likely that it is through the action of the sulphates and chlorides on the liver rather than that of the alkalies they contain that these waters are efficient. This is the more likely, as other alkaline waters nearly as rich as those of Vichy and richer than Carlsbad in sodium carbonate, but without sulphate of sodium, are without reputation. The alkalies may, however, increase the effect, and are especially of service where there is acidity.

The celebrated Saratoga Springs in this country have an undoubted action on the liver, probably through the chlorides they contain, which are in very large proportion, reaching in the Geyser Spring 70 grains (4.6 gm.) to the pint (0.5 liter), and in the Empire and Hathorn 63 grains (4.19 gm.) to the pint (0.5 liter). They contain no sulphates, but the carbonates in considerable proportion, though much less than in the Vichy waters. Saratoga Vichy, which of the Saratoga waters contains most sodium carbonate, contains ten grains (0.6 gm.) to the pint, the Geyser nine grains (0.58 gm.). In the absence of the Carlsbad and Vichy waters I would use the purgative Saratoga waters, especially the Saratoga Vichy and Geyser.

Muscular exercise should be taken daily by the diabetic, both by walking and gymnastics. Glycogen is undoubtedly consumed in the muscles during their action, and that in diabetes there is an undue accumulation of sugar in the muscles is quite certain. It should be sustained regularly day by day, even in wet weather, care being taken to keep the feet dry, while it should never be carried to real fatigue.

III. *The Medicinal Treatment.*—Like all diseases in which treatment by drugs is relatively inefficient, diabetes has its full share of reputed remedies, most of which are useless. This dare not, however, be said of all.

The only drug that can be relied upon to produce an effect in diminishing glycosuria is opium. It seems that it was used for diabetes as early as the second century by Archigenes. It was also used by Ætius, the physician, in the fourth century, and in the latter part of the eighteenth century and beginning of the nineteenth by Rollo, Frank, Tommasson, and especially the English physician, Pelham Warren, in 1812. It is certainly a useful agent in diabetes, but its use is united with disadvantages in the locking-up of the secretions which attends it. On account of its comparative freedom from these effects, codeine has come to be the favorite alkaloid of opium in diabetes. It may be given in ¼-grain (0.016 gm.) doses three times a day, or ½ grain (0.032 gm.) twice a day, increasing ¼ grain (0.016 gm.) daily until the desired effect is produced or it proves useless. If the sugar disappears the drug should be gradually withdrawn. If constipation is caused by it, aperient remedies should be associated, and very suitable are the natural aperient waters, including the bitter waters, Friedrichshalle, Hunyadi Janos, Racoczy, Püllna, etc. I have seen a patient entirely relieved under its use, and the use of it alone, with no return of the sugar after its omission.

I rarely exceed ten grains (0.65 gm.) a day, and usually defer its use until I find other measures insufficient.

After opium, arsenic has longest maintained its reputation as a remedy in diabetes, and I use it in all mild cases, preferring Fowler's solution, beginning with three-drop doses for an adult and increasing. It seems to me there is something more than a simple tonic action in it. Possibly it acts partly on the gastro-intestinal tract and partly on the red blood discs, increasing their oxidizing power over glucose.

The bromide of potassium is sometimes efficient in diabetes accompanying functional nervous disorders due to mental over-work or psychic disturbance. Bromine and arsenic are combined in the shape of Clemens's solution of bromide of arsenic, of which the dose is three to five minims (0.184 to 0.3 c. c.).

Substances which possess the power of destroying sugar in the blood have long been sought. The alkalies, and especially the alkaline carbonates, at one time enjoyed considerable reputation in the treatment of diabetes, after Mialhe claimed for them the power of destroying the sugar in the blood, and of neutralizing the volatile acids retained within the organism in consequence of the defective action of the skin. Whatever their mode of action, the carbonates continue to be used by many physicians, both in Germany and in England, with good reason. Potassium or sodium bicarbonate, in ten-, 15-, or 20-grain doses (0.6, one, or 1.3 gm.), may be administered. The efficiency of the alkaline mineral waters is thus explained. Much was hoped of pancreas preparations, especially since the brilliant results that followed the use of thyroid extract in myxœdema. As yet they have proved disappointing. The glycolytic ferment isolated by Lepine from the pancreas and from malt diastose has not been thoroughly tested.

The coal-tar derivatives, antipyrin and antifibrin, and phenacetin have been highly recommended by the French physicians, and I have found them of service in mild cases, giving ten to 15 grains (0.6 to one gm.) three times a day on an empty stomach. Their efficacy is said to be increased when combined with an equal bulk of sodium bicarbonate. Salicylate of sodium has warm advocates, and in gouty cases it is likely to be useful. According to van Noorden, it is especially in neurogenous diabetes that the last of the remedies just named is useful, quieting the irritability of the central nervous system.

Iodide of potassium has produced some striking results in the case of diabetes due to syphilitic lesions of the brain.

Ergot is occasionally useful. Lactic acid was strongly advocated by the Italian physicians. Cantani recommends that from 75 to 150 grains (five to ten gm.) of the acid should be taken daily in from eight to ten fluidounces (240 to 300 c. c.) of water.

Cod-liver oil becomes a useful remedy where the carbohydrates are totally converted into sugar and excreted, the albuminoids for the most part, while the body albumin is being encroached upon as a source of energy. Especially useful does it become when associated with alcohol in the shape of whiskey or brandy, which always helps the assimilation of fat. In the same category as cod-liver oil must be placed butter, cream, bacon, and the like.

Treatment of Complications.

Eczema and Pruritus.—These sometimes intensely annoying symptoms commonly abate with the reduction of the glycosuria, but require also other measures. Scrupulous cleanliness is in the first place necessary, accomplished by warm, tepid bathing. In addition, we may use solutions of boric acid or sodium hypophosphite, one ounce (31 gm.) to a quart (liter) of water. Zinc ointment or ointment of acetate of lead, corrosive sublimate solutions, very weak,—one to 3000,—and tumeral sulphone may be used, one to 20. As a last resort in pruritus, nitrate of silver may be used in the strength of 20 grains (1.3 gm.) to the ounce (30 c. c.), making daily applications, and though sometimes painful, they are ultimately effectual.

Diabetic Coma.—Treatment is here usually futile. The alkalies and alkaline mineral waters should be pushed. Intravenous injections of alkaline solutions have been disappointing. More hopeful is intravenous injection of a 0.6 per cent. salt solution, as recommended by van Noorden, using a liter in four doses at intervals of four hours. Hypodermoclysis, which is much easier, will accomplish the same result, as I can attest from personal experience. Copious diuresis follows, and may be expected to carry out noxious substances.

More hopeful is a *prodromic* treatment of diabetic coma, called for when diacetic acid or oxybutyric acid and large amounts of acetone are found in the urine. Under these circumstances it seems conceded that whatever be the form of diet in use at the time it must be changed, and if it be remembered that these substances are now conceded to arise from the disintegration of body albumin and not from food disintegration, as formerly supposed, diet would at least seem a matter of indifference under the circumstances, while a change of itself seems desirable. The patient should be immediately placed upon alkaline treatment, associated with the free use of alkaline mineral waters. Thus 20 grains (1.3 gm.) of sodium bicarbonate may be given every three hours dissolved in eight ounces (250 c. c.) of Vals or Vichy water. The bowels should be kept freely moved and alcohol in the shape of whiskey or brandy should be freely given.

DIABETES INSIPIDUS.

Definition.—Any excessive secretion of non-saccharine and non-albuminous urine which has continued for a long time.

The condition, unlike diabetes mellitus, affects more frequently younger persons, being rare in those over fifty years of age, relatively frequent in infancy, and most common between the ages of twenty and thirty.

As to sex, it is much more frequent in males than females, two to three times as many of the former being affected as the latter.

Etiology and Pathology.—As to causes, the same uncertainty prevails as with diabetes mellitus. An examination of cases shows an association with a certain number of conditions, such as cerebral disease, including tumor of the brain, meningitis, paralysis of the 6th nerve, sun-

stroke, cerebro-spinal fever, blows on the head and falls, exposure to cold and drinking cold fluids, drunkenness, pregnancy, hysteria, emotion, especially fright, hereditary influence, syphilis and previous disease, etc., but this does not show causation. The proportion, however, of cases in which the condition is associated with brain diseases and injuries to the head, taken in connection with the fact of Bernard's discovery that puncture of the floor of the 4th ventricle above the diabetic centre produces increased secretion of urine without glycosuria, makes it very likely that central nervous irritation, however induced, is at the bottom of the symptom. It is reasonable to suppose, too, that diabetes insipidus may be the result of some irritation, direct or reflex, of this centre in the medulla oblongata, or of the sympathetic ganglia in the abdomen. The latter explanation also applies to cases of polyuria attending the presence of abdominal diseases, such as tumor, aneurism, or peritonitis, though it is doubtful whether these are really cases of diabetes insipidus.

The essential morbid anatomy of diabetes insipidus would be the lesions of the nerve-centres or sympathetic ganglia which may underlie the symptoms. But as these are often undiscoverable, or at least indefinite, it is impossible to describe them.

Associated nervous lesions, when present, are found more frequently in the vicinity of the base of the brain.

Symptoms.—The *enormous secretion of urine*, of almost spring-water-like clearness and of specific gravity often as low as 1003, is the most conspicuous symptom, but more annoying, probably, is the *extreme thirst* which always attends it. These may be said to include all the essential symptoms, others which may or may not be present being rather their consequence. Very constantly among them are *dryness of the skin* and *absence of perspiration*. The health is otherwise often perfect. Occasionally there are derangements of digestion, and sometimes also the appetite is large, as in diabetes mellitus, though less frequently.

These symptoms may occur suddenly in the midst of apparent health, or they may supervene upon others or be substituted for them, chiefly of a nervous character, which may be the result of the nervous lesion causing the polyuria. Such symptoms are headache, restlessness, irritability, sleeplessness, what is commonly called *nervousness*, more rarely convulsions, delirium, paralyses—indeed, any one or more of the great variety of symptoms which result from organic or functional nervous disease. Sometimes these symptoms succeed upon the polyuria or are increased by it. It is certain that the milder nervous symptoms are sometimes the result simply of the inconvenience and annoyance caused by the two cardinal symptoms, polyuria and thirst. The patient is kept busy, as it were, night and day, in passing water. It is not surprising that such a patient should be fretful and irritable, and that sooner or later his health should be broken if the symptoms are not relieved.

In addition to the symptoms detailed, there are said to occur at times dryness of the tongue, epigastric and lumbar pains, diarrhœa, debility, impairment of mental faculties and of the sexual function. The debility is sometimes extreme. In some instances there is the most extraordinary tolerance of alcoholic drinks, while at others there is an exaggerated sus-

ceptibility to their influence. A very slight lowering of the body temperature has been observed, amounting, however, to but a few tenths of a degree, and is never below 97° F. (36.1° C.). In advanced stages of the disease œdema of the ankles sometimes occurs.

The *duration* of the condition is very various. Sometimes it continues through life with no inconvenience except that from the constant diuresis and thirst. Dr. Willis records a case lasting fifty years. On the other hand, it is seldom of brief duration; indeed, there is needed a certain chronicity in order to admit it in the category of diseases. One case is reported as terminating fatally in seven weeks. Under prognosis will be found some further information as to duration, but it may be said, in general, that most cases which terminate unfavorably and most which recover completely do so within a year. I have now under my care a lad of fourteen who has been under treatment for four years.

It has *no complications* except such as are its cause or its results. Among the latter is occasionally dilatation of the pelvis of the kidney, and atrophy of the latter is mentioned, due to pressure of the accumulated urine and resulting in a sacculated condition of the organ. Its symptoms are almost always influenced and sometimes even cut short by intercurrent disease, especially of a febrile character, or even by a profound physical impression, as long-continued suppuration after a blister. The boy referred to was an aggravated choreic before he became diabetic.

Physical and Chemical Characters of the Urine.—As to the quantity of urine passed, it is enormous, exceeding often the amount passed in saccharine diabetes. As many as 43 liters (90 pints) are recorded by Trousseau, and one-fourth this quantity is common. It has been said, even, that the quantity secreted sometimes exceeds that ingested, but this is impossible for any length of time, unless water is absorbed from the atmosphere, which is not likely. In point of fact, the water excreted is always a little less than that ingested, either as drink or in the solid food. As the quantity of urine excreted increases or its normal acidity diminishes, its color disappears and its specific gravity declines. In one case under my care the specific gravity was scarcely 1001, while the urine in moderate bulk was absolutely colorless. Again, a faint greenish tinge is exhibited by the urine in bulk.

As to the other constituents of the urine, it may be said in general that they are all increased, except possibly uric acid. Thus the urea is increased to three and even four times its normal amount. In a case reported by J. M. Da Costa * the urea was diminished.

Sulphuric and phosphoric acids are both increased, and especially, according to Dickinson, the combination of phosphoric acid with the earths, lime, and magnesia. The same is true of the *chlorides*.

Of abnormal constituents, inosit has been found, and *albumin* very rarely, but care should be taken not to confound the polyuria with small albuminuria of a contracted kidney with an albuminous polyuria in which there is no organic disease of the kidney.

* Transactions of the College of Physicians of Philadelphia, 3d series, Vol. 1, 1875, p. 139.

Some of the accounts published as to the quantity of water consumed and excreted are almost incredible, yet they seem well authenticated. In illustration may be mentioned the following instances from Dr. Willis's work on "Urinary Diseases:"* An artisan fifty-five years old had had constant thirst with commensurate diuresis since he was five years of age. From the age of sixteen he had drank, on an average, no less than two pailfuls daily. While in the Hôtel Dieu, to which he was admitted for an injury of the knee, he drank on an average 33 pints of water every day, often swallowing two liters, or about two quarts, at a draught. He passed daily about 34 pounds of urine and one pound of fæces. He otherwise enjoyed good health, and was the father of several children. The long duration of this case and the otherwise excellent health enjoyed by him is by no means exceptional. Very little serious disturbance seems to result as long as water is supplied to quench the resulting thirst. In extreme cases patients have even been known to drink their own urine.

An extraordinary flow of *saliva* was met in one instance by Kuelz† along with polyuria in a hysterical girl of eighteen years, from whom as much as 17.72 ounces (525 c. c.) were collected in twenty-four hours, while the quantity ranged during four months from 360 to the former amount. The quantity of urine passed during this time ranged from 200 to 260 ounces (6000 to 7800 centimeters). The increased flow of saliva may be explained by the fact that in some of the experiments of Eckhard,‡ Loeb,§ and Gruetzner‖ puncture of the medulla oblongata was followed by ptyalism.

Diagnosis.—The diagnosis of diabetes insipidus is very easy. The persistent thirst, polyuria, and absence of sugar from the urine are pathognomonic. The only possible error is mistaking the polyuria of chronically contracted kidney of interstitial nephritis for that of diabetes insipidus. In addition, however, to the fact that a careful examination for albumin will discover it in the urine of contracted kidney, the quantity is never so large, nor is the thirst extreme; so that it would seem only necessary to mention the possibility of such an error in order to avoid it.

Prognosis.—It is extremely rare for a case of diabetes insipidus to terminate unfavorably unless there have been also present symptoms pointing to serious nervous lesion. Recovery is not infrequent. According to Dr. Roberts, of 67 cases collected, 16 are reported as complete recoveries and 14 ended fatally, nearly an equal proportion. The remaining 37 were still in progress. In cases of recovery or death, the duration is comparatively short. Of the 16 recoveries, in nine the duration was less than a year; in one, four years; in two, eighteen and nineteen years, and in the remainder some years. Of the 14 fatal cases, nine terminated in less than a year, one *in seven weeks*, two in two months;

* American edition, Philadelphia, 1839, p. 23.

† "Diabetes Mellitus and Insipidus." Marburg, 1875.

‡ Eckhard, "*Beiträge zur Anat. und Physiol.*," IV, p. 191.

§ Loeb, Eckhard's "*Beiträge*," V, p. 1; and "Dissertation," Giessen, 1869.

‖ Gruetzner, *Pflüger's Archiv*, VII, p. 552.

the other two survived for periods varying from eighteen months to twenty years. Of the 37 cases in progress only five had continued for a year or under. The remainder had continued for periods ranging from something over a year to fifty-nine years.

These results seem to be tolerably independent of treatment. It may be said, therefore, that, as a rule, cases that last more than a year are apt to continue, but ordinarily only require to be furnished with an abundance of water to keep them tolerably comfortable. According to Dr. Dickinson, cases due to drunkenness are very apt to run a severe and rapid course, usually terminating fatally within a few months, and one terminated thus in two months.

The disease appears to me altogether less serious than diabetes mellitus, and I quite concur with Senator, who says "it is rather a troublesome than a dangerous complaint." But Trousseau and Da Costa are inclined to consider it more serious than diabetes mellitus.

Treatment.—The treatment of diabetes insipidus would naturally resolve itself into the treatment for the disease of which it is the symptom than of the symptom itself; but as the former is very frequently undiscoverable, it must consist mainly of efforts to diminish the secretion of urine, and with it the thirst.

First, it is generally conceded that there should be no restriction in the drinking of water or other harmless fluids, for the diuresis is not so much caused by the large ingestion of water as the thirst is caused by the diuresis. It should be mentioned, however, that one or two instances are reported wherein improvement seems to have resulted from such restriction. And if, as in some cases, a habit of drinking has been the initial event, moderate restriction may be reasonable. Caution should be used in the administration of drugs, though my experience is not that of Dickinson, who says that "remedies designed to restrain the urinary secretion seldom fail to do harm." The usual remedies are ergot, opium, gallic acid, and valerian; of all the doses ultimately used are usually large. I have found the symptoms to subside in one case under the use of gallic acid after I had failed with full doses of ergot. In another, probably due to syphilis, the effect of the iodide of potassium was shown in an aggravation of the symptom whenever it was discontinued and an amelioration when it was resumed.

Trousseau and Rayer claimed extraordinary results from the use of *valerian*, the former using the extract in enormous doses—2½ drachms (6 c. c.) a day, which was increased to one ounce (30 c. c.) daily in one instance. Rayer used the powdered valerian and the valerianate of zinc, giving the latter in pills in gradually increasing doses until 20 grains (1.25 gm.) a day were given. At the present day the more palatable elixir of valerianate of ammonia combined with bromide of potassium is to be preferred.

Reasoning from the effect of intercurrent disease and powerful physical and nervous impressions, Roberts suggests a large *blister* at the nape of the neck or epigastrium, according as the associated symptoms and the anamnesis point to the nervous or digestive system, a suggestion which may be acted upon with advantage.

The constant galvanic current has been recommended, and in cases of spinal lesion may be expected to be of advantage. Seidel and Kuelz have both used it with good results. The former applied one pole of a "strong battery" over the loins near the spine, and the other as deeply as possible over the hypochondrium, upon each side daily for five minutes. In eight days the urine fell from 195.9 oz. (5957 c. c.) to 153.3 oz. (4600 c. c.) per diem, in three weeks to 76.6 oz. (2300 c. c.), and the next month 63.5 oz. (1904 c. c.), while the weight of the body increased nine pounds. Kuelz applied one pole of a battery of 30 to 40 cells as high as possible in the nape of the neck, and the other to the loins or epigastrium, the best results being apparently obtained with the positive pole to the nape of the neck, and the negative first to the loins for four minutes, and then to the pit of the stomach for four minutes.

Tonics and nervines, such as strychnia, iron, arsenic, salts of quinia, cod-liver oil, etc., are appropriately added to the treatment with a view to sustaining the strength of the patient, which is apt to give way. To these are to be added fresh air, sea air, exercise, and all possible favorable hygienic influences.

Hygiene is even more important than in diabetes mellitus, and should include a careful attention to the skin, warm clothing, warm baths, frictions, etc., in order to divert a portion of the circulation from the kidneys to the skin. The thirst should also be quenched when possible, by bits of ice and acidulous fluids.

OBESITY.

Synonyms.—*Adipositas universalis; Polysarcia adiposa; Corpulence.*

Definition.—Obesity may be defined as an inconvenient accumulation of adipose tissue in the body.

Etiology.—The most usual cause of an excessive accumulation of fat is doubtless over-eating, and though it may be true of some of these cases that they are really small eaters, careful examination will generally prove that they are liberal eaters. Heredity exerts an undoubted influence, and we find corpulence running in families. Commonly it does not make its appearance until after thirty-five years of age, but in this country particularly it is often seen earlier, in boys and girls of ten years and upward.

Of foods, each one of the representative varieties, albuminous, carbohydrates, and fats, is capable of contributing fat deposited in fat vesicles in the body, and it has even been said that albuminoids furnish more of the fatty tissues of the body than the carbohydrates. Certain it is that a person may become corpulent who eats very little fat. In most cases, however, corpulent persons are found to be liberal consumers of all three of the food elements. While the carbohydrates are direct sources of fat, it is generally conceded that they act largely by sparing the fats derived from other sources. They decompose and oxidize so rapidly and thus give themselves up so readily to force production that the stored fats are not called upon. Thus it is that sugars and starches indirectly favor corpulence. To this class belong also alcohol, and especially beer, which

contains over five per cent. of carbohydrates in addition to three to four per cent. of alcohol, and it is well known that liberal beer drinkers furnish a large quota of fat men. A second way in which large quantities of alcohol contribute to adiposis is by hastening albuminous metamorphosis, setting free non-nitrogenous substances readily converted into fat, which is deposited, among other situations, in the liver, giving rise to the fatty liver so constantly found in drunkards.

Another cause of corpulency is muscular inactivity. Fat is consumed by muscular contraction and its absence must contribute to fat accumulation, and one need not go far to see its evidence in many who lead lives of idleness. Oertel has especially called attention to the fact that a simple diminished ingestion of fluids without other changes in the diet will reduce the amount of fat. The effect may be brought about in two ways: first, by diminishing the work of the heart and thereby favoring oxidation; second, by an effect, which is not so much the diminution of fat as a withdrawal of water,—a sort of "desiccation," it is called by Strümpell.

The subjects of anæmia and chlorosis often become fat, probably because of defective oxidation, growing out of a diminished supply of oxygen, which the crippled corpuscles are unable to carry in sufficient quantity.

Sexual continence probably contributes to corpulence, since eunuchs are well known to grow fat, and both women and men are disposed to grow fat when the sexual function begins to abate. Finally, corpulence itself favors the further accumulation of fat, first by interfering with the muscular activity of its subject and therefore with the oxidation of fat, and again diminishing combustion by reason of a reduced demand for heat, the fat itself, by preventing the radiation of heat, causing a lessened want of it.

Symptoms.—A description is scarcely needed of the anatomical condition which constitutes obesity. The round, plump face, the double chin and hanging cheeks, the enormous girth of body, the pendulous belly and elephantine arms, legs, and thighs need no further description. The labored, waddling gait is often conspicuous. The first evident indication of harmfulness due to corpulence is an increased frequency in the breathing rate, at first on slight exertion and later independently of it. This is in part a true cardiac asthma,—due, first, to the fact that the heart cannot push the blood through the lungs rapidly enough to permit its aëration at the ordinary breathing rate; and, second, to the fact that the motion of the lungs is also restricted. The latter is due to the accumulation of fat over the thorax and in the mediastinum, and the accumulated intra-abdominal fat and probably enlarged liver, which interfere with the proper descent of the diaphragm. This leads at first to cardiac hypertrophy, further stimulated by the extra work demanded of the heart on account of the increased bulk of the blood, the resulting arterio-sclerosis, and the venous movement due to limited muscular activity. Later the fatty infiltration of the muscular walls of the heart leads to further embarrassment in its action and impairment in its nutrition, whence come cardiac weakness, ultimate failure, with œdema, pericardial and pleuritic effusion, and sometimes sudden death.

The pulse, hard to reach, is usually frequent, but may be slow and irregular. The heart can be examined with difficulty on account of the large accumulation of fat, and the normal sounds are feeble and distant. The situation of the apex can be found only by the aid of the stethoscope. Intertrigo is often an annoying symptom and great care is required to avert it. Interstitial nephritis may be superadded. By no means all corpulent persons run this course. Many lead lives of considerable comfort.

Treatment.—This consists in acting upon two principles: first, furnishing less food to oxidize, and, second, increasing the oxidation of the fat in the body.

The first is accomplished by cutting down the quantity of all kinds of food, but especially carbohydrates. Sugar should be prohibited altogether, bread may be allowed in small amounts, say two ounces well toasted and with it a thin layer of butter. Or hard biscuit may be substituted. A single egg may be permitted at breakfast or luncheon. Meat may be allowed once a day. It may be of any kind and with it may be taken green vegetables, such as peas, string beans, tomatoes, cabbage, spinach, Brussels sprouts, lettuce, celery, and the like, omitting altogether rice, potatoes, and the farinæ in general. A little cheese may be allowed.

Only small quantities of fluid should be permitted at meals, just enough to aid in the solution and digestion of food. This may be tea, coffee, water, or skimmed milk, the first two without sugar or cream. Beer, porter, and sweet wines should be prohibited, but a glass or two of hock or claret with an alkaline mineral water may be allowed.

A diet of skimmed milk only is a sure way of reducing fat, and a start may be made with it, commencing with two ounces every two hours and increasing until six to eight ounces are attained. Unfortunately, very few persons will bear this treatment for any length of time, but, as stated, a beginning may be made with it, and when the patient tires the other treatment just described may be instituted.

The second indication to promote oxidation is accomplished by exercise, by gymnastic work, walking, mountain climbing, or cycling. The last has done good work in reducing the weight of the corpulent, and if combined with a proper diet may be expected to do more. Massage is also useful. The Turkish bath and steam bath may be associated and help also in the "desiccation" of the body, which in turn facilitates oxidation. One of the greatest difficulties is in getting the patient to assiduously carry out the dietary and exercise.

Certain health resorts have much reputation for their efficiency in reducing corpulence. Carlsbad is one of the most celebrated of these, and I have seen many patients return thence markedly improved in all the symptoms which come from obesity. The effect is probably altogether due to the strict diet, the systematic exercise, and the laxative effect of the waters.

Thyroid extract has been administered with reputed advantage in obesity in doses of from three to five minims (0.184 to 0.3 c. c.) three times daily, which may be gradually increased to 20 (1.3 c. c.) with the same precautions as advised in the treatment of myxœdema.

RICKETS.

SYNONYM.—*Rachitis.*

Definition.—"There is a disease of infants called the rickets, wherein the head waxeth too great, whilst the legs and lower parts wane too little" (Thomas Fuller, 1608–1661). This quaint description of the celebrated English chaplain, written over two hundred and fifty years ago, remains so nearly correct at the present day that I cannot forbear adopting it. "The rickets" was evidently known for some time by the laity before it received its description by F. Glisson in 1650.

Etiology.—Rickets rarely begins before the child is six months old or after eighteen months, though a late form is described by Sir William Jenner coming on as late as the ninth or even twelfth year. On the other hand, it may begin early in life, or the child may be rickety in the womb. Yet it cannot be allowed that rickets is hereditary in the usual sense of the term. The child may become rickety in the womb if the mother is feeble, under-fed, and over-worked while carrying it and if the father is weak at its conception, but not because the father or mother were rickety when children. On the other hand, certain races tend to be rickety, especially the negro and Italian. Bad air and bad food, with absence of sunlight, exposure to dampness and cold, are more potent factors, and it is likely that a defective composition of the breast milk, including a deficiency in the phosphates, is the stronger. Prolonged lactation may contribute to this. It is a disease of the city rather than the country, and of the Continent of Europe rather than America. Vienna, London, and Paris are fertile soil. In the first-named cities 50 to 70 per cent. of all children brought to the clinics are said to be rickety. Parrot held that congenital rickets was a form of syphilis, basing this view on studies in the French capital. This is not conceded. On the other hand, there is reason to believe that the changes in prenatal rickets are not identical with those of the postnatal form. The subjects of the former are usually still-born, are short of limb, and though the curves of the bones are exaggerated, there is no proliferating zone of cartilage between the epiphysis and apophysis, whence the term *achondroplasy* suggested by Parrot, and *chondrodystrophia fœtalis* by Kaufmann. Boys and girls are equally affected with rickets.

Morbid Anatomy.—This shades somewhat into symptomatology, and the two can scarcely be separated. The changes are mainly in the bones, more particularly the skull, the long bones, and the ribs. The first may escape if the disease sets in after the middle or end of the second year. The frontal and parietal eminences are exaggerated while the top of the head and the occiput are flattened, the whole effect being toward converting the head into a square box shape. The fontanelles remain open long, until the second or third year of life, while the edges of the bones where they come together to form the sutures are thickened, though soft and yielding. In addition to these changes, or instead of them, there may be large areas of delayed ossification in the parieto-occipital regions, producing yielding spots and constituting the so-called

cranio-tabes of Elsässer, but as cranio-tabes occurs in connection with syphilis and other wasting diseases in infants a few weeks old exhibiting no other sign of rickets and in new-born infants, it cannot be regarded as pathognomonic.

In the long bones, such as the radius and ulna, swelling of the cartilage between the epiphysis and shaft is apparent, seen also on section, when the cartilage is found soft, irregular, and thickened, and corresponding to this also the line of calcification is irregular. Correspondingly ossification is also imperfect, the resulting bone being soft and spongy and the periosteum loosely attached. Hence the long bones bend easily, especially the tibiæ, producing the characteristic bow-leg, which may occur before the child walks, when it is caused by sitting cross-legged. The thighs may also become bowed, the inner ends of the condyles prolonged downward and the tibia set outward, producing the "knock-knee." This does not, however, appear until the child begins to walk. In extreme cases the long bones may fracture. Sometimes both the femora and tibiæ are bowed forward.

Quite as characteristic are the changes in the chondral ends of the ribs and the shape of the chest. The former are enlarged and nodular at the junction with the bone, producing the well-known beaded appearance, which may often be recognized at a glance. The altered shape of the chest walls, most marked in children who have had much cough, is due to the yielding of the soft costal ends of the cartilages and a falling in of the ribs at these points, while the sternum and cartilages are pushed forward, as seen in Fig. 57.

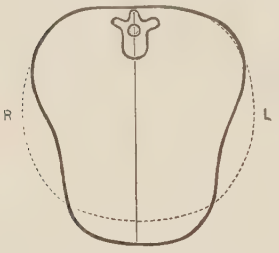


FIG. 57.—RICKETY CHEST. Dotted line indicates the shape of the chest of an infant about the same age. (*After Gee.*)

This is especially the case in the region between the 4th and 8th ribs, which is often so bent in as to form a vertical groove increased during inspiration. Associated with this is sometimes a transverse groove known as Harrison's groove, starting at the ensiform cartilage and passing transversely outward toward the axilla. At the same time the arch of the ribs below may be widened and the belly thrown forward by the arching inward of the vertebræ. Extreme degrees of this chest deformity produces the prominent sternum constituting the "chicken-breast" or "keel-shaped" thorax. Other changes in the bones are an exaggeration of the normal double curve in the clavicle, a bending of the humerus usually at the insertion of the deltoid, sometimes produced by lifting the child by the arms; the radius and ulna may be curved and twisted, the articulations knotted and bulbous, loose and mobile, because of relaxed ligaments. The spine is also often altered, the change being for the most part an increase in the normal curve outward in the cervico-thoracic portion and inward at the lumbo-sacral. In other cases there is a lateral curvature. The scapula is often thickened. The pelvis is distorted and twisted, the antero-posterior diameter markedly lessened. The rickety pelvis is one of the well-recognized causes of dystochia. These

changes are all the result of mechanical causes, such as the weight of the body or muscular traction.

Chemical analysis of rickety bones approximately reverses the normal proportion of organic and mineral constituents (calcium salts), reducing the latter to 35 per cent., while the gelatinous or organic matters amount to 65 per cent. Minute examination recognizes in the spongy spaces in the bone numerous cells. Owing to the rapid proliferation of cartilage cells, resulting in a broad band of jelly-like material between the cartilage and the bone, a spongy structure is rapidly built up, deficient in strength and stiffness. Beneath the periosteum the same gelatinous material is deposited, and a spongy tissue is formed instead of normal bone. The studies of Kassowitz lead him to believe that a hyperæmia of the periosteum, the marrow, the cartilage, and the bone itself is the fundamental condition responsible for the abnormal development. His views may be regarded as a refinement and development of those originally suggested in 1650 by F. Glisson, who held that an excessive vascularity was at the bottom of the changes.

An enlarged liver and spleen are usually present, and sometimes also the mesenteric glands are enlarged.

Symptoms.—(See also Morbid Anatomy.) The earliest symptoms noted are not invariably the same. Profuse sweating, especially about the head and neck, mild degree of fever, as the result of which the child is prone to throw off the bed-clothing, and evident discomfort in being handled are among the first. The last is apparently due to a tenderness of the skeleton, causing pain when the child is raised or danced up and down after the manner of amusing children. Along with these are the less distinctive symptoms of indigestion, indicated by nausea and vomiting, offensive stools containing partly-digested milk, and flatulent distention, causing the belly to be prominent.

Concurrently it is noted that teething is delayed, and we have the authority of Sir William Jenner that if there are no teeth at nine months there is something rickety about the child. But I am confident it has happened to me to see dentition delayed after this time in children who were not and who did not become rickety. Moreover, the teeth which are cut soon decay. Muscular weakness is characteristic, so that the child cannot sit up and makes feeble or no effort to walk. Such muscular weakness has been mistaken for paralysis, whence it has been called the pseudo-paresis of rickets. Close upon these symptoms, or at least within two or three weeks of the first symptom, follow the skeletal changes described under morbid anatomy, p. 779. The head is large in comparison with the face, the skin is pale and thin, and the little child has often an old and a wise look quite beyond its years. The appearance of the beaded ribs, the bowed legs, or "knock-knees," prominent belly, and curved spine often serve to make the diagnosis easy at a glance. The prominent belly requires some further allusion, varying somewhat at different periods. Before the child walks the normal cervical anterior curve may be increased and a posterior curve present from the 1st dorsal to the last lumbar vertebra, which may be recognized by holding the child up. After it begins to walk, however, the dorsal spine becomes

curved backward and the lumbar forward. The latter therefore contributes also to the prominent belly produced in part by flatulent distention, and partly at times by an enlarged liver and spleen.

Among other less essential symptoms may be mentioned nervousness, restlessness, peevishness, and infantile convulsions, the relationship of which to rickets is not accidental and was pointed out by Jenner. Tetany and laryngismus stridulus are also often symptoms.

Complications.—These include especially bronchial catarrh and broncho-pneumonia, the effects of which are aggravated by the conformity of the chest, the weakness of the ribs, and feebleness of the respiratory muscles. Collapse of the lung is often a consequence of lung affections. Chronic hydrocephalus is a complication, while many of the conditions mentioned under symptomatology, viz., diarrhœa, convulsions, laryngismus stridulus, and the like may also be so regarded. The rickety child is weak and vulnerable to all the illnesses of childhood.

Diagnosis.—This is usually easy, although, of course, all the symptoms detailed are not always present in their typical expression. The various spinal curvatures may be somewhat confusing. Thus the question of caries may arise. But the rickety spine differs from that of caries by the large curve, the absence of angularity, the flexibility of the spine, and the fact that by laying the child flat on its face the curve disappears. The other symptoms of rickets are also present. The lordosis of rickets produces a deformity resembling that of congenital dislocation of the hips and of hip disease, but here again other signs of rickets are present, while the distinctive signs of the disease in question are absent.

Prognosis.—Rickets is never in itself fatal, and the course is toward recovery. But the child is always in danger from the complications. Such are bronchitis, broncho-pneumonia, laryngeal spasm, and convulsions. Walking is always delayed, and the child may be still unable to walk at the end of the second and third year. Allusion has been made to the fact that the rickety pelvis in women is one of the most frequent causes of difficult labor.

Treatment.—We should seek to avert rickets by a judicious prophylaxis, which consists in keeping the health of the mother at the highest point at all times. This, however, not by organic food only, but by a judicious admixture of salts such as are contained in the whole cereal grain, especially wheat and barley. Frequent pregnancies and prolonged nursing, being acknowledged causes, should be regulated.

The treatment of the child should be dietetic, hygienic, and operative or mechanical. As the condition depends often upon the absence of ordinary good food, the simple addition of such food in lieu of the mother's milk, if this be found defective, may be all that is required, especially if it be possible to secure that rarely attainable article, a healthy wet-nurse. In the absence of this, beef juice, the yolk of eggs, peptonized milk, and beef peptonoids may be substituted. Due consideration must, however, be paid to digestion in the selection of food, the stools should be daily examined, and if undigested residue is found the food should be changed. Pepsin and hydrochloric acid in doses adapted to the age of the child should be given, while the predigested foods are often highly

useful. Cod-liver oil inunctions are invaluable, and though in some respects unpleasant, I have seen so many children seemingly wrested from death by their use that I value nothing more highly. Saccharine and starch foods must be avoided except in very moderate quantities. The flours of the whole cereals well baked and cooked as thin gruels and strained make a suitable addition to the food, while the fruit juices of orange and lemon may be given in small quantities. Medicines should be cautiously given. Among them are lime salts, as the hypophosphite of calcium or lactophosphate of calcium, ten grains (0.65 gm.) of either three times a day, or lime water, or the syrups of the above-mentioned salts in doses of a few drops only, as the stomach must be carefully protected. Minute doses of iron, preferably the citrate or malate, may be given. Phosphorus was recommended by Kassowitz and is endorsed by Wegener, Jacobi, and Strümpell in doses of $\frac{1}{200}$ to $\frac{1}{100}$ grain (0.00033 to 0.00066 gm.) two or three times a day dissolved in olive oil or cod-liver oil. The principle of the administration of these two drugs is different. The salts above mentioned are convenient modes of administering calcium, while phosphorus is supposed to stimulate *bone growth.*

The hygienic treatment is more important than the medicinal. Fresh air and out-door life are indispensable. If the child is warmly clothed and protected, it may be taken out even in cold weather. It should not be allowed to walk or even to sit up unless properly supported—in fact, should be handled as little as possible. After ossification is complete, deformities may be corrected by the orthopædic surgeon.

OSTEOMALACIA.

Definition.—A softening which takes place in the bones by a solution of lime salts subsequent to their complete development.

Etiology.—The precise cause is unknown. A geographical distribution, however, exists, in accordance with which it is common on the Rhine, in Westphalia, in Eastern Belgium, and Northern Italy. In this respect it is similar to goitre, which prevails in special localities, and it has been suggested on this account that it may be due to some local cause. It is for the most part a disease of adults between thirty and forty years old, and of women more than men. It is favored by unhygienic surroundings, such as damp and badly-ventilated dwellings. Frequent pregnancy is supposed to be an exciting cause.

Pathology.—There is primarily increased vascularity. To this succeeds a solution and disappearance of the lime salts of the bone. This takes place from within outward, from the marrow cavity with its lacunæ and canaliculi, dissolving out first the lime salts and then melting away the matrix, enlarging the central cavity until the cortical portion acquires a paper-like thinness. The whole bone has been compared to an "inflated and dried intestine." The product of the solution at first is a mucoid matter which mixes with the marrow, which soon loses its vascularity and gradually acquires a thinner but still viscid character and yellow color.

The periosteum is likewise hyperæmic and at first thickened. The process is compared to the artificial solution of the earthy salts from bone by hydrochloric acid, and it is supposed that the solvent agent exerts its effect from the medullary spaces and Haversian canals. It has been suggested that lactic acid is the solvent, as it has been found in the medullary canal and in the urine of subjects of osteomalacia. The process extends unevenly. It differs from rickets in being a degeneration of fully-formed bone, while the latter is a degeneration of developing bone.

Morbid Anatomy.—The favorite seats of the process are the vertebræ and the bones of the pelvis and thorax. The result in the former is an S-like curve of the spinal column, due to a kypho-scoliosis or backward curvature of the dorsal and a lordo-scoliosis or forward curvature of the lumbar part, while the cervical portion in connection with the upper spinal portion protrudes anteriorly. The thorax is distorted and compressed laterally, while the sternum is prominent and bent. The pelvis is also compressed laterally, the symphysis projects like a beak, and the sacrum projects forward, producing a deformity of the pelvis often discoverable only to internal examination.

Symptoms.—The first symptom is usually *pain*, deep-seated and severe, oftenest in the sacral region, nape of the neck, back, and thighs, and this pain is persistent and increased by motion. There is also *tenderness.* Walking, therefore, becomes more and more difficult and finally impossible and the patient takes to bed. But this affords no relief, the pain being kept up by the pressure of the bed-clothing and the weight of the body. In the meantime the deformities described under morbid anatomy take place, though those of the pelvis are less obvious externally. Difficult labor is an inevitable consequence should the patient conceive, just as in rickets. Dyspnœa is a frequent consequence of compression of the lung by the distorted thorax. Fractures, complete and incomplete, are frequent events, even of the ribs as well as of the extremities. In this respect osteomalacia differs from rickets, in which the bones bend but do not break. Such fractures repair imperfectly. Sometimes, on the other hand, the limbs are soft and yielding, and may be bent like lead pipe. The bones of the head and face are for the most part exempt, though the head is much bent toward the chest, making the stature less.

The general condition of the patient often remains for a long time unaltered. There is little or no fever. The organic functions are normally maintained. The presence of lactic acid in the urine has been mentioned. It is said that phosphoric acid is diminished. Albumin is also sometimes present. Calcareous concretions have been found in the kidneys and bladder.

Diagnosis.—At first there may be doubt as to the nature of the disease, but as the characteristic symptoms present themselves its real nature becomes evident. Disease of the vertebræ and cord has been confounded with it, but the hobbling gait peculiar to it does not usually resemble any of the gaits of spinal disease. Being a disease of adults, it is not likely to be mistaken for rickets. Moreover, it is a disease which affects the shafts of bones rather than the epiphyses.

Prognosis.—The disease is usually ultimately fatal, although death is often long deferred and the course is a chronic one, from two to three to five and ten years. Arrest sometimes occurs, but is only temporary. The disease again starts and its course is generally irresistible. Death commonly takes place from exhaustion or from some complication like pneumonia. Recovery is not impossible. The so-called cystic degeneration of bone is said to be a consequence.

Treatment.—Theoretically, the indications are the same as for rickets, viz., to supply the blood with lime salts. Practically, they have not proved of much value. They may, however, be prescribed in the shape of the syrup of the lactophosphate of lime in the dose of one to two fluidrachms (3.7 to 7.3 c. c.) or the syrup of the hypophosphite in the same dose or the latter in combination with iron or with cod-liver oil. A proper hygiene and good food are of the utmost importance. Phosphorus itself is a drug highly commended. (See Rickets.) Strümpell gives the following prescription for it:—

℞. Phosphori, ... 0.01 gm. (0.15 grain)
Olei Amyg. express., ... 10. gm. (150 ")
Misce, deinde adde,
Pulv. Acaciæ,
Syrupi Simplicis, ... āā . . . . . 5. gm. (77 ")
Aquæ dest., ... 80. gm. (1234 ").
M. et S.—Two to four teaspoonfuls a day.

Women who are subjects of osteomalacia should be warned against marriage.

PURPURA.

Synonyms.—*Morbus maculosus; Peliosis; Hæmophilia.*

Definition.—A name given to several dyscrasic states, all attended by subcutaneous or submucous extravasations of blood. Such extravasations do not disappear on pressure, and vary in size from that of a pinpoint to larger areas a centimeter or more in extent. When minute or punctiform, they are called petechiæ, when larger than this ecchymoses. Purpura is always a symptom rather than a disease, but in certain conditions it forms the most conspicuous symptom of a group which scarcely admit of any other classification. In this event an adjective term derived from some more conspicuous one of these symptoms or from the name of some investigator who has described the condition is added to give precision. In other instances it is so purely a symptom and plays such a minor rôle in the disease that it is called symptomatic. Under any circumstances, it is not always easy to keep the varieties separated.

Symptomatic Purpura.

This includes the forms of purpura in which the petechiæ and ecchymoses are usually of minor importance. In a few instances where the dyscrasia is very great they become by their number and extent indices of the degree of such dyscrasia. Such are:—

(*a*) The purpura which often invades the extremities of the old (senile purpura).

(*b*) The purpura of the infectious diseases (infectious symptomatic purpura), especially typhus fever, small-pox, scarlet fever, measles, pyæmia, mycotic endocarditis.

(*c*) The purpura of poisons (toxic symptomatic purpura),—as, for example, that which occurs in connection with venomous snake bites, with over-doses of certain medicines, such as ergot, mercury, copaiba, quinine, iodide of potassium, and others. Certain persons possessed of idiosyncrasies acquire purpura on the administration of much smaller doses. In these the extravasations may occur anywhere on the body.

(*d*) The purpura attending certain diseases which, though not infectious, induce cachectic states, viz. (cachectic symptomatic purpura), cancer, tuberculosis, Bright's disease.

(*e*) Certain nervous diseases (neurotic symptomatic purpura), including locomotor ataxia, myelitis, rarely neuralgia, and hysterical states associated with the bleeding points known as *stigmata*.

(*f*) Mechanical symptomatic purpura, when induced by some cause resisting the onward movement of the blood, as a paroxysm of whooping-cough, croup, or an epileptoid fit.

The diagnosis of these conditions is a purely etiological one, and the prognosis and treatment are those of the diseases causing them.

Scorbutic Purpura.

Synonym.—*Scurvy*.

Definition.—A disease characterized by a dyscrasic state of the blood, which favors subcutaneous or submucous hemorrhage, by a peculiar spongy state of the gums, and by extreme general weakness.

Etiology.—Less than half a century ago the idea of scurvy was always associated with the sea-faring life, since sailors were its chief victims, though almshouses and prisons also held their complement. In the food of these persons fresh vegetables and vegetable juices and organic salts were wanting. So it came to be acknowledged that such privation was responsible for scurvy, and proof of this proposition was thought to exist in the fact that with the quicker voyages of ships and a supply of suitable food scurvy has almost vanished from the nosology. It appears, however, that, after all, the effect of such causes is predisposing and the disease may be really infectious, being due to some as yet undetected organism. Especially firm in this belief are those who have by reason of its distribution had the best opportunities for its study, as, for example, in Russia, where the disease is endemic, sometimes epidemic. Finally, it turns out that not only sporadic cases, but even epidemics, occur quite independently of the dietetic causes named.

At the present day scurvy has become an unusual disease, but is still met in camps, prisons, and almshouses, and situations where the food causes named exist along with dampness, bad air, and depressing influences generally, among which nostalgia is supposed to be especially potent. The effect of the food causes has been held by Garrod and by Ralfe to be the deficiency of the potassium salts, more especially of the alkaline carbonates derived from the conversion of the organic salts of

the vegetable and fruit juices, viz., the malates, tartrates, citrates, and lactates. Experimental proof of the latter is adduced by feeding animals upon acid salts, the effect of which is to impair their nutrition and produce dyscrasic states of the blood similar to those of scurvy and followed by similar extravasations.

The disease attacks the old and young of either sex, though the old are more susceptible, and it happens, probably from accidental circumstances, that more males are affected than females.

Morbid Anatomy.—This consists of the extravasations of blood which may be anywhere, subcutaneous, submucous, intermuscular, and interstitial, and in the alterations of the blood. The latter are not distinctive. The blood is dark and fluid, the blood corpuscles and hæmoglobin are concurrently reduced, and there is no leucocytosis. The gums are swollen, soft, and ulcerated, and the teeth sometimes drop out. Rarely there is even sloughing of the skin and mucous membrane, leaving ulcerated patches in the skin and bowels. The spleen is soft and enlarged, and there may be degenerative changes as well as hemorrhages in the kidneys, liver, and muscles, as well as hemorrhages into these organs.

Symptoms.—The features which especially characterize scurvy are the *etiology*, the *changes in the gums*, the *deep-seated* and *superficial hemorrhages.*

The etiology has been sufficiently discussed.

The gums are swollen, soft, and spongy, with disposition to bleed easily. In the more severe cases there is ulceration, with loosening of the teeth, the tongue is swollen, and the breath excessively foul. The gums of young children and of the aged are more often uninvaded. In rare cases only there is necrosis of the jaw.

The hemorrhages, always petechial, appear usually first in the lower extremities, then on the arms and trunk, but they occur anywhere as roundish, dark red spots, which may assume larger size. They are less common under the mucous membranes and in deep-seated tissues, and are rare in the face and scalp. Subperiosteal hemorrhages may occur. Nasal hemorrhages may be frequent, melæna and hæmaturia rare, hæmatemesis and hæmoptysis still more rare. The extravasations are slow to disappear, even when recovery takes place. The occasional sloughing has been referred to. A residual, slowly-healing ulcer results.

Other symptoms are *debility*, extreme in severe cases, and *anæmia.* The *pulse* is small, feeble, and frequent, and correspondingly the heart's action, which is sometimes irregular; more rarely is it slower than in health. The *temperature* is normal, rarely somewhat elevated. *Sore throat* is mentioned as a premonitory symptom. In bad cases, *nephritis* and *endocarditis* occur. *Articular* swelling is an occasional symptom; it is one of the results of the dyscrasia. So are *wheals* and *vesicles.*

Diagnosis.—This depends, as stated, on the etiology, the gingival changes, and the hemorrhages. It is these which chiefly distinguish it from the other forms of purpura.

Prognosis.—Sporadic cases always get well, and epidemic cases usually, unless too far advanced before coming under treatment.

Treatment.—This is usually most satisfactory when the necessary conditions are fulfilled,—a restored wholesome hygiene and suitable food. Good ventilation and out-door life in healthy localities with plenty of fresh vegetables, fruits, and fresh meats ordinarily suffice to accomplish a prompt cure. It is usual to give lemon and orange juice as the types of the fruit juices. Tonics and roborants, of which iron, quinine, and strychnine are the type, are the medicines needed. Antiseptic and astringent mouth washes should be used, and ulcers should be stimulated by local applications, of which nitrate of silver in solution is the best.

Infantile Scurvy.

Synonyms.—*Barlow's Disease; Periosteal Cachexia.*

Historical.—In 1878 and again in 1882 Cheadle published in the Lancet and about the same time Gee published cases of a cachexia associated with hemorrhage in very young children, occurring in England, and due to imperfect food supply. About the same time Barlow made an exhaustive study of the subject and gave his results in the Medico-Chirurgical Transactions, Vol. xlvi, 1883, and in the Bradshaw lecture for 1894. W. P. Northrup published a paper on "Scorbutus in Infants," describing cases in this country, in the New York Medical Journal, December 12, 1891, and another with Crandall in the same journal, Vol. i, 1894. Barlow's account is graphic, almost sensational, but I gather that the condition exists essentially in a hemorrhagic subperiosteal extravasation, causing thickening and tenderness in the shafts of the bones beginning in the lower extremities, but invading also the forearm and arm, more rarely the scapulæ, vault of the cranium, and face. Rarely there is intermuscular extravasation. The resulting tenderness and pain on motion causes the child to keep still with the legs drawn up, and to cry out when handled. The lesions are symmetrical, joints remaining free. The sternum and adjacent cartilages and a small portion of the contiguous ribs may be sunk bodily back as though subjected to violence. There may be a sudden prolapsus of an eyeball. Along with these symptoms is profound anæmia and erratic temperature, which may be subnormal, normal, or as high as 102° F. (38.9° C.).

The disease occurs at any period after four months, but it is most common from the ninth to the eighteenth and is of rapid development.

Treatment.—It has been ascribed to the use of the proprietary forms of condensed milk and preserved foods for infants. These should therefore be omitted and fresh cow's milk substituted, with beef juice and strained gruel made from whole-grain cereals only. Orange juice or lemon juice in water may also be given in moderate doses. Under this treatment the prognosis is favorable and recovery prompt.

Arthritic Purpura.

Synonym.—*Rheumatic Purpura.*

The characteristic feature of arthritic purpura is joint involvement. Hence it is also called rheumatic purpura, though its true rheumatic nature cannot be said to be absolutely settled. Three varieties are made :—

(*a*) *Simple Arthritic Purpura.*—This is a mild form most frequent in children. The articular pain is very mild and scarcely attended with fever. The spots are found for the most part on the legs, more rarely on the trunk and arms. There may be digestive derangement, manifested by loss of appetite and diarrhœa. The condition terminates favorably in a week or ten days. It may be associated with a slight degree of anæmia.

(*b*) *Peliosis Rheumatica.*—Schönlein's disease. This is a much more serious affection from every standpoint, occurring usually in young persons from fourteen to thirty. The joint symptoms are pronounced and multiple and there is decided swelling, pain, and fever, with a temperature of 101° to 103° F. (38.3° to 39.4° C.). The eruption first appears on the legs at about the affected joint, but I have seen it extensively on the arm, distant from the joint, followed by sloughing, and in the same case were retinal hemorrhages. Sloughing and necrosis of the uvula have occurred. It may be simply purpuric or may be associated with urticarial wheals—exudative, or vesicles—(pemphigoid purpura). When severe it is often associated with hæmaturia and hemorrhagic nephritis with œdema. I saw, with Agnew and Osler, a remarkable case in which the latter condition hastened a fatal termination by uræmia. Endocarditis is also a complication.

(*c*) *Henoch's Purpura.*—This is a variety occurring mostly in children, but also in adults, characterized by severe gastro-intestinal disturbance in addition to the above-named symptoms. There are pain, vomiting, and diarrhœa, rarely intestinal ulceration, and perforation with fatal peritonitis. Acute enlargement of the spleen has been observed.

Here, also, recovery is the rule.

Diagnosis.—The diagnosis is easy by reason of the associated joint symptoms, but the same doubt exists as to a true rheumatic nature in all forms.

Prognosis.—This is regarded as favorable, but fatal terminations do occur, especially in peliosis rheumatica where there is nephritis. Relapses in this form may occur at the same time of year for several years in succession.

Purpura Hemorrhagica.

Synonym.—*Morbus maculosus Werlhofii.*

Symptoms.—This is a grave form with hemorrhage from the mucous membranes, including nose, mouth, palate, stomach, and intestinal canal, in addition to extensive subcutaneous ecchymosis. The brain and kidneys and serous membranes may also be seats of hemorrhage—apoplectic symptoms indicating the first. A prodrome of languor and weakness may precede for a couple of days, to be succeeded by a rapid

succession of ecchymoses and hemorrhages. More decided constitutional disturbances follow, including typhoid symptoms and fever, though the latter is mild and may be altogether absent, even in severe cases.

In the *purpura fulminans* the hemorrhages are mainly confined to the skin, producing confluent ecchymoses and dense infiltrations covering large areas, with sanguineous blisters. The internal organs, on the other hand, remain free, while the urine and bowel evacuations are natural. At times there is fever, at others not. It has occurred after pneumonia and scarlet fever, and again in children apparently healthy.

Diagnosis.—As to diagnosis, scurvy is almost the only condition liable to be mistaken for purpura hemorrhagica. In the latter the gums are intact, and there is an absence of the conditions favoring scurvy.

Prognosis.—The termination is usually favorable in ten days to two weeks, although there may be fulminating cases, usually in children, terminating fatally in twenty-four hours. Severe cases take much longer.

Treatment of Arthritic Purpura and Purpura Hemorrhagica.—Treatment is best directed to improving the quality of the blood and general tone rather than to the control of the hemorrhage, though the latter must not be entirely ignored. Most that has been said of the treatment of scurvy is applicable to these forms of purpura. Iron and arsenic are the typical roborants and blood builders, to which good food, including vegetable juices, is to be added. Arsenic should be given in full doses, beginning with smaller ones and ascending rapidly.

In the articular forms the salicylates and salicin should be used in such doses as the stomach will tolerate.

Of the astringent remedies found serviceable may be mentioned ergot in doses of five to 30 minims (0.3 to 2 c. c.) of the fluid extract; persulphate of iron in doses of ¼ to ½ grain (0.016 to 0.032 gm.); acetate of lead, one to three grains (0.0648 to 0.2 gm.); dilute or aromatic sulphuric acid, five to 15 minims (0.33 to one c. c.).

Hemorrhagic Diseases of the New-Born.

Hemorrhagic Syphilis of the New-Born.—Usually about the third to the fifth day after birth hemorrhage is observed at the navel of the child, or it may occur earlier. Blood also flows from the mucous membranes, the mouth, the bowels, and from the kidneys. The skin becomes jaundiced. The stomach rejects food, and though it may appear at birth well nourished, the child rapidly wastes and dies at the end of a week or ten days. The autopsy discloses syphilitic lesions in the liver, lungs, nasal passages, and elsewhere.

Epidemic Hæmoglobinuria of Infants, or *Winckel's Disease.*—As described by Winckel in 1879, in an epidemic at the Foundlings' Hospital, at Dresden, the first symptoms, noted usually on the fourth day after birth, are a bluish tinge on the skin of the face, trunk, and limbs, with a more or less icteroid hue. There is fever, rapid breathing, and sometimes cyanosis. Occasionally there is vomiting and diarrhœa. The urine is light brown, albuminous, contains methæmoglobin, and

deposits a sediment consisting of epithelium and tube casts. The blood contains an excess of leucocytes and numerous granular bodies. The child lives, on an average, two days, though in one case death supervened in nine hours.

The autopsy disclosed yellow staining of the skin and internal organs; the spleen large, hard, and darkened; the kidneys dark brown in color, their tubules filled with granular pigment; the liver and heart fatty. There may be punctiform hemorrhages on the surface of the internal organs. There is no septic condition of the umbilical vessels. An infectious origin is not unlikely.

Acute Degeneration of the New-Born, or Buhl's Disease.—How far this disease, described by Buhl in 1861, differs from Winckel's disease, or the latter from the former, remains to be settled. For, in the first place, fatty degeneration of the heart and liver are found in many cases of Winckel's disease, while in others there is found the general fatty degeneration of kidneys, liver, heart, etc., described by Buhl. In the second place, infants surviving the first few hours after birth in Buhl's disease have the same symptoms as those described under Winckel's disease, while the other symptoms, such as minute hemorrhages and bile staining with various internal organs, are not essentially different.

Morbus Maculosus Neonatorum.—Still another form of hemorrhage from one or more of the surfaces and especially of the alimentary canal in the new-born is described under this title. The bleeding generally begins within the first week, but may be as late as the second or third. Hemorrhage from the bowels (melæna neonatorum) is the most frequent form, but it may be from the stomach, mouth, nose, and navel, or the navel alone. It may be accompanied by hæmatogenous jaundice—indeed, by any or all of the symptoms described under Winckel's disease—but differs in the occasional presence of fever and apparent absence of post-mortem lesions, though ulcers of the œsophagus, stomach, and duodenum have been found. It is generally fatal in from one to seven days. All of these conditions can be appropriately considered as forms of purpura.

Treatment.—The treatment of hemorrhagic affections of the new-born often avails little, though recoveries sometimes take place, especially in the last described form, in which C. W. Townsend reports 19 recoveries out of 50 cases collected.

The treatment demands absolute rest with the head low. Even the exertion necessary in nursing at the breast should be interdicted and the infant should be fed while recumbent with a teaspoon, using also the mother's milk if this be not condemned as worthless. The utmost care in providing uniform warmth should be taken. This can best be accomplished by means of an incubator. One may be improvised as suggested by F. A. Hoffman out of a box in the bottom of which hot bricks are placed. Over them is swung the infant's bed. A thermometer is so inserted as to be readily observed and the temperature kept at 89.6° F. (32° C.). To control the hemorrhage ergotin may be used hypodermically, one grain or minim (0.06) every six hours. Gallic acid may be given by the mouth in one-grain (0.06 gm.) doses, but extreme care should be taken in using remedies by the mouth.

HÆMOPHILIA.

Definition.—A hereditary vice of constitution, manifested by a tendency to uncontrollable hemorrhage, occurring either spontaneously or as the result of trifling injury.

Etiology.—No other explanation has as yet been offered of hæmophilia than that it is usually hereditary, though rarely also sometimes acquired. While individual instances of fatal hemorrhage were described centuries ago, "families of bleeders" were first described in this country. Of great importance because of its bearing on the marriage of these hæmophilia subjects is the fact that the tendency is transmitted through the female line rather than by the male. Thus, if a man not a bleeder marries a woman who is healthy and not a bleeder, his offspring are usually exempt from the affliction. On the other hand, a woman, a member of a bleeding family, marries, and even if she be exempt herself, she may have offspring who are bleeders. These facts were pointed out by Grandidier. Not all of the children of a bleeding family are afflicted, while the male members are more frequently subjects than females. The families of bleeders are apt to be large, and their appearance is that of health as a rule. It is said that blondes predominate, with delicate, soft skin, and distinct, distended veins.

Morbid Anatomy.—Two facts of importance have been recognized, viz., that in some instances the walls of the blood-vessels have been found thin with a fatty degeneration of the intima, and in many there is deficient coagulability of the blood. A third fact, which adds nothing in explanation of the hemorrhagic tendency, is a superficial situation of the arteries. A striking case of this kind in a blonde woman, formerly a bleeder, is now under my observation. Beyond this we know nothing, notwithstanding the exhaustive studies of Wickham Legg, Grandidier, and Hössli.

Symptoms.—Attention is commonly called to a bleeder by the occurrence of a hemorrhage difficult to control, though induced by some trifling cause. The extraction of a tooth is one of the most frequent of these events. It may be the prick of a pin, or a scratch, or a slight cut as in vaccination, or nothing at all may be discoverable. The tendency may manifest itself at the umbilical cord at birth or in Jewish children at the circumcision. On the other hand, the same accidents which are without result early in life may induce it later. Uncontrollable epistaxis is one of the most frequent manifestations, occurring in 169 out of 334 cases collected by Grandidier. It may be induced by simply blowing the nose. Other situations are the mouth, stomach, ear, eyelids. On the other hand, hemorrhages rarely occur in the interstices of organs, and though interstitial hemorrhages do occur, it is usually the result of trifling blows, when the well-known "black and blue appearance" is produced. The absence of interstitial hemorrhages, except as the result of some cause, however trifling, may be said to distinguish hæmophilia from the acquired hemorrhagic tendency.

The external hemorrhages, including those of the mouth and nose,

may be profuse and even fatal. They often last twenty-four hours and longer. When checked, reaction from them is rapid, and the victims rapidly resume their natural appearance, though repeated hemorrhages may engender a permanent anæmia.

Joint affections may be associated with this as with the acquired hemorrhagic tendency. They involve usually the larger joints and may include swelling and pain with fever, producing close resemblance to rheumatism, or there may be pain only.

Diagnosis.—This is apparent if the family tendency is known and any alarming hemorrhage without sufficient cause should excite inquiry.

Prognosis.—Sooner or later hæmophilia is apt to be fatal, though there may be many bad hemorrhages before the last one comes. The younger the subject the more serious the outlook. It does, however, happen that it is outgrown, subsequent attacks becoming milder and milder. In the majority of cases death takes place between the first and eighth year, and adolescence once survived, the chances of outgrowing it are greatly increased. Menstruation may be very copious in women but not fatal, while the natural loss of blood in child-bearing is rarely augmented. In the case referred to on p. 792 the hæmophilia disappeared with the appearance of the menopause, soon after which it was substituted by a chronic nephritis.

Treatment.—This may be prophylactic. The children of bleeding families should be carefully guarded against traumatic causes, however slight, while they should be carefully looked after from the hygienic and food standpoint. Fresh air, daily bathing, out-door exercises, and judicious measures intended to harden the threatened subject should be practised. Plain, wholesome, and nourishing food should be given and due attention paid to digestion. Toward prophylaxis, too, is discouragement from marriage, especially in the case of women.

During an attack absolute quiet must be enjoined. Styptics are to be employed locally, rather than internal medicines. Of styptics the solution of the perchloride or persulphate of iron is the best, beginning at first with dilute solutions and increasing to full strength of the officinal solution if necessary. Tannic acid is another good styptic, and if at hand may be dusted well upon the part or applied to cavities on cotton. In epistaxis the nose must be plugged if the ordinary methods of applying these agents fail.

Though little is to be expected from internal remedies, they may be tried. Wickham Legg recommends the tincture of the perchloride of iron in half-drachm (2 c. c.) doses every two hours. Ergotin and acetate of lead may also be used.

The usual treatment for consequent anæmia should be employed.

SECTION X.

DISEASES OF THE NERVOUS SYSTEM.

GENERAL INTRODUCTION.

HISTOLOGY OF THE NERVOUS SYSTEM.

The difficulties in the diagnosis of diseases of the nervous system are gradually diminishing as the thread of its histology is being unraveled. The studies of Waldeyer, Ramon y Cajal, Dejerine, Lanhassek, Golgi, van Gehuchten, and others have considerably altered previously accepted views, though in some respects the change is rather of terms than of more essential matters. A brief statement of the fundamental principles of the newer histology seems, however, necessary.

The studies of these observers resolve the nervous system into an immense number of individual units, to which Waldeyer has given the name of neurons. Each *neuron* is made up of :—

(*a*) A nerve cell.

(*b*) Protoplasmic processes or dendrons.

(*c*) An axis cylinder passing into the nerve fibre.

The *axis cylinder* after leaving the cell gives off lateral branches at different intervals known as collaterals. These *collaterals*, and finally the axis cylinder itself, break up into many fine fibres known as *end brushes* or branched tufts. Each neuron is independent of every other, that is, no protoplasmic process of one neuron is continuous with that of another, nervous communications being through simple contact or proximity. The protoplasmic processes conduct to the cell, are *cellulipetal* according to Cajal, the axis cylinders away from it, that is, are *cellutrifugal.* The end brushes or arborizations surround the body of another cell, or interlace with its protoplasmic processes or around the motor-end plates, communicating impulses to them. The nutrition of the neuron depends on the cell body. If the latter is intact, the processes are preserved. If it is injured, they waste, or if they are cut off they degenerate.

The cell bodies of the neurons are gathered in the grey matter of the brain and spinal cord and in the ganglia on the peripheral nerves. Their processes and axis cylinders traverse chiefly the white tracts of the brain, spinal cord, and peripheral nerves. The neurons of the anterior roots of the spinal nerves which lie in the anterior cornua of the grey matter have the protoplasmic processes short and the axis cylinders long.

In those of the posterior roots, which reside in the ganglia on those roots, the reverse is the case, the axis cylinders being short and the protoplasmic processes long, extending to the periphery, from which they gather impressions. Communication between different parts of the nervous system and with the rest of the body is thus kept up. The protoplasmic processes extending to the periphery receive impressions from the exterior and carry them cellulipetal to the ganglion cells on the posterior root of the spinal nerves, whence they are conveyed cellutrifugal to the nerve cells in the cord. This impression may result in a reflex act without the coöperation or knowledge of the brain, or it may proceed to the brain and give rise to a volitional act through the motor tract.

A motor impulse starting from the brain cortex must pass through at least two neurons before it can reach the muscles. In the course of this it is cellutrifugal from the cell in the cortex, cellulipetal to the cells in the nuclei on the motor cranial nerves and in the grey matter at different levels in the anterior cornua, and thence cellulifugal to the various muscles of the body, ending in arborization about the end plates. Hence we speak of the motor tract as being composed of two segments, an upper and a lower. The neurons of the upper motor segment have their cell bodies and protoplasmic processes in the cortex about the fissure of Rolando. The axis cylinder processes run through the internal capsule and the peduncles, through the pons, medulla, and cord, ending in arborizations around the protoplasmic processes and cell bodies of the lower segment. The neurons of the lower segment are those having their cell bodies and protoplasmic processes in the ganglia of the motor cranial nerves and in the anterior cornua of the grey matter, while their axis cylinders leave the spinal cord by the anterior roots of the spinal nerves, to be distributed as described. The *upper segment* is a *crossed tract*, that is, the neurons composing it have their cell bodies and protoplasmic process in the cortex, while their axis cylinders cross the middle line to end in the neurons in the opposite half of the lower segment and thence to the muscles on that side of the body. So that motor impulses starting in the left half of the brain produce contraction in the muscles of the right half of the body, and *vice versa*. The *lower motor segment is a direct tract*, that is, its neurons, with their protoplasmic processes and the muscles to which they are distributed, are all on the same side of the body.

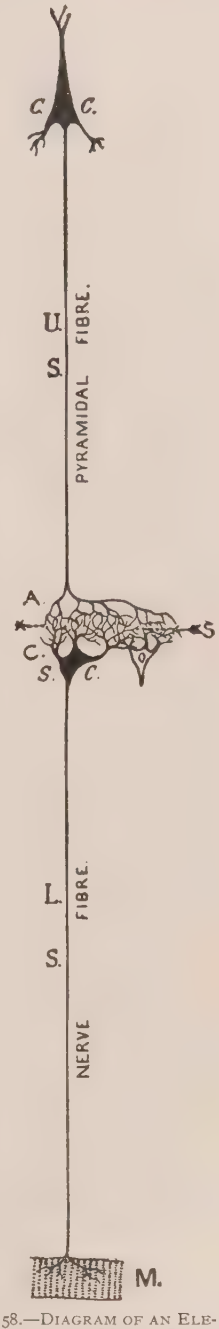


FIG. 58.—DIAGRAM OF AN ELEMENT OF THE MOTOR PATH.

U. S. Upper segment. L. S. Lower segment. C. C. Cortical cell. S. C. Spinal cell. A. C. Anterior cornu. M. Muscle. The dotted line at the level of S. indicates the junction of the two segments. S. Path from sensory nerve roots.—(*After Gowers.*)

The path for sensory conduction is also composed of two segments, an upper and a lower sensory neuron, but the direct route of sensory conduction is more complicated and our knowledge is much less exact. Nor are the tracts so definitely separated. The cell bodies of the lower neurons are on the ganglia of the posterior roots of the spinal nerves and in the ganglia of the sensory cranial nerves. These ganglion cells have a single process which, after leaving the cell, divides in a T-shape manner, or it may be that there are two processes from the outset, one running into the central nervous system and the other toward the periphery of the body. The process which conducts toward the periphery is a protoplasmic process, while that which conducts to the centre is the axis cylinder. The former runs in the sensory nerves, starting from the various specialized sensory apparatus of the periphery; the axis cylinder enters the cord by the posterior roots. After entering the cord it divides into an ascending and a descending limb, which traverse the posterior columns. The descending branch runs a short distance and ends in the grey matter of the same side of the cord, giving off a number of collaterals, which also end in the grey matter. The ascending branch may end in the grey matter soon after entering the cord, or it may run in the posterior columns as high as the medulla, ending in the nuclei of the posterior pyramids. Thus the *lower segment* is also a *direct tract* terminating in what is essentially the neurons of the grey matter of the posterior cornua at different levels. See also section on spinal cord. The *upper segment* starting from these is a *crossed tract*, crossing at different levels, so that sensory impressions are ultimately lodged in the brain on the side opposite that whence they start in the periphery. The muscular sense alone is conducted upward on the same side in the columns of Goll on each side the posterior median fissure. The exact termination of the sensory processes in the cerebral hemisphere is not known, but they pass up externally and posteriorly in the pons and internal capsule, and seem to terminate in the so-called motor cortex, which is, probably, therefore, strictly speaking, sensory-motor.

Topical Diagnosis.—Both motor and sensory nerve roots arise from definite segments of the spinal cord. After uniting at the same level they descend a short distance within the spinal canal and pass out between the vertebræ as spinal nerves. But in their distribution they do not retain the same definiteness, the same sensory and motor areas being supplied with nerves from different segments of the cord, and there is an overlapping, as it were, of parts supplied by different nerves. At the same time, by the combined aid of experiment and morbid physiology, we have learned that movements in certain muscles are accomplished by motor nerves which emanate from certain segments of the spinal cord, and that from certain sensitive areas are gathered up impressions which are carried to corresponding sections of the spinal cord. Nay, more. By the same means we have learned that there are certain areas in the cortex of the brain that preside over certain motions, and certain areas which have to do with sensation; though with respect to the latter our knowledge is much less definite. We know more of the cortical representation of the *special* senses than of general sensibility and pain. These

facts are the foundation of what is known as *topical diagnosis*, in the case of the brain as *cerebral localization*, by which is meant the inference, from the study of local derangements of sensation and motion, of the more or less exact site of lesions of the nervous system. These will be considered with appropriate detail in our study of the diseases of different parts of the nervous system.

GENERAL SYMPTOMATOLOGY.

(Investigation of a Case of Nervous Disease.)

The advantages of a careful method in the study of disease are perhaps more apparent in the case of the nervous system than in that of any other of the anatomical divisions of the human body. This is partly because of the number and variety of the affections to which the nervous system is subject, and partly because of the association with widely different lesions of certain identical symptoms.

The primary steps of family and personal history are the same as for other diseases, including age, sex, occupation, and whether married or single. We may therefore pass at once to the study of such symptoms as are special.

I. Phenomena of Motion.—It does not matter much whether we set out with sensory or motor phenomena, but it appears somewhat easier to begin with derangements of motion, and, of these, (*a*) *voluntary motion* is naturally first investigated. To this end, the patient is asked to move his limbs, while the strength of whatever motion he is capable of is easiest measured by resisting it, and by testing the strength of his hand grasp. For more accurate measurement, the dynamometer is used, an instrument devised to measure both compression and traction, although it is more commonly restricted to the former. Advantage may be taken of the fact, too, that the same motion requires different degrees of strength in different positions of the body. Thus it is easier to draw up the thigh when lying on the back than when on the side, and it may be possible in the former position when it is not in the latter. Both extensor and flexor muscles must be thus tested. By such an investigation, we discover the presence of a complete *paralysis*, or total loss of voluntary motion, and *paresis*, or simple weakening of such motor power.

By a *monoplegia* is meant an isolated paralysis of one part of the body, as an arm or leg. By a *hemiplegia* is meant a paralysis of the entire half of the body, including half of the face, one arm, and one leg, also known as unilateral paralysis. By a *paraplegia* is meant a simultaneous paralysis of corresponding halves of the body. Paralysis of the two arms is spoken of as a *superior paraplegia*, of the two legs as an *inferior paraplegia*, while the word paraplegia alone is often used for the latter condition. A *diplegia* is a paralysis with spasm of all the extremities.

Impairment of voluntary muscular power, as thus tested, must be the result of structural change in the motor area of the cortex, in the

great motor tract of the brain or cord, or impairment in the integrity of the efferent nerves, or it may be more rarely in the muscle itself, "myopathic palsy;" or the power of the will may be abrogated. In diseases of the peripheral nerves, when the paralysis is called peripheral, it is limited to the region of distribution of the affected nerves, whether one or many. It may be said in general that hemiplegia is the usual form of cerebral paralysis, while paraplegia is the expression of spinal paralysis. Monoplegias are usually either due to lesions of the cortex, or are peripheral palsies.

In all hemiplegias caused by lesions above the pons, the palsy, including face and extremities, is on the side opposite the lesion, but in most lesions in the pons there is *crossed paralysis*, that is, there is paralysis of the extremities on one side, and of the face on the other side, because the fibres of the facial nerve cross much higher than the fibres to the extremities. The result is a paralysis of the face on the same side with the lesion, and of the extremities on the other. This would be the case with a lesion at *b*, Fig. 59. If, on the other hand, the lesion is higher up, above the decussation of both the facial and pyramidal tracts, as at *a*, the paralysis is on the side opposite the lesion, in both face and extremities. Other nerves may substitute the facial in this crossed paralysis, as the hypoglossal or abducens. In rarer instances there may be lesion at the very decussation of the pyramids, cutting the motor fibres of one extremity before they cross, and those of another after crossing, producing the very rare event of paralysis of an arm on one side and leg on the other.

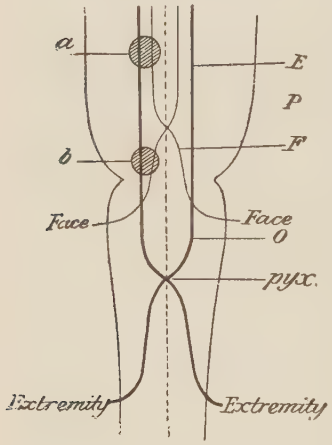


FIG. 59.—ILLUSTRATING CROSSED PARALYSIS.

O. Medulla oblongata. *pyx*. Decussation of anterior pyramids.—(*After Hirt.*)

(*b*) Having determined this question of muscular strength, and the corollaries which grow out of it, we have next to ascertain to what extent the power of *coördination* is influenced. Every muscular act requires the duly proportioned coöperation of a number of muscles; and as the complexity of the act increases, the number of muscles required to cooperate also increases. Such coöperation is termed coördination, and its absence is recognized in the tottering gait of the drunkard, and the condition is known as *ataxia*. There are two separate parts of the nervous system which preside over coördination—the cerebellum and the posterior columns of the spinal cord. Disease of either of these may, therefore, produce ataxia. In point of fact, it is most frequently the latter.

A corollary, growing out of the investigation of the coördinating power, is the study of *station*, or the steadiness with which one stands with the eyes closed or open, and it is measured by sway of the head and body, laterally and antero-posteriorly. In health a lateral sway of the head exists to the extent of half an inch (1.25 cm.), and an antero-posterior sway of an inch (2.5 cm.). A sway much beyond these limits is abnormal.

(*c*) After ascertaining the condition of voluntary motion, coördination, and station, we have to inquire into the question of possible *motor irritation* or *excessive muscular action* or *spasm*. Spasm may be continuous, *i. e.*, lasting for minutes, hours, or days, when it is known as *tonic;* it may be intermittent or *clonic;* or it may be an admixture of both, when it is *tonic-clonic*. Spasm occurs also in involuntary nonstriated muscular tissue, but as a manifestation of nervous disease only in voluntary muscles. The presence of spasm implies irritation of motor centres, motor tract, or motor nerves, but motor irritation may also be excited secondarily by some reflex route, the result being a reflex spasm.

Spasm and paralysis are often associated. Thus a limb may be paralyzed in a state of contraction, exhibiting a peculiar rigidity, and to such a condition the name *spastic paralysis* is applied. This condition may also exist as a state of persistent contraction of the antagonists of the paralyzed muscles, constituting the so-called *contractures*. Paralyses in which there is no such resistance to passive motion are known as *flaccid* paralyses.

Through the combination of tonic and clonic spasm result different varieties of morbid involuntary movements more or less complex. Some of these are the following :—

(1) *The Epileptiform Convulsion.*—This consists in a succession of clonic and tonic-clonic spasms extending over the whole or a part of the body, throwing the part involved into violent motion. The masseter and the temporal muscles share in the contraction, whence the tongue is often bitten. The convulsion of epilepsy is the type of this form, but the convulsions of uræmia, of hysteria, and of organic disease of the brain are also epileptoid.

(2) *Rhythmical Contractions.*—These occur in single groups of muscles and are sometimes seen in apoplexy and cerebral sclerosis. They may usher in the epileptiform convulsion, or the convulsion may terminate by a gradual substitution of these for the more violent spasms.

(3) *Tremors or Trembling Motions.*—These are limited movements, *i. e.*, movements of short excursion which rapidly succeed each other. "Shaking" is a more pronounced degree of tremor. Tremor is characteristic of paralysis agitans and other nervous affections. It occurs in old people as senile tremor, in users of alcohol and tobacco. In paralysis agitans it is present, at first, only when the muscles are moved voluntarily. Later it occurs independently of voluntary motion. The former is known as intention tremor, and is characteristic of multiple sclerosis. The immediate anatomical changes on which they depend are not known.

(4) *Single Contractions.*—These are either sudden twitchings or slow contractions of muscles, seen especially in diseases of the spinal cord. They may be single or multiple and persistent. They may be the result of direct motor irritation or reflex in origin.

(5) *Fibrillary Contraction.*—These are contractions of separate small bundles of muscular fibrillæ, comparable to the "quivering" of raw flesh. They are independent of motion. They may be pronounced and wave-like over the muscular substance. It is especially seen in the atrophied muscles of progressive muscular atrophy. The "quivering"

of the eyelid and of the orbicularis muscle below the eye, often an annoying symptom, are instances of this condition.

(6) *Choreic Movements.*—These are short, sudden movements usually separated by short intervals of time, most always at first seen in the face, later in one limb or over the whole body. They may be very complex and general, resembling the true epileptic convulsion, but they differ strikingly from it in that consciousness is retained. They are characteristic of chorea, but also accompany other nervous affections, such as post-hemiplegia chorea.

(7) *Athetosis.*—This is a peculiar slow, involuntary movement, usually of the fingers and hands, but also of the head and trunk. The fingers make slow movements of the nature of extension and flexion, spreading and approximating and surrounding each other in a striking way. They are characteristic of a disease known as "athetosis" and are a symptom of certain central nervous diseases, especially the cerebral palsies of children.

(8) *Constant or Coördinate Spasms.*—These consist in irresistible complicated movements like moving forward or moving in a circle or rotating on the axis of the body; also complicated forms of spasm resembling jumping, laughing, screaming, all involuntary and forced. The first group of these is especially seen in disease of the cerebellum and cerebellar peduncles, the latter in severe forms of hysteria.

(9) *Tonic Spasms.*—These, as already stated, are continuous contractions, also called tetanic because so characteristic of tetanus. They occur in trismus or lock-jaw, while tonic contraction of the muscles of the back produces the condition known as *opisthotonos*, in which the vertebral column is arched and the body rests upon the back of the head and heels. Tonic spasms are often attended by pain in the conscious person, probably due to pressure on intramuscular sensory nerves, when they are called "cramps." Such are cramps in the calf of the leg, and the exquisitely painful so-called "kinks" in the back.

(10) *Cataleptic Rigidity.*—In this there is also a tonic contraction of muscles whereby a limb remains for a considerable time in any position in which it may be passively placed, the will being abrogated. If the position of the limb be changed it remains again in this situation, and from a certain resemblance to the behavior of wax under like circumstances it has received the name of "waxy flexibility." It is characteristic of certain forms of hysteria and is one of the results of hypnotism, but is found in association with true organic brain disease, as meningitis and progressive muscular paralysis. In hysteria it is commonly associated with anæsthesia and loss of consciousness. It is also associated with certain psychoses, especially certain grave forms of melancholy known as *melancholia attonita and katatonica.*

(11) *Associated Movements.*—These are unintentional and irresistible movements which take place in muscles at the same time with other motions actually intended, as, for instance, a motion in the arm when the patient wills to move only the leg.

(*d*) *Bladder control* and *rectum control* are next to be looked into. Full control over the acts of these organs implies, first, an integrity of the

lumbar portion of the cord in which reside the reflex centres regulating these acts; and, second, the integrity of volition, which, to a certain extent, fortifies such regulation. Through the operation of the reflex centre, bladder and rectum both empty themselves when a certain degree of distention is attained. Through the operation of the will, such evacuation is put off to a convenient time. Through an undue irritability of the reflex centre, such evacuation is imperative, and does not bide the will, or it may take place while the will is in abeyance, as in sleep. Thus may be explained some of the incontinences of urine in children. Again, if will-power is lost from disease of the cerebral cortex, so long as the lumbar cord is intact evacuations of the bowels and bladder take place involuntarily.

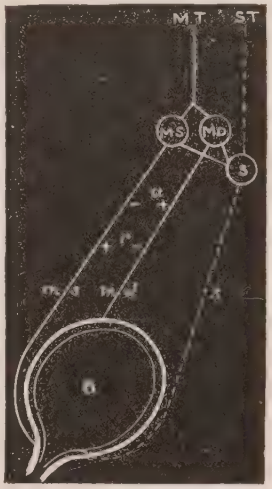


FIG. 60.—DIAGRAM SHOWING PROBABLE PLAN OF THE CENTRE FOR MICTURITION.

M T. Motor tract. S T. Sensory tract in the spinal cord. M S. Centre, and *m s*, motor nerve for sphincter. M D. Centre, and *m d*, motor nerve for detrusor. *s*. Afferent nerve from mucous membrane to S, sensory portion of centre. B. Bladder. At *r* the position during rest is indicated, the sphincter centre in action, the detrusor centre not acting. At *a* the condition during action is indicated, the sphincter centre inhibited, the detrusor centre acting.

On the other hand, if the integrity of the lumbar cord is lost, if the cord is destroyed or degenerated, there will be no response to the sensory impressions conveyed from a full bladder or rectum, and it remains unemptied; whence torpor or complete paralysis of the bowels and bladder are common symptoms of spinal disease; and while the repletion of the latter organ may finally overcome the resistance of its sphincter and lead to dribbling, the rectum may go on filling up until it is emptied by the finger or the handle of a spoon.

(*e*) The state of the *reflexes*, as they are called, is next tried. As here used, the term "reflex" is applied to a muscular contraction stimulated by a sensory impression, the simplest illustration of which is the retraction of the leg of the sleeper when the sole of the foot is tickled. For diagnostic purposes the reflexes are divided into the "cutaneous reflexes" and the "tendon reflexes."

The *cutaneous* or superficial reflexes are muscular contractions which take place in different parts of the body in response to irritation of sensory nerves of the skin, as by tapping it lightly or drawing the finger or a pointed instrument lightly over it. The sudden application of heat or cold or the prick of a pin or pinching are other modes of excitation. The contractions are generally confined to the neighborhood of the locality irritated. The skin reflexes are very much more easily excited in children than in adults, and in the lower extremities rather than the upper; also with varying facility in different persons. They receive various names, according to the situations where they are readily excited. Thus we have the "plantar reflex," where contraction is excited by tickling the sole of the foot, resulting in a

flexion of the toes or foot, or even in a drawing up of the leg; the "cremaster reflex," contraction of the cremaster muscle and consequent drawing up of the scrotum on stroking or scratching the inside of the thigh. The retraction may take place on the one side of the scrotum only or on both. Then there is the "abdominal reflex," or a contraction of the abdominal muscles when the skin of the abdomen is stroked or scratched. A subdivision of the latter is the "epigastric reflex," produced by an irritation on the side of the thorax in the 4th, 5th, and 6th interspaces. The result is a dimpling of the epigastrium on the side stimulated. Cutaneous reflexes may be brought out in other portions of the body, as in the gluteal region by irritating the skin of the buttock. A contraction of the muscles about the scapula, the "scapular reflex," is produced by an irritation between the scapulæ. To test for the cutaneous reflexes is more important in the lower extremity than in the upper.

The *tendon* reflexes, or deep reflexes, are so called because they are generally elicited by striking upon tendons, the corresponding muscles being placed slightly on the stretch, care being taken, however, to avoid all active tension in the muscle by the person examined. The blow is made either with the edge of the hand or a hammer adapted to the purpose, usually of rubber. A sharp, sudden contraction of the muscle usually takes place. A similar, though less decided, contraction may be elicited by the mechanical irritation of parts analogous to tendons, as periosteum and fasciæ, and by striking the muscle itself.

The most commonly tried of the tendon reflexes is the *knee jerk*, or *patellar tendon reflex*, produced by striking the tendon of the quadriceps femoris between its insertion and the patella, while the leg is crossed upon its neighbor. The weight of the pendant leg gives a sufficient degree of tenseness. When the knee jerk is normal, there is a decided rise of the foot with each blow of the hand or hammer. This motion may become abnormally increased or diminished. A more limited movement may also be produced by striking the patella itself or the quadriceps tendon above the patella, and, when the reflex is exaggerated, by a very light tap in these situations or even on the tibia. When thus exaggerated, the knee jerk may also be brought out in bed as follows: the quadriceps tendon being put on the stretch by pressing the patella downward in the direction of the leg with the finger, the patella is percussed in the same direction. With each stroke there is a contraction, and the finger and patella are drawn upward. A "clonus" or repeated contraction may even be thus produced.

Similar is the *ankle reflex*, produced by tapping the *tendo-Achillis* when the calf muscles are placed slightly on the stretch by a slight dorsal flexion of the foot. In health, the ankle reflex is not always produceable, but in disease in connection with this contraction is shown the most remarkable of the exaggerated reflexes, the "ankle clonus" or "foot clonus." It consists in contractions rapidly repeated so long as the tension of the calf muscle is kept up by pressing the foot toward flexion. Six to nine such contractions may occur in a second, and sometimes the whole leg is thrown into vigorous contractions.

Reflexes are also elicited in the upper extremities, but they are much less striking, and are often totally absent in health. The most important of these are the *arm jerks*, produced by striking the biceps tendon at the elbow-joint in front, or by striking the triceps tendon above the olecranon. So-called periosteal reflexes—reflexes excited by striking the periosteum —may in exaggerated states be produced in the supinator longus and biceps of the upper extremity by striking the lower end of the radius and ulna; also in the adductors of the thigh by striking the internal condyle of the femur. The *jaw jerk* is a similar contraction, produced by tapping on the front of the jaw, while the closing muscles of the jaw, viz., the pterygoids, masseters, and temporals, are placed on the stretch by partially opening the mouth.

The tendon reflexes were first studied by Charcot, Erb, and Westphal, and later by Tschirjew, Gowers, Jendrássik, Weir Mitchell, Lombard, and others. Erb explained the phenomena as purely reflex in their character, requiring the offices of a centripetal and centrifugal nerve, an intermediate centre, and the operation of an excitant. Westphal, on the other hand, regarded them as simple muscular contractions, stimulated as are the bared, quivering muscles of the recently-killed animal, the tendon being simply the intermediary substance through which the irritation is conveyed. It was early objected to the purely reflex nature of these phenomena that a shorter time is usually required to produce them than to produce an ordinary reflex action, being but $\frac{1}{40}$ to $\frac{1}{30}$ of a second, as compared with $\frac{1}{15}$ of a second. But the strongest objection is found in the results of the experiments of Tschirjew, who cut all the nerves to the patellar tendon, and found the reflex still remained excitable.

Nevertheless, the tendon reflexes are arrested by any lesion which arrests reflex action. Hence, reflex action must come somewhere into play. Accordingly, Gowers suggests that the *irritability* on which depends the contraction is the result of a reflex action excited by the passive tension. Thus the contraction is local, but the irritability which makes the local contraction possible is reflex. Hence, too, according to Gowers, the term "tendon reflex" is altogether inaccurate, and he suggests the word *myotatic*, from μυών, muscle, and τατικος, extended, because tension is necessary for the production of the contractions. Weir Mitchell puts it well in these words: "A muscle moves when struck because of its innate capacity to twitch when irritated, but it does not move when excited by a blow on its tendon unless it has, besides its own excitability, a constant influx of tone-waves from spinal centres." *

Hence in a complete examination the "muscle jerk," or idio-muscular contraction, also known as mechanical muscular irritability, should be tested also. It is done by a sharp, sudden tap on the muscle with the hammer. The response is of two kinds, first as a sudden contraction, and second as a hump-like rise which subsides slowly. The pectoral muscles are favorite seats for eliciting the pure muscle reflexes. It is, of

* Mitchell and Lewis, "Tendon and Muscle Jerk," Trans. Asso. of Amer. Physicians, Vol. 1, p. 13, 1886.

course, impossible to deny that there is nerve irritation as well as muscular in such a blow.

Both the tendon jerk and muscle jerk are capable of reinforcement by coincident muscular exertion, as lifting weights or clinching fists, originally discovered by Jéndrássik * in 1883 in the case of the tendon jerk. Mitchell and Lewis † also discovered in the course of their study of ataxic cases that the pure *muscle* jerk or hump could be produced after the tendon reflex could no longer be elicited, and that both could be produced by the reinforcement referred to after they had disappeared to ordinary conditions.

What are the conclusions to be drawn from modifications in the reflexes? In the first place, it is to be remembered that they vary somewhat within the limits of health. Especially is this true of the cutaneous reflexes, which are also less easily elicited than those of the tendons. In general terms, *diminution* or *absence* of a reflex implies a breach of integrity somewhere in the reflex arc, formed by the centripetal nerve, the grey matter, the special anterior cornu, and the motor nerve, or an increase in the reflex cerebral inhibitory action from above. The latter would be irritative. The former may lie in the reflex centre of the spinal cord, in the anterior cornua of its grey matter, or in the centrifugal or centripetal nerve; hence it may be found in diseases of the peripheral nerves as well as of the spinal cord. If it is in the centripetal nerve, it will be accompanied by impaired sensation; if in the centrifugal, there will be defective motion. Disease of the latter may also cause degeneration and wasting of muscle with loss of its irritability, as will also degeneration of the motor centre of the cord. Increase of the reflexes, on the other hand, implies increased irritability of the motor areas of the cord (anterior cornua and pyramidal fibres, especially in their terminal part adjacent to the grey matter) or a deadening of cerebral inhibition, as in certain cases of brain disease or disease of the cord high up. Thus it is well known that disease of one cerebral hemisphere lessens or abolishes the superficial reflexes on the opposite or paralyzed side of the body. In certain diseases of the cord there is a *delay* in the manifestation of the cutaneous reflexes after the irritation of the skin is applied, an interval of ten to fifteen seconds being often recorded before the response ensues. Increase of cutaneous reflexes is manifested by an unusual readiness of response in the normal areas or an extension of these areas beyond their normal boundaries.

Segments of the Cord Presiding over Certain Reflexes.—In general it may be said that absence of the tendon reflexes is especially characteristic of poliomyelitis and locomotor ataxia, and of all peripheral paralyses, traumatic paralyses, and neuritis; also of advanced diabetes mellitus. Abnormal increase is present in spastic spinal paralysis and in cerebral paralyses, being due in the latter instances to withdrawal of the normal inhibitory influences.

---

* "*Beiträge zur Lehre von den Sehnenflechsen*," *Deutsches Archiv für klinische Medicin*, 1883, Vol. XXIII, p. 175.

† *Loc. cit.*

Further accuracy in the application of a knowledge of the reflexes and their modifications is secured by a knowledge of the exact portion of the grey matter presiding over the most important of them. Premising that some of these centres are of considerable vertical extent, the following from Gowers may be regarded as approximate for each of the reflexes named :—

Superficial Reflexes.—Plantar, opposite 2d sacral nerve; gluteal, 4th lumbar; cremaster, 2d lumbar; abdominal, 6th to 7th dorsal; epigastric, 6th dorsal; scapular, 5th cervical to 1st dorsal.

Tendon or Deep Reflexes.—Calf muscles (foot clonus), 5th lumbar and 1st sacral; knee jerk, 3d and 4th lumbar; flexor digitorum and triceps, 7th cervical; biceps and supinator longus, 6th cervical.

(*f*) *Paradoxical contraction* is a symptom allied to the reflexes for which no satisfactory explanation has been afforded. It was first studied by Westphal, and is a contraction commonly observed in the *tibialis anticus* muscle, induced by forcibly flexing the foot on the leg. As a result, the foot remains thus flexed for a considerable time, in one case twenty-seven minutes, after which it slowly relaxes. On repeating the flexion, the phenomenon recurs, but the response gradually diminishes in intensity. Contractions induced by faradism may similarly persist. It has been noted in both spinal and cerebral disease, including the early stage of tabes dorsalis, multiple sclerosis, and paralysis agitans. More rarely it may be induced in the flexors of the leg and forearms.

(*g*) *Electrical excitation of motion* is an important means of investigation in nervous diseases. In health, motion may be excited by electrical stimulus applied to the muscle through its nerve or directly to the muscle itself, which cannot exclude the possible excitation of intra-muscular nerves. The latter is called direct, the former indirect. This is true of the constant or galvanic current, and of faradism or the induced current.* The same uniformity of response does not occur to the two kinds of electricity in pathological states. Hence every complete investigation should include the use of both currents.

In order to test the electrical condition of muscles and nerves, one electrode, the indifferent pole, may be held in the hand of the patient or placed over the sternum or at the back of the neck, while the other or *testing* pole is applied to the nerve or muscle, selected in accordance with the well-known nerve points of Erb in Ziemssen's plates. The testing electrode should be small enough to permit the isolation of a single nerve or muscle.

With the faradic battery contractions may generally be produced in health with great facility, either directly or indirectly, although stronger currents are required for direct stimulation. So they can with the gal-

* Under all ordinary circumstances electrical contraction produced in muscles is indirect, that is, through the nerve filaments distributed to the muscle. That the two are, however, distinct may be shown through the influence of curare, which destroys nerve irritability, but allows that of muscle-protoplasm to remain. In this way may be shown, too, that the *nerve* is more sensitive to electrical stimulation in health than muscle, since under the influence of curare the muscle requires a stronger faradic current to excite its contraction than in the normal state, while the slowly interrupted galvanic current excites muscular contraction as in the normal state.

vanic battery. Contractions take place with the latter only at the making and unmaking of the current by the "commutator" or "reverser." A definite law of response also exists with galvanism. Thus, beginning with very weak currents, it is observed that contraction first takes place at the moment of that closure which makes the testing pole the kathode or negative pole—kathodal closure (KaCl). As the strength of the current is increased, the kathodal-closure contractions become stronger, and anodal-closure (AnCl) contractions make their appearance. With still stronger currents, the anodal-opening (AnO) contraction occurs, and last of all, when the kathodal-closure contractions become tetanic (Te), slight kathodal opening (KaO) contractions appear. These facts are equally true of normal muscle and nerve, and may be formulated. Representing slight contraction by a small (c), decided contraction by a large (C), and the absence of contraction by a minus sign (—):

With weak currents, KaCl*c*, KaO—, AnCl—, AnO—.

With stronger currents, KaCl*C*, KaO—, AnCl*c*, AnO*c*.

With strongest currents, KaCl*Te*, KaO*c*, AnCl*C*, AnO*C*.

In *pathological states*, two sets of deviations from the normal reaction to electrical stimulus are observed, viz., *quantitative* and *qualitative*.

In the *quantitative* deviations there is simply an increase or diminution of the normal irritability of both nerve and muscle to either faradism or galvanism. These differences are, of course, most easily measured when the alteration exists only on one side of the body, which may then be compared with the other. When both sides are affected, estimates can only be made by comparison with a healthy body or by appliances for measuring the strength of electric currents used, as the galvanometer. For this purpose superficial nerves, such as the frontal, ulnar, and peroneal, are usually selected. *Instances.*—Increased quantitative changes are found in tetanus and the early stage of certain peripheral palsies, while diminished electrical excitability is found in spinal paralysis, progressive muscular atrophy, and bulbar paralysis.

More important from a diagnostic point of view, at least, are the so-called *qualitative* deviations from the normal law of contraction *known as the reaction of degeneration.* These affect the *direct* stimulation of *muscle* by *galvanic* currents only, and may, in general terms, be regarded as consisting in a reversal of the usual phenomena. They are best illustrated by describing the electrical phenomena which present themselves in an ordinary case of peripheral paralysis. In two or three days to a week after its appearance there begins a gradually diminishing response in the *nerve* to both faradic and galvanic currents. This goes on for one or two weeks, at the end of which time it disappears to both currents, even the strongest. During this same time the *muscle* is also losing its responsiveness to the faradic current, but not with the galvanic. There is also at first, although not always, a slight diminution, lasting, say, one week, and constituting the "first degree" or "first stage" of degeneration. But during the second week this is substituted by an increased excitability, so that there is now marked response to weak currents—increased quantitative deviation. But there is also *qualitative* change. The anodic-closure contractions become now as strong as

or stronger than the kathodic-closure contractions. Nay, more, the kathodic-closure contractions, which in health were exceedingly weak and could only be brought about by the strongest currents, are now often stronger than the kathodic closure. This state of affairs may be represented thus :—

First stage of reaction of degeneration—one week :—	Diminished quantitative response to galvanism. No qualitative deviation.
Second stage of reaction of degeneration—four to eight weeks :—	Increased quantitative response to galvanism. Qualitative deviation as follows :— AnCIC = or > KaClc. KaOC > KaClc.

The phenomena of qualitative change are purely muscular, and it should be mentioned that they are not always typically present.

More constant and equally distinctive is the second qualitative change in the muscular contractions excited by galvanism in this stage. Instead of being quick or sudden, they become slow, prolonged, and vermicular, often continuing throughout the whole period of closure of the current.

The second stage lasts from four to eight weeks, increasing during the third and fourth. In cases of recovery, the abnormal muscle irritability to galvanism often persists after return of voluntary power, but it diminishes as the faradic irritability returns. In severe cases, where recovery does not take place and the nerve is not restored to its natural state, *all nerve* irritability and *faradic muscular* irritability remaining permanently absent, the *increased galvanic muscular* irritability may continue for months, but ultimately also decreases, disappearing finally with the muscular substance.

Certain exceptions to these rules must be mentioned. Thus, when the nerve lesion is slight, the fall in quantitative *nerve* irritability is sometimes preceded by a slight rise, or the rise may persist throughout, and such rise may be considered as evidence of a slight lesion. Further, the change is not always the same to faradism and galvanism, and is often brought out much better by the slow interruptions in the faradic battery than by the rapid interruptions in the same or by the galvanic current. Gowers noted in one instance slight but prolonged diminution to faradic irritability when no change could be found with voltaism, and Bernhardt has noted lessened irritability to faradism with distinct increase to galvanism in an ulnar nerve the seat of traumatic paralysis. Again, *faradic* irritability may not diminish to the degree in the muscle as in the nerve in slight cases, and conduction of voluntary impulses from the brain may be possible when there is no response to electrical currents, and *vice versa*. In still milder peripheral paralyses there is no reaction of degeneration at all, whence a favorable prognosis may always be made. It is to be especially observed in recovery from nerve lesions that *voluntary* motion often returns decidedly earlier than the electrical excitability of peripheral nerves.

What do reactions of degeneration teach us? Simply that the disease is seated in the anterior cornua of the grey matter of the cord, or in the peripheral nerves. They teach us nothing as to the nature of the

lesion. Upon the integrity of the cell bodies of the neurons in the anterior cornua and their "trophic influence" depend the nutrition of the nerve and the muscle to which it is distributed. Hence, with disease of the cornua results degeneration of the nerve and wasting of the muscle. The muscular fasciculi become reduced in size and ultimately totally disappear. This is associated with a certain amount of interstitial overgrowth. In the transition referred to, certain fasciculi assume the yellow, glassy appearance known as waxy degeneration. The sensibility of the muscle, if the sensory nerve is intact, becomes increased, and there may be pain, partly due to compression of the nerves by morbid contraction, and partly to a morbid sensitiveness of the nerve endings and to the interstitial inflammation. The recovery of the nerve is not only followed by gradual restoration of its power over the muscle, but also by restoration of the nutrition and redevelopment of the muscle. For this, however, much time is required, and it often remains permanently smaller than normal.

Lesions of motor nerves, whether inflammatory or traumatic, are followed by similar results,—degenerative atrophy of nerve and muscle because of interference with the conduction of the trophic influence. In all cerebral palsies, and in such spinal paralyses in which the lesion is above these ganglion cells, there is no such wasting, and no reaction of degeneration is developed, because the nutrition is maintained by the intact cell body of the lower neuron. Such palsies are "non-atrophic," as contrasted with the atrophic palsies due to lesions in the anterior cornua or peripheral nerves.

From the above the diagnostic and prognostic value of the reaction of degeneration is at once apparent. The seat of the lesion, whatever its nature, is easily determined; while we are also informed that recovery, though not impossible, must be long delayed because of the extensive repair necessitated in muscle and nerve. Much experience with the use of electricity should, however, be had before the physician permits himself to draw conclusions.

II. Sensory Phenomena.—Under this head naturally fall first the subjective sensations of the patient. These include, strictly speaking, only the various modifications of sensibility appreciable to him alone and independent of external impression, preëminently pain; also those peculiar modifications due to internal irritation as contrasted with external impression, and known as *paraæsthesias*, viz., numbness, tingling, prickling, formication, or a feeling as of ants crawling over the skin; also a sensation like that of the contact of wool or fur—a furry feeling—vertigo, tinnitus aurium or ringing in the ears, and a sense of unpleasant odors or tastes. Headache in one of its numerous modes of manifestation is another subjective sensation, which occurs in numerous morbid states of the nervous system.

After these come modifications of the different varieties of cutaneous sensibility as influenced by external impressions. They are of the nature of increase or decrease, the former being known as *hyperæsthesias* and the latter as *anæsthesias*, the latter being further characterized as partial

or complete. To the latter the term paralysis of sensation, partial or complete, is also applied.

(*a*) *Tactile sensibility*, the sense of touch or pure contact, is usually first investigated. The simplest method is by the touch of a finger or other blunt object near the same temperature as the body, for both heat and cold must be eliminated in this test. The patient should be directed to close his eyes or avert his head. More refined measures are the application of rough, smooth, or coarsely uneven surfaces. More delicate still is the æsthesiometer, essentially a pair of compasses with blunt and sharp points and graduated quadrant attached, by which the distance between the two points is accurately measured. By this instrument, in connection with a normal standard of relative sensibility worked out by E. H. Weber, the degree of impairment in delicacy of touch may be measured. Closer approximation may be recognized if the two points of the compasses are put down one after the other and varying the test by touching the same place twice or a different place each time. Weber's table is as follows :—

Minimum distance at which the two points of a pair of compasses in contact with the skin may be recognized as two points :—

Cheek, 11 to 15 millimeters.
Tip of the nose, 6 millimeters.
Forehead, 22 millimeters.
Tip of the tongue, 1.2 millimeter.
Back of the tongue and on the lips, 4 to 5 millimeters.
Neck, 34 millimeters.
Upper arm, 77 millimeters.
Forearm, 40 millimeters.
Back of the hands, 31 millimeters.
Backs of the fingers, 11 to 16 millimeters.
Tips of the fingers, 2 to 3 millimeters.
Back, 55 to 77 millimeters.
Chest, 45 millimeters.
Thigh, 77 millimeters.
Leg, 40 millimeters.
Instep, 40 millimeters.

These figures can, however, only be used within limits, as they are by no means constant for different individuals, or, indeed, for the same individual at different times. Marked deviations from them may, however, be accepted as indicating derangements of tactile sense.

(*b*) *Sense of Locality.*—This is tested in the same way as the tactile sense inquiring of the place touched. While cutaneous sensibility may remain intact, the sense of locality may be seriously deranged. Thus, a patient may think he is being touched in the leg when the contact is with the foot.

(*c*) *The sense of pain* is of equal importance with that of pure touch, because these two not infrequently fail to diminish or increase *pari passu* in morbid states. Parts insensible to touch may respond decidedly to painful impressions. Pain is most easily investigated by pricking with a pin or pinching a fold of skin, by painful electrical currents or painfully hot metals. The special term *analgesia* is applied to loss of sense of pain while the tactile sense is preserved. Analgesia exists in peripheral and central nervous disease and in syringo-myelia.

Tenderness or pain on pressure in the course of nerves should be studied in connection with the sense of pain. It is found in nerves which are the seat of inflammation, especially in sciatic neuritis and multiple neuritis. It is one of the symptoms of lead-poisoning, also of arsenical poisoning.

(*d*) *Variations in the sense of pressure* are less often tested. Such variations may be roughly measured by pressure with the hand. Following Weber's studies, also, differences in the sense of pressure are estimated by the amount required to be added to an existing weight on the skin before the addition is appreciated. Thus it has been ascertained that in health an addition of $\frac{1}{20}$ or $\frac{1}{30}$ to an existing weight can be appreciated. Thus, if a weight of 95 gm. be placed on the skin, an addition of a single gram will not be recognized, but nearly five gm. must be added before the increase is appreciated, while if considerable more than this is necessary it means that the sense of pressure is less delicate. Sufficiently accurate measures are coins of different weights. Temperature must be eliminated by placing non-conducting substances between the weight and the skin, while the part to be tested must also be supported.

It is not unusual to find, in paralysis of the sense of pressure, failure to recognize a doubling and even tripling of weights. It is more especially in locomotor ataxia that such paralyses are found while the tactile sense proper is intact, a light touch of the skin being felt while a considerable pressure is not appreciated.

(*e*) *The sense of temperature* may be roughly tested by ascertaining the power of the patient to discriminate between the warm breath close to the skin and the cooler current produced by blowing from a distance. More precisely, the sense of temperature is studied by testing the ability to recognize differences in the temperature of flat-bottomed test-tubes filled with water of different temperatures and brought into contact with the skin. The therm-æsthesiometer has been devised by Eulenburg for the same purpose, but the student is referred to works on nervous diseases for its description.

In health, differences of ½° to 1° F. (0.27° to 0.55° C.) may be recognized on the fingers and face at temperatures from 80° to 100° F. (26° to 37° C.), while on the back differences to be recognized must amount to 2° F. (1° C.).

In disease, we sometimes note complete loss of sense of temperature, while the skin appreciates other forms of irritation, and again this state of affairs is precisely reversed. This occurs sometimes in ataxic states and in syringo-myelia. Strümpell has called attention to a peculiar reversal of the sense of temperature, as the result of which cold objects appear warm. This has been noted in disease of the medulla oblongata, in locomotor ataxia, and in syringo-myelia.

(*f*) *Delayed conduction of sensory impressions* represents a form of modified sensibility of which after-sensations are a further subdivision. In delayed conduction an irritation, more particularly a painful one like the prick of a pin, is noticed by a patient after an appreciable interval, whereas in health the recognition is instantaneous so far as the unaided perception is able to judge. Touch and pain may even be thus separated, the immediate contact of the pin being promptly recognized, while the sense of pain presents itself a few seconds later. It is likely, also, that the sense of touch may be delayed, but such delay is not appreciable to the unaided perception.

An *after-sensation* is a prolonged sense of pain which succeeds a

momentary impression. Such is the prolonged burning on the sole of the foot which sometimes succeeds the prick of a pin, or which may occur one or more times after a short interval, as if additional pricks had been made.

These abnormal sensations occur also in diseases of the spinal cord, and especially progressive locomotor ataxia.

(*g*) *The muscular sense* is that sense through which we become aware of the position of any of our limbs without the aid of vision as well as of any degree of motion by them. It is thought probable, however, that the sensibility of the articular surfaces, ligaments, tendons, and skin aids the sensibility of the muscles in furnishing this information. This power is diminished in nervous diseases and is tested by having the patient first touch a certain object with his eyes open and asking him to repeat the act with the eyes closed.

The muscular sense is not only thus estimated, but the strength required to lift a leg or arm, more plainly evident when one is tired, is also measured through the muscular sense. It is the muscular sense which causes the paretic to say that his leg feels heavy. By the muscular sense, too, or by the "sense of power," we estimate the amount of strength demanded by any muscular contraction and thus measure the difference in weight of objects, eliminating, however, the sense of pressure, which may be done by suspending the object in a towel.

In locomotor ataxia, as well as in paralysis of cerebral origin and in cortical lesions, the muscular sense is defective; also in hysterical affections.

Anæsthesia is said to be peripheral, spinal, or cerebral, in accordance with the seat of the broken conduction between the terminal apparatus of the sensory nerves and the cerebral cortex. *Peripheral* anæsthesia occurs after chilling of the skin, the action of ether, cocaine, aconite, veratria, as well as corrosive substances like acids, alkalies, and carbolic acid. Spasm of the small vessels, forming the so-called spastic anæmias, is also attended by anæsthesia. The anæsthesias of washerwomen who have their hands in water all day long may belong to this class. Injuries of nerve trunks may also cause anæsthesia, known as the "peripheral anæsthesia of conduction." Pressure, inflammation, and degeneration of nerve trunks are included among such causes. The *paræsthesias* alluded to—numbness, formication, and tingling—are among the effects of such lesions of nerve trunks. *Spinal* anæsthesias are found, especially in connection with disease of the posterior roots, posterior columns, and posterior cornua of the cord. Such a disease is especially locomotor ataxia. Anæsthesia is found, however, also in inflammations of the cord, acute and chronic, and where there is pressure on the cord from hemorrhage into the spinal canal or pressure by diseased or broken vertebræ or tumors. Such anæsthesia is usually bilateral and known as para-anæsthesia. *Cerebral* anæsthesia occurs as the result of hemorrhages, softening, or tumors, which impinge on the posterior third of the posterior limb of the internal capsule, in which the sensory fibres pass upward and thence through the tegumentum to the cerebral cortex. If the cerebral anæsthesia affects half of the body it is known as hemianæsthesia, and the half of the body affected is

opposite the hemisphere of the brain in which the lesion lies, since the sensory fibres also decussate in their course to the periphery, not, however, in one spot, as the motor fibres, but most of them throughout the cord very soon after their entrance by the posterior roots.

The hysterical anæsthesias and anæsthesias due to the action of narcotics and anæsthetics are regarded as cerebral in their action. Those succeeding such acute infectious diseases as typhoid fever have been ascribed to both peripheral and spinal origin.

III. Vaso-motor and Trophic Phenomena.—We pass next to the study of *vaso-motor* and *trophic* alterations. Two sets of vaso-motor nerves have been demonstrated by physiologists—the vaso-constrictors and vaso-dilators—the former contracting the arteries when stimulated and permitting their dilatation when paralyzed. The vaso-dilators are influenced in an opposite manner by the same agencies, but their number as far as proven is not numerous, as they include up to the present time only fibres in the chorda tympani, nervi erigentes, and sciatic. Blushing may be the result of stimulation of vaso-dilators. Moreover, pathology has as yet failed to separate lesions of the two sets of nerves and their consequences, and the latter are generally looked upon as results of *paralysis* or of *irritation* of vaso-constrictors. Instances of the former are redness, a feeling of warmth, and sometimes an actual elevation of temperature, sweating, all in circumscribed areas or half the body. They may persist or intermit. Instances of vaso-motor irritation are pallor, coldness, accompanied by stiffness, formication, and even pain. These are the phenomena of vaso-motor spasm. A more or less permanent condition of the hands sometimes results, characterized by a blueness or mottled appearance accompanied by a lowered temperature, further augmented by external cold. Still higher degrees are said to have produced circumscribed gangrene (Raynaud's disease).

Symptoms of vaso-motor paralysis occur in connection with cerebral and spinal lesions and with injuries of the sympathetic system and nerve trunks which include vaso-motor fibres. The essential causes of vaso-motor spasm are less easy to locate. It is found associated with prolonged convulsive seizures and in angina pectoris at the beginning of the attack, as if caused by irritation of the sympathetic ganglia in the heart.

That *trophic* or nutritive phenomena are closely allied to vaso-motor phenomena is commonly admitted. That they are under the control of the same nerves is doubtful, although the proof of the existence of separate trophic nerves is still wanting. Vesicular eruptions in the area of distribution of nerves, such as herpes zoster, certain atrophic skin diseases, pigmentations and depigmentations, such as morphœa, Addison's disease and vitiligo, scleroderma and the glassy skin which succeeds certain injuries to nerve trunks, are illustrations of trophic influences. Similar are the changes in the skin, hair, and nails, as the result of which the first becomes dry, the second is lost or becomes rapidly grey, and the latter grow brittle or thicken or drop off. The latter events are the result of spinal and even cerebral lesions. The circumscribed œdemas known as acute angioneurotic œdema and the more permanent condition

of myxœdema are also probably trophic. So also are the atrophies which result from disease of the cells of anterior horns of the grey matter of the cord, or injuries to nerves by which they are essentially cut off from the trophic cells; also unilateral facial atrophy, including even atrophy of bone, and the still more remarkable spinal *arthropathies* of Charcot, as the result of which the joints enlarge or become the seat of effusions and stimulate even the joints of acute articular rheumatism.

Finally, there is the acute bed-sore or eschar, so well described by Charcot,* beginning in an erythematous patch on which bullæ and blebs are rapidly developed, quickly succeeded by gangrene. While pressure or irritation may be necessary to the production of these sores as exciting causes, there can be no doubt but that they are more easily invited in spinal paralyses than in non-paralytic conditions. Such phenomena may be caused by cerebral lesions, lesions in the medulla oblongata, spinal cord, and sympathetic nerve.

It is well known that the vaso-motor nerves surrounding the various blood-vessels are derived from the sympathetic trunks, which, in turn, receive their vaso-motor filaments mainly from the anterior roots of the spinal nerves, the anterior columns of the spinal cord, and the anterior motor tracts of the medulla oblongata, into which they come down from the cortex of the brain. Thus the vaso-motor nerves are evidently a part of the motor system, but their presence among sensory filaments is also probable.

IV. Mental Phenomena.—Under this head come the phenomena of consciousness or unconsciousness, coma, the state of the will, the various perversions of mental process, including delirium, hallucinations, delusions, illusions, and insane acts. *Hallucinations* are states of the mind in which the subject conceives that he perceives external objects which do not exist. The victim of delirium tremens who conceives he is pursued by monsters of various sorts is the subject of hallucinations. A *delusion*, on the other hand, is a false idea without sensory impression. The deluded person imagines that he is the happy possessor of millions when he is actually a pauper, or complains of poverty although affluent. An *illusion* is based upon an actual perception, but an erroneous impression arises therefrom. It differs from a hallucination in that in the latter no object is actually seen; there is no sensory impression. The impression of relief obtained on looking at a picture in the stereoscope is an illusion; and Gowers adds that if used in connection with morbid mental states it should be confined to false ideas and images, the erroneousness of which the patient recognizes.

Delirium is the more or less acute manifestation of one or all of these perversions of mental process, associated with muttering or active speech suggested by them or with action growing out of them. Thus constituted, delirium may be the result of toxic states or acute disease other than of the brain.

The same perversions of mental process continued and without fever

* "Lectures on Disease of the Nervous System," Philadelphia, 1879.

constitute *insanity*, which is probably always associated with structural change in the brain or its membranes, although such may not be always demonstrable. Other symptoms are, however, added in insanity, such as extreme depression of spirits, while hallucination, delusion, and illusion may be present in various degrees. Special insane acts should be specified and modifications of normal sleep noted.

V. Alterations in Vision and Hearing.—In addition to the ordinary defects of vision, the *response of the pupil to light* should be noted; also its *accommodating power*. The former is absent in three-fourths of all cases of locomotor ataxia, while the latter remains. The pupil thus failing to respond to light, but retaining its accommodation to distance, is known as the Argyll-Robertson pupil. Each eye should be tested separately, the other being covered. Finally, the eye-ground should be examined in every exhaustive study of a nervous case.

Modifications in *hearing* are of the nature of increased and diminished intensity, and there is that very common symptom known as tinnitus aurium, or ringing in the ears, already alluded to as a good instance of a subjective symptom. Hyperacusis of hearing occurs in association with augmented acuteness of the other senses in acute affections of the brain or where there is hyperæmia of the brain from any cause. It is often complained of also in hysteria. Deafness, on the other hand, is rarely the result of nervous disease, but is the consequence rather of diseases of the ear itself. Ringing in the ears occurs in many conditions, known and unknown. While some more than usual impression on the nerve is a condition of tinnitus, it by no means follows that the cause resides in the nervous system. In addition to the numerous forms of irritation due to ear disease, the blood in an adjacent vessel may be thrown into vibration and produce an audible murmur. On the other hand, tinnitus is sometimes due to intra-cranial irritation either of the nerve or the auditory centres.

VI. Alterations in Breathing and Pulse.—Alterations of breathing are very common in nervous diseases. Respiration may be rapid or slow, and labored and sighing, or irregular, but especially peculiar is the so-called *Cheyne-Stokes breathing*, in which, succeeding a long pause, so long sometimes that it seems as though the patient would never breathe again, follows gentle and shallow respiration, which gradually grows deeper and more frequent until an acme of dyspnœal breathing is reached, when it again diminishes in depth and frequency until the pause again occurs. During the pause the pupil often contracts and the heart's action becomes less frequent. It may occur in tubercular meningitis, cerebral hemorrhage, in embolism, and thrombosis of the basilar artery; also in uræmia, heart disease, and more rarely in other conditions, including the infectious fevers, where the respiratory centre is influenced. The period of arrest varies from five to forty seconds, and the duration of each cycle may be from fifteen to seventy-five seconds, and may vary..

A modification of Cheyne-Stokes breathing is a form in which there

are periods of deep and energetic breathing which begin suddenly, in which the respirations gradually lessen in depth until they cease and after a pause energetically recommence.

Cheyne-Stokes breathing was ascribed by Walsh and later by Traube and Rosenbach to lessened excitability of the respiratory centre in the medulla. Filehne suggested that the rhythmnogenic purpose of the respiratory centre is modified by a periodical vaso-motor spasm due to stimulation of the vaso-motor centres by the asphyxiated blood. The irritability of the respiratory centre being lowered, it is less excitable than the vaso-motor centre. The arterial spasm in the medulla prolongs the stimulation of the respiratory centre as well as that of the vaso-motor centre by hindering the access of oxygenated blood. The respiratory movements therefore continue energetic after the blood has become oxygenated. The gradual onset of the breathing may be due to the fact that the reactive vaso-motor dilatation exceeds the normal, and thus the quantity of blood reaching the respiratory centre lessens the stimulating influence of its quality.

Changes in the order of investigation proposed in this section will, of course, be demanded by circumstances, while at times certain steps may be altogether omitted.

AFFECTIONS OF THE PERIPHERAL NERVES.

NEURITIS.

Definition.—Neuritis, or inflammation of a nerve, may be confined to a single trunk, whence it is called *localized;* or it may involve a large number of nerves, when it is known as *multiple* neuritis or *polyneuritis.* In *perineuritis* the connective tissue surrounding a nerve is the seat of the inflammation; in *interstitial* neuritis, the tissue between the bundles of nerve-tubules is involved, and in *parenchymatous* or *degenerative* neuritis the substance of the nerve-tubules themselves is affected.

Localized Neuritis.

Etiology.—Cold is the most frequent cause of neuritis, and the nerve most frequently thus affected is the facial. Trauma is another cause, including contusions, stabs or cuts, or stretching and laceration such as occur when there is dislocation, fracture, and other violent injury. Pressure is also a cause, and even muscular contraction is sometimes sufficient to act in this manner. Neuritis may also occur as the result of extension by contiguity, as from caries in a bone or foramen through which the nerve passes, adjacent joint inflammation, pleurisy, and meningitis. Finally, neuritis may be caused by toxines and morbid states of the blood, such as produce the infectious and constitutional diseases, of which may be mentioned the toxine of diphtheria and the causes of syphilis and gout. The mineral poisons, especially lead and arsenic,

are frequent causes. Alcohol is also a cause of this kind of neuritis, although it more frequently produces multiple neuritis.

Morbid Anatomy.—An actually inflamed nerve is reddish from hyperæmia of the vasa nervorum, though the stage of demonstrable hyperæmia may have passed away when the nerve comes under observation. In perineuritis and interstitial neuritis the primary change is in the connective tissue—in the former, an infiltration of the nerve-sheath with leucocytes, and in the latter, of the interstitial tissue with lymphoid cells. There may even be minute extravasations of blood. These changes are more apt to be in places along the course of the nerve where it is exposed to special irritation, as in passing through foramina or over bone. The lymphoid cells gradually become fusiform cells, resulting in the formation of true connective tissue. The pressure of this new tissue gradually destroys the nerve itself, the medulla being gradually broken up into drops which subsequently disappear, while the nuclei of the sheath of Schwann also increase. Finally, the axis cylinder also becomes granular and disappears, all this in varying degrees. The nerve-fibre may be substituted by a fibre of connective tissue, in which there may be a deposit of fat, seen in the lipomatous neuritis of Leyden.

In parenchymatous neuritis the primary change is in the nerve tubule itself. Here the medulla and axis cylinder are first involved, the former breaking up into drops as described, and the latter into granules, both ultimately disappearing, while the interstitial connective tissue remains comparatively intact. But the nuclei of the sheath of Schwann proliferate and become a part of the interstitial connective tissue. The muscles connected with the inflamed nerve also atrophy, in the case of the motor nerves, at least, being practically cut off from their centre of nutrition. The change in the nerve is essentially the Wallerian change noted in the nerve-tubules of a cut nerve, the motor nerves being degenerated in their distal and the sensory in their proximal ends. In some instances the changes noted in the sheath of Schwann extend over into the interstitial tissue of the muscle, constituting the *neuritis fascians* of Eichhorst.

Symptoms.—There is not much constitutional disturbance in localized neuritis, though the thermometer may show some rise of temperature. *Pain*, especially pain on motion, and *tenderness* are the salient symptoms. The pain may be confined to the seat of the inflammation or may involve the distribution of the nerve, or the whole limb may be involved. It varies in degree and also in character, being sometimes burning and at others aching, boring, or shooting. It is apt to be worse at night, and in positions involving pressure on the nerve itself. The nerve may be appreciably swollen, and even rarely the skin over it reddened.

The pain in the trunk of an inflamed nerve is probably due to pressure on the nervi nervorum. Weir Mitchell has especially called attention to this, although an interesting fact would appear to militate against it —that nerves composed almost purely of motor fibres are less tender than sensory nerves. Its acknowledgment would imply that fewer sensory nerves are distributed to the motor nerve trunks than to sensory nerves. Mitchell also describes elevation of surface temperature and trophic dis-

turbances, such as sweating, herpes, and effusion into neighboring joints. Other motor disturbances, including twitchings and contractions, are noted. Trophic derangements, including muscular wasting, associated with peculiar "glassy skin" or slight œdema, may be present. Or there may be thickening of the skin and a condition resembling ichthyosis. Ultimately the hyperæsthesia and paræsthesia may become anæsthesia, though usually limited to small areas.

The course of the disease is variously prolonged. Many acute cases terminate favorably in a few weeks. More cases become chronic, extending over months and even years, after which they may gradually subside.

A rare variety is "ascending neuritis," in which the inflammation extends from smaller to larger branches, until finally may be involved most of the nerves of a limb, or even the spinal cord, with resulting myelitis, with or without spinal meningitis. Paralysis may result from such condition. Such is possibly the paralysis that succeeds visceral disease, as that of the bladder. Even the corresponding nerves of the other side may be involved either through the cord or through its membranes, or without such intermediation, constituting sympathetic neuritis.

The electrical condition of the nerves and muscles must be studied. It may be normal in slight cases. In more severe cases there may be the reaction of degeneration, with the slow, lazy contraction of the muscles, the reversed reaction of health to opening and closing currents, described on page 806.

Diagnosis.—The diagnosis is chiefly from neuralgia. This depends upon the pain and tenderness in the course of the nerve and the limitation of the symptoms to its distribution, though it may be less diffuse at the onset, and in this respect more like rheumatism. Neuralgia is more intermittent, and is relieved by pressure rather than aggravated thereby. The presence of the paræsthesias points to neuritis, confirmed by ultimate lessened sensibility. In neuralgia nerve and muscle reactions remain normal. The distal pain of central spinal disease must be separated.

Prognosis.—The prognosis varies greatly, being favorable in mild cases and most traumatic cases. Cases consequent upon local suppuration are the gravest. In ordinary cases from cold or contusions recovery ensues, sooner or later, although some cases last a long while and recurrences are not unusual, especially in neuropathic dispositions, in which, too, recovery is slower.

Treatment.—Here, as elsewhere, if a cause is discoverable it should be removed. Exposure to cold and dampness should be removed, pressure by cicatricial tissue or dislocated bones and constitutional states favoring neuritis, such as gout and syphilis, should also be corrected.

Of curative measures, rest is the most important. Where a limb can be splinted it should be, pressure by muscular contraction being thus obviated. Cold may be a useful application, as by an ice-bag. In other instances heat, now dry and again moist, subserves a useful purpose. A blister or blisters may be applied over the tender nerve. But especially convenient is the Paquelin's cautery, which should be used earlier than is usual;—it is so instantaneous and painless after the application for a few

minutes of ice and salt to the spot to be burned. Morphine is sometimes indispensable, and the hypodermic method of application is best, $\frac{1}{6}$ to $\frac{1}{4}$ grain (0.011 to 0.165 gm.) for an adult. But the morphine habit is easily acquired and the patient should not be allowed to use the syringe himself. Cocaine may be similarly used, $\frac{1}{10}$ to $\frac{1}{3}$ grain (0.0066 to 0.022 gm.), and Gowers recommends it highly, more particularly for its power in arresting local transmission of the impulses that cause pain. Here, too, the injection should be made by the physician or a trusted attendant at the seat of the pain. This authority also considers mercury a most efficient agent, in the shape of a blue pill, a grain (0.066 gm.) once or twice a day, associated, if necessary, with morphine, the constipating effect of which it counteracts. Salicylate of sodium is sometimes useful, as is also more rarely iodide of potassium.

In the chronic form also, Paquelin's cautery should be repeatedly used, or, if not at hand, the blister may be substituted. Electricity here comes into play, and galvanism is the form to be used, the positive electrode being placed over the nerve or seat of pain, and the negative indifferently placed. A weak current should be used, but its strength may be increased if the weak current is inefficient. The application should continue about ten minutes. The wasted muscles usually recover with the nerve, but massage and galvanic electricity help them. Faradism is less favorably regarded, especially in the active stage.

SPECIAL VARIETIES OF NEURITIS.

Two varieties of neuritis possess such a distinct symptomatology that they deserve special mention. They are brachial neuritis and sciatica.

Brachial Neuritis.

Definition.—This term is applied to an inflammation of the brachial plexus. The condition is essentially a perineuritis, an inflammation of the sheaths of the branches that form the brachial plexus. The term *radicular neuritis* is applied to a sub-variety in which the symptoms in their distribution suggest that the nerve roots rather than the plexus are the seat of the inflammation, the view being further supported by the presence of pain about the spine, although Gowers, on whose authority this sub-variety is mentioned, admits that it has as yet received no anatomical confirmation.

Brachial neuritis occurs by preference in gouty subjects, is about as frequent in women as in men, and five-sixths of all cases are over fifty years of age. Muscular rheumatism, especially lumbago, and sciatica are almost invariable previous conditions.

Symptoms.—*Pain* of the usual character in the course of the nerve distribution, on the wrist, the back of the forearm, in the plexus itself, above the clavicle, in the axilla, or in the region of the scapula may be the first symptom, and from these points it may extend throughout the course of the nerve. The pain is usually intermittent in its first visitation, or felt during certain movements, but it may be suddenly severe.

Motion almost invariably increases it. It has at times a peculiar tendency to radiate to the side corresponding to the arm affected. There is usually associated with it sensitiveness and very rarely loss of sensation. The *loss of sensation* in connection with pain constitutes *anæsthesia dolorosa*. As in other forms of neuritis, there may be *slight atrophy* and corresponding muscular weakness. So also the skin may become shining and there may be subcutaneous œdema and arthritic changes in the fingers and joints, more frequently in cases occurring late in life.

Diagnosis.—The disease is rare, occurs late in life, and most resembles neuralgia, from which it is distinguished by the persistent tenderness of the nerves, the effect of motion, and the history of the attack, which may elicit the fact of injury to the nerve. The tendency to radiate to the left side suggests angina pectoris, and there is even a tendency to association of cardiac distress, but there is no tenderness in the course of the nerve in the latter disease. Primary joint affection, with secondary neuritis and atrophy of muscle, is not to be mistaken for the similar effect of brachial neuritis. The muscular wasting is at most slight.

Prognosis.—It is a tedious malady, lasting for months and even a year or more, while recovery is sometimes incomplete. The limb remains small and feeble and the joints are fixed from perverted nutrition and adhesion between articular surfaces. This may occur in all the joints. Recurrences and relapses are liable to happen.

Treatment.—The treatment is precisely that of the simple forms of neuritis and need not detain us.

Sciatica.

Definition.—This term is applied to all painful affections in the distribution of the sciatic nerve, some of which may be neuralgic, but the vast majority are inflammatory, or, rather, peri-neuritic, as it is the sheath of the nerve that is usually involved. Such painful affection of the nerve may be due to pressure by tumors or other causes within the pelvis.

Etiology.—Sciatica is far more common in men than in women, in the ratio of about four to one. It will be remembered that brachial neuritis affects both sexes about equally. It is also a disease of adults, being unknown in children and very rare in the second decade. It is most frequent between twenty and fifty, next between fifty and sixty, and next between thirty and forty.

Gout and rheumatism are favoring causes, especially fibrous rheumatism. Very rarely syphilis may be a predisposing cause. Exposure to cold is the most frequent exciting cause, especially after severe muscular exertion; while standing in water, sitting or lying on the cold ground, and the like are frequent causes. Exposure to drafts, though less frequently than in neuritis of the upper extremity, is still a cause. A sciatica may also arise by extension from a rheumatic focus, especially that form of lumbago involving the fibrous attachments of muscles at the back of the sacrum, whence the inflammation extends to the sheath of the sciatic nerve. Pressure by mechanical agents and possibly muscular contraction may be a cause. In addition to the intra-pelvic causes alluded

to, secondary sciaticas may be caused externally to the pelvis by bone disease and other foci of suppuration.

Morbid Anatomy.—The morbid changes are those already described under neuritis.

Symptoms.—The leading symptom is, of course, *pain in the course of the nerve*. Felt first and most commonly in the back of the thigh, it also travels above the hip-joint in the sciatic notch, behind the knee, below the head of the fibula, behind the internal malleolus, and on the back of the foot. It may be more diffuse, but the course of the main trunk of the nerve is often indicated by it, and the spots above named will often be pointed out by the patient as special seats of tenderness. It usually begins gradually, but it may start up suddenly, especially in cases of rheumatic origin. Movement, especially walking, and positions in which the nerve is in a state of tension or compression aggravate it. The characters of the pain are those already described under neuritis. The other rare symptoms of neuritis may also be present, as herpes, œdema, wasting, but the reaction of degeneration almost never. Very rarely, too, the neuritis ascends to the spinal cord.

Diagnosis.—This is not difficult, although a careful study should be made of each case with a view of determining its primary or secondary origin. Pelvic tumors, especially in women, and rectal accumulations should be sought for. Lumbago, hip disease, and sacro-iliac diseases are all to be thought of. But in none of these is there pain on pressure in the course of the nerve. In the latter only is there sometimes pain in the posterior part of the thigh. Pain felt only in the outer side of the thigh is not sciatica. The rare cases of sciatic neuralgia are not characterized by tenderness. They occur in persons subject to neuralgia, and the pain is not influenced by position and motion. It is purely spontaneous. Disease of the vertebræ, cauda equina, and even of the spinal cord may produce sciatic pain ; but here, again, there is no tenderness in the course of the nerve, the pain is peripheral, and is more apt to be bilateral. Bilateral pain is strong presumption against sciatica, pointing rather to disease of the nerve roots. The shooting pains of tabes dorsalis are like those of sciatica, but the other symptoms of the former disease are present.

Prognosis.—Cases of sciatica, however obstinate, sooner or later get well, although they may persist for months. A case has recently come under my observation which has lasted seven years.

Treatment.—Every case of sciatica should be at once put to rest, and the more complete the rest the sooner the recovery. Splinting of the limb, as recommended by S. Weir Mitchell, is necessary in some cases and probably would hasten cure if used at once, but it is so inconvenient that the temptation to temporize is very strong. Rest being secured, I am confident that recoveries would be prompter if Paquelin's cautery were oftener used at the onset. Counter-irritation by blisters, mustard, and iodine are all relatively inefficient. Treatment by cold along the course of the nerve certainly relieves the pain for the time, but in my experience the relief thus obtained is not permanent.

First, attention should be paid to the local causes, if these are dis-

coverable, and to constitutional causes as well. If of rheumatic or gouty origin the salicylates will be found useful, in other cases useless. Here, as in neuritis from other causes, Gowers, whose large experience commands respect for his suggestions, commends the pill of blue mass, one grain (0.006 gm.) twice daily, where there is active inflammation.

For the relief of mild degrees of pain phenacetin and antifebrin, and especially a combination of phenacetin and caffein citrate, say ten grains (0.660 gm.) of the former and three grains (0.198 gm.) of the latter, are often efficient. For severe degrees morphine is necessary, and is best given hypodermically in doses of $\frac{1}{4}$ to $\frac{1}{8}$ grain (0.0165 to 0.008 gm.). The danger of the possible morphine habit must always be kept in mind, and cocaine should be tried first as a deep-seated injection in doses of $\frac{1}{4}$ to $\frac{1}{8}$ grain (0.0165 to 0.008 gm.). Acupuncture over the course of the nerve is of service for the same purpose of relief of pain rather than cure. Anodyne liniments may be used, and, although not curative, do give some comfort and meet the wishes of the patient that something should be done.

Change of scene in chronic cases is often of advantage, and if associated with thermal-bath treatment may accomplish a cure in otherwise obstinate cases. The mud bath is a measure of treatment applied in Europe with some success. In the chronic stage electricity, in the shape of galvanism, also meets the demands of patients and friends and may do some good. Nerve-stretching may be tried as a last resort. I have had it done in cases with uncertain result.

MULTIPLE NEURITIS.

SYNONYMS.—*Polyneuritis; Peripheral Neuritis.*

Definition.—An inflammatory condition involving many nerves, either at one time or in rapid succession.

Historical.—Multiple neuritis is a disease of modern recognition. The symptoms peculiar to the condition were described first, probably, by James Jackson, Sr., of Boston, Mass., as early as 1822. In 1854 Robert Bentley Todd, of London, wrote of lead palsy: "The nervous system is thus first affected at its periphery, in the nerves, and, the poisoning influence continuing, the contamination gradually advances toward the centre." Duchenne also described the symptoms fully in 1858. Samuel Wilks described alcoholic paraplegia, but the existence of multiple neuritis as an actual disease was first demonstrated by Duménil, at Rouen, in 1864, further contributed to by Joffroy in 1879, Leyden in 1880, Grainger Stewart in 1881, Buzzard in 1886, James Ross, Henry Hun, and Charles K. Mills in 1892. W. R. Gowers' article in the second edition of "Diseases of the Nervous System," 1891, is a very complete one.

Etiology.—The causes of multiple neuritis are numerous, and by no means easy of classification. They include:—

(1) The commonly acknowledged poisons introduced from without. (*a*) Organic, including alcohol, by far the most frequent cause, ergot,

morphine, ether, carbon-monoxide, carbon-bisulphide, benzine and its products, and aniline. (*b*) Inorganic, including lead, arsenic, phosphorus, and mercury.

(2) Organic matter generated in the organism by chemical changes. Such is the cause of the neuritis of diabetes mellitus, whether oxybutyric acid, diacetic acid, or acetone, fully discussed in treating diabetes.

(3) Poisons inherent to the infectious diseases, whether the direct cause of the disease or a product of such cause. Instances of the former are malarial neuritis, leprous neuritis, beri-beri, or so-called endemic neuritis, also, probably, the neuritis of acute infectious jaundice (Weil's disease). In these instances the cause is an organism. Of the latter diphtheritic neuritis, septicæmic neuritis, the neuritis of small-pox, typhoid fever, tuberculosis, and possibly syphilis are instances. The cause is here a toxin generated by an organism.

(4) Intrinsic states of the blood of undetermined nature, with which cold may or may not coöperate as an exciting cause, viz., rheumatism, gout; also the puerperal state and chorea. Advanced microbic doctrines would place rheumatic neuritis in (3), while a greater conservatism might place septicæmic neuritis in (4). It is not impossible that cold alone may, by its operation, generate a poison capable of producing a poly-neuritis.

(5) Cachectic and senile states, the result of malnutrition, such as is inherent to old age, cancer, tuberculosis, and wasting diseases generally.

General Etiology.—Multiple neuritis is a disease of adults. The only form met with in children is a complication of acute polio-myelitis, of which Gowers says it "may, perhaps, now and then be met with apart from the spinal malady as an infantile variety of multiple adventitial neuritis irregular in distribution." The remaining chief forms occur usually between the ages of twenty and fifty, the alcoholic between thirty and forty or later, and senile neuritis at still later age. The alcoholic form is more frequent than all others put together, and of this form 70 per cent. occur among women. More than one cause may coöperate, when one may be the predisposing and the other the exciting. Cold probably most frequently plays the latter rôle, but there may be others, such as depressing emotions, anæmia, and the like.

Morbid Anatomy.—The special character of multiple neuritis is that it is parenchymatous as contrasted with interstitial and perineural. That is, the changes begin in the nerve-tubules themselves, as described on p. 816, rather than in the connective tissue between and around them. Yet this is not invariable. In rare instances the interstitial tissue is most involved and in others the two processes are associated. It is further characteristic of multiple neuritis that the involvement is symmetrical, that is, the corresponding nerves on the opposite side of the body are affected. The more this is the case the more likely is the change parenchymatous. The changes are also more marked in the peripheral distribution of the nerve than in the trunk of nerves. Macroscopic changes are very rarely appreciable. In acute changes the nerve may be swollen, reddened, hemorrhagic, or in old cases softened.

Symptoms.—The symptoms vary greatly in different varieties of neuritis, but there are some more or less common to all forms, particularly illustrated, however, by the alcoholic and rheumatic varieties. These will be considered first, and afterward some special features of varieties due to specific causes, particularly the metallic poisons, the acute infectious diseases, and beri-beri.

The symptoms are easily divided into three classes: Motor weakness, sensory derangement, and incoördination. The first is the result of the involvement of motor nerves, and manifests itself usually first in the extensors of the wrists and fingers, flexors of the ankle, and extensors of the toes. The sensory disturbances are tingling, numbness, pain, while the incoördination resembles that of the slightest degree of locomotor ataxia. According as one or the other of these sets of symptoms predominates, we have a motor form, sensory form, and ataxic form.

The onset of the rheumatic form, or that due to cold, is more usually sudden, with chill and fever and temperature of 103° and 104° F. (39.5° to 40° C.), headache, and backache. The slow onset is characteristic of alcoholic neuritis, though it may be precipitated by some exciting cause, as cold, exposure, fatigue, or some toxic state. It is rarely febrile. To this initial stage succeed numbness and tingling of the fingers and toes, palms and soles, and other parts of the lower arms and legs. There are hyperæsthesia, tenderness, and pain, more marked in the legs, including cramps in the calves. Vaso-motor disturbances are manifested by pallor of the fingers. These may in mild degree precede the onset as premonitory symptoms for weeks and for months, especially in the alcoholic form. The motor symptoms, seldom absent, soon follow the sensory phenomena above noted. They include palsy or incoördination or both in upper and lower limbs, but with this characteristic that it is symmetrical and involves their distal extremities, as the feet and hands, the former more frequently.

Motor symptoms may exist in the feet and sensory in the hands, the latter preceding. The loss of power is generally accompanied by some loss of coördinating power manifested by difficulty in balancing while standing or executing finer movements with the fingers. Indeed, these may be the first symptoms, and may lead on their investigation to the knowledge of some defect in extending the wrist and fingers, or raising the toes or foot from the ground in walking. Thus the muscles first involved are those supplied by the peroneal branch of the external popliteal nerve in the lower and the radial branch of the musculo-spinal in the upper extremity. Tremor is a marked feature in some alcoholic cases and may precede some loss of power. With weakness in the legs comes loss of knee-jerk and ankle-jerk, quite constant, but not invariable. The muscles above the knee are less frequently affected, and still less frequently those which move the hip-joint. The paralysis of extensors of the feet gives rise to a peculiar and distinctive walk known as the *steppage* gait. In it the foot is thrust forcibly forward with the toe lifted high in the air to avoid tripping upon it, the heel is brought down first, and then the entire foot. In general, the effect is that of a person stepping over obstructions. It is also called the pseudo-tabetic gait,

resembling somewhat the gait of locomotor ataxia yet still different. As contrasted with the tendon reflexes, the reflex action from the skin may be increased, especially where there is hyperæsthesia, even where there is considerable motor paralysis, the movement being caused by the muscles which escape. In severe cases, on the other hand, where there is much loss of sensation and motion, the skin reflex is absent; exceptionally it may be absent when sensation is perfect. Myotatic irritability is always lost.

Very characteristic is the tenderness of the muscles themselves, developed as they become weaker, and elicited by grasping them, the least pressure often causing the patient to cry out with pain. This is regarded as evidence that all the nerves of the muscles suffer, the sensory as well as the motor. The nerve trunks are also tender, although this tenderness is less marked than in simple neuritis because the contrast with the hyperæsthesia of the surrounding skin is less conspicuous. In the arms it is the extensors of the wrist which are first affected, and these symmetrically, illustrated by one of the best recognized toxic forms of neuritis, lead palsy. As in it, the extensor of the metacarpal bone of the thumb and supinator longus may escape. After the extensors are involved the flexors of the wrists and fingers, then the interosseous muscles, and, finally, the thenar and hypothenar muscles, always to a less degree than the flexors. The muscles above the elbow suffer less and those of the elbow least. Rarely the fibres of the pneumogastric are involved, causing frequent pulse and paralysis of the vocal cords, cardiac failure, and death; still more rarely the diaphragm and muscles of the thorax and abdomen. The facial and motor oculi nerves are possible seats. The sphincters are also rarely affected as the result of associated spinal involvement.

The muscles exhibit the reaction of degeneration, faradic irritability being lost and that to galvanism being increased, but not always altered in quality. In the nerves irritability to both currents diminishes and ultimately disappears, although in the very first stage there may be increased irritability, as described under the reaction of degeneration. In severe cases the total loss of excitability may be immediate on account of a corresponding destruction of muscular substance instead of the production of the intermediate state of increased excitability.

Wasting of the muscles is sooner or later inevitable, although it may be obscured by a temporary œdema or a condition of fatty infiltration, in which the fat accumulates between the wasting fasciculi, keeping up for a time the bulk. The less affected muscles are apt to undergo shortening and contracture because of maintaining so long a fixed position, either from being given over to gravitation or as a result of an effort to relieve pain. They occur most frequently in the lower extremity, contributing to intensify the "foot drop" at the ankle, and more rarely to produce flexure at the knee-joint and to a less degree even at the hip, both of the latter being the result of posture.

The sensory and motor phenomena are commonly associated *pari passu*, the latter extending from the hands and feet up the outside of the arm and leg. Very rarely either set of symptoms may occur alone.

Ataxic phenomena are usually associated with the sensory and motor symptoms. The ataxia is more marked in the lower extremities, and is believed to depend upon the afferent muscle-nerve involvement, since these sensory muscle-nerves are supposed to have most to do with coördination. Because of the associated absence of the knee-jerk, the term pseudo-tabes has been applied to the ataxic stage, the phenomena of which, however, always fall short of those of true tabes. It may be said, too, of the ataxic form that the sensory disturbances are less severe than in other typical cases.

Trophic changes may occur in prolonged cases, including mainly glossy skin, arthritic adhesions, and thickening; and also vaso-motor derangement, shown by œdema, especially about the ankles and back of the foot.

Mental symptoms are found more particularly in connection with the alcoholic form of neuritis. Besides irritability and general ill temper, more active symptoms are at times present. A childish jocularity in women, hysteria, and skilful duplicity in obtaining alcohol are characteristic. The phenomena may be those of delirium tremens or simple hallucination with extravagant ideas. Especially peculiar is the condition described by Wilks, in which there is a loss of appreciation of time and place, the patient describing with minute detail impossible journeys recently taken and persons whom he imagines he has seen. Convulsions and optic neuritis are rarely present, the probable result of meningeal inflammation. A simple mild delirium may occur in toxæmic cases from the action of the poison on the brain cells. Mental symptoms are not usually present in multiple neuritis from other causes.

The number and variety of the symptoms vary greatly in different forms, being most widespread in those due to alcoholism, to cold, or to combined causes, and limited in the cases due to metallic poisons, as lead. The more acute the case the more widespread are the symptoms.

Complications.—These are the other diseases to which alcoholics are subject,—cirrhotic and fatty livers, gastric catarrh, diseased kidneys and their consequences. The chief one in the toxic form is gout the result of mineral poisons, almost exclusively lead. Tubercular consumption is common, and pneumonia, invading especially the middle portion of the lung and sometimes bilateral, is a frequent cause of death.

Diagnosis.—The diagnosis of alcoholic cases is usually easy from the history, although sometimes skilful deception deprives the physician of this assistance. The distinctive features of the disease are the symmetrical localization of the sensory and motor symptoms first and mainly in the extremities, and the tenderness of the skin, nerve trunks, and muscles. There are, however, great differences in different cases even dependent on the same cause, some being very acute and general and even rapidly fatal, others slow and limited to groups of muscles; some mainly motor, others sensory and ataxic (pseudo-tabes).

Differential Diagnosis.—The possible sources of confusion are rheumatism, acute and chronic; neuralgia, tabes dorsalis, polio-myelitis, acute and subacute; pachymeningitis damaging the nerve roots, acute ascending paralysis, hysterical palsy.

In rheumatism there is not the tingling characteristic of neuritis, and although a tender nerve passing in the neighborhood of a joint, especially apt to be aggravated in motion, is liable to be mistaken for joint-pain, careful examination will elicit its true nature. Neuritis differs from neuralgia in the bilateral symmetry of the pain, the persistence of tenderness and hyperæsthesia as contrasted with spontaneous pain.

The ataxic form of the disease, especially the form called neuro-tabes, sometimes resembles tabes dorsalis very closely. In neuro-tabes the lesion consists only in the nerve degeneration, while the spinal cord is free, its claim to the title being the fact that there is no actual loss of power in neuro-tabes or in true tabes. The association of absolute paralysis or distinct weakness of extensors with incoördination would mean neuritis. With this exception the diagnosis from tabes may generally be easily made. The *steppage gait* is really not that of tabes, and the defective coördination is not very striking. There is no loss of power in tabes, and the "lightning pains" of this disease are seldom found in neuritis, nor is waist constriction nor pupillary symptoms, while the "muscular tenderness" is not found in tabes, nor is the foot drop. The extreme hyperæsthesia, such a distinctive symptom of neuritis, is less valuable in diagnosis, because it is often absent in the ataxic form. Girdle pains or constriction, paralysis of the sphincters, and unusual distribution, such as complete paralysis of all parts of the legs or upper parts, all point to cord involvement, even in alcoholic cases.

Polio-myelitis, inflammation, acute or subacute, of the grey matter of the cord, resembles the rheumatic and toxæmic forms of neuritis, which have, like the former disease, a febrile onset, initial rheumatic pains, muscular wasting with the reaction of degeneration and tendency to spontaneous recovery. But again we contrast the symmetrical distribution of the palsy of neuritis with the random distribution of polio-myelitis. Increased "myotatic irritability," increased knee and foot jerks, and increased muscular spasm generally point to cord disease.

In pachymeningitis damaging the nerve roots and producing paralysis, wasting, and anæsthesia, the legs rarely suffer; while the upper parts of the arms and trunk are invaded by anæsthesia, there is no tenderness of the nerve trunks. Acute ascending paralysis (Landry's disease) resembles the most rapid form of multiple neuritis in some of its symptoms, but these ascend the trunk from the legs to the arms and do not begin in the hands and feet at the same time or successively, nor do they affect the trunk last, as in neuritis. There is, moreover, no anæsthesia in ascending paralysis.

Some assistance in diagnosis may be had from the etiological standpoint, the history of exposure to metallic poisoning, to alcoholic excesses or infectious diseases, or the presence of diabetes being suggestive.

Prognosis.—A very large number of cases of multiple neuritis get well, though slowly, especially if the cause can be discovered and removed. Especially is this true of the alcoholic cases, although improvement does not always begin immediately on withdrawing the cause, indeed, the disease may even progress for a time. Hence the prognosis should not be too sanguine. The acute and widespread cases are the most dangerous to life,

and in such the prognosis should always be guarded. The involvement of the muscles of respiration including the diaphragm is most to be feared. Pain in the trunk muscles is a grave symptom if the motor power of the limbs has declined much. Paralysis of the diaphragm may be insidious and unnoted until that of the intercostals is added, when there may be accumulation of mucus, bronchitis, and death by suffocation. Involvement of the cardiac nerves is also serious and is manifested by frequency of pulse. Superadded involvement of the spinal cord increases the danger. At best, months are required for recovery and even years may be necessary. Involvement of spinal cord precludes total recovery. The return of faradic irritability in nerve and muscle is favorable. To sum up with Gowers, "The prognosis is better in the sensory than in the motor form, better when the arm escapes than when all the limbs are involved, better in cases of chronic than acute onset, and better if a case of apparently acute onset is really such than if it succeeds slight symptoms of longer duration."

Treatment.—The removal of the cause if possible is a primary step in treatment. Along with this, rest is most important, and the rest should be complete—in bed. There should be no compromise with alcohol, although in cases of great debility gradual withdrawal may be justified. The patient should, on the other hand, be fed on the most nutritious food. Local anodyne applications may be resorted to to relieve the pain and may be varied according to effect. Dry heat, moist heat, applications of lead-water and laudanum, and ointments of aconite and veratria are some of those which may be employed. Wrapping in cotton or wool is sometimes beneficial. Warm baths are soothing; sometimes very hot ones give relief.

Postures assumed because of the relief they give to pain should not be too long permitted lest deformity result by contractions and adhesion difficult or impossible to overcome. Dropping of the feet should be prevented by splints or support by sand-bags. The same is true of flexion at the knee and hip.

As to drugs, they are of little use; the salicylates, phenacetin, antifebrin, and antipyrin may be useful in mild cases and should be tried in doses of five to 15 grains (0.3 to one gm.). They are more particularly useful in cases due to cold. Extreme pain may demand the cautious use of morphine hypodermically in doses of $\frac{1}{6}$ to $\frac{1}{3}$ grain (0.011 to 0.022 gm.) combined with $\frac{1}{150}$ grain (0.00044 gm.) of atropine, which modifies and improves the action of morphine most happily. For the mental symptoms the hydrobromate of hyoscine in doses of $\frac{1}{200}$ to $\frac{1}{100}$ grain (0.00033 to 0.00066 gm.) hypodermically, or hyoscine in doses of $\frac{1}{400}$ to $\frac{1}{150}$ grain (0.00016 to 0.0044 gm.) may be tried. Mercurials, so highly approved of in simple neuritis, are useless here. The iodides are sometimes beneficial in chronic cases.

Roborant medicines, such as iron and cod-liver oil, are indicated to build up the patient, who is generally broken down. Electricity and massage are very useful after convalescence has set in.

Endemic Neuritis.

Definition.—This term is applied to certain forms of multiple neuritis, supposed to be due to vegetable organisms, limited or endemic to certain localities. Three separate varieties have been recognized. Probably more exist. The three referred to are malarial neuritis, beri-beri, and leprous neuritis.

(1) Malarial Neuritis.—This corresponds in its clinical features with the simpler forms of multiple neuritis, and requires no detailed description. Its malarial nature is based on its prevalence in malarial districts and its curability by quinine. While it is believed to be caused by the plasmodium of malaria, I am not aware that this organism has as yet been discovered in the blood of patients suffering with it.

(2) Beri-beri, the Kak-ké of Japan.—Beri-beri is a disease prevalent in Japan, the Eastern Archipelago, India, New Zealand, Ceylon, the South Pacific Islands, and the coast of Brazil. It is especially prevalent in the Dutch East Indies among soldiers and in prisons, and has been especially investigated under the Netherlands Government. In this country J. J. Putman has described a similar disorder among New England fishermen who frequent the Grand Banks of Newfoundland. It is, also, not uncommon among Norwegian sailors. Seguin, of New York, has described cases originating in the West Indies and coming to this country.

Etiology.—Sheube and Baelz first determined its true nature, but our knowledge has been greatly increased of late by the studies of Pekelharing and Winkler.* It is believed to be due to a special organism of which rods and cocci have been described, and of which cultures have been made, and peripheral neuritis of the same distribution as beri-beri produced by their inoculation. Repeated inoculations having, however, been required to produce it, it has been concluded that repeated exposures are necessary before infection results. It is transmissible from individual to individual. The disease is also acquired by residence in certain houses, and patients recover after removal to a district which is free, relapsing on returning. It has been thought that a nitrogenous and especially an exclusive fish diet predisposes to the disease, and again a rice diet. Roll † has recently shown that in all probability the disease is transmitted through drinking water, since he traced two epidemics on board ship to this cause. In both cases the sailors were free as long as they had a supply of European water; but in one instance, after laying in fresh water at Batavia, and another in Mauritius, in both of which places the disease prevailed endemically, it appeared among the crew at the end of five weeks.

Symptoms.—There are several types of cases. Among the earliest symptoms is a *change in the electrical excitability of the peroneal nerves*

* Pekelharing and Winkler, *Centralblatt f. Nervenkrankheiten*, 1889, and *Deut. Med. Wochenschr.*, 1888, No. 30.

† *Norsk Magazin for Lægevidenskaben*, Nov., 1895, and May, 1896.

and the flexors of the ankles, consisting in a slight degree of reaction of degeneration, quantitative and often qualitative, this even before there are any subjective symptoms. Sometimes, indeed, the disease goes no farther. Generally, however, the subjective symptoms begin as a *sense of heaviness of the legs,* a *tendency to tire easily, perverted sensation, diminished tactile sense in the lower legs,* and *irritability of the heart.* In an acute pernicious form the nervous phenomena are less marked. There are *fever, anæmia,* and *general anasarca.* The *œdema* is quite constant, beginning in the legs. The urine is scanty, but not otherwise altered, containing no albumin. A critical increase in the quantity of urine indicates an improvement. In the second group the neuritic symptoms are more marked, there being *numbness, anæsthesia, loss of tendon reflexes, muscular atrophy,* and *anasarca.* In the third group the *atrophy* and *paralysis* are most conspicuous, and the clinical picture is that of a rapidly progressing multiple neuritis with sensory and motor symptoms. The mortality varies from three to four to 60 and even 70 per cent. The diaphragm and larynx may become paralyzed, and the cardiac branches of the vagus involved, producing cardiac failure and death.

Treatment.—The treatment calls for the removal of the cause by disinfection or removal of the patient from the infected house or district and the withdrawal of suspected food or drinking water.

The symptoms are treated as in other forms of neuritis. In consequence of the tendency to cardiac weakness, heart tonics may be needed, such as digitalis, strychnine, strophanthus, and caffein.

(3) Leprous Neuritis.—Similar to beri-beri is leprous neuritis, already considered as to its etiology and symptomatology and treatment. (See Infectious Diseases.) It differs from beri-beri in being transmitted from parent to offspring and in its extreme slowness of development after exposure, as much as ten years intervening. It differs also from beri-beri in that the neuritis is not an essential part of the disease. The neuritis is a symptom of the so-called "anæsthetic leprosy." Leprous neuritis differs further from the usual forms of multiple neuritis in not being perfectly symmetrical and in being a peri- and interstitial neuritis, instead of parenchymatous. The bacillus is also found in the tissue, causing by its presence the inflammation, while in beri-beri the virus circulates in the blood. Hence the irregular distribution of the neuritis in leprous neuritis, resembling in this respect the more isolated neuritis of syphilis.

Symptoms.—The special symptoms are muscular wasting and anæsthesia, more marked toward the extremities of the limbs, but not confined to them, being met, as in the face, involving the 5th and 7th pairs of nerves. Sometimes tenderness and pain are present; the latter is, however, not severe. There may be tingling, also anæsthesia, and diminished electrical excitability, with reaction of degeneration. The irregular areas of anæsthesia are generally associated with irregular patches of pigmentation and pallor.

The **diagnosis, prognosis,** and **treatment** are those of leprosy.

NEUROMATA.

Definitions and Morbid Anatomy.—This term should be restricted to tumors composed purely of nervous tissue, which are to be distinguished from tumors whose composition is fibrous in character, often seated on nerves, and also known as false neuromata. Another form of false neuroma is a variety of the small subcutaneous painful tumors—*tubercula dolorosa*—found on nerves of the skin in the neighborhood of the joints, on the face and the breast. This variety has been shown by Hoggan to be composed of an adenomatous growth of sweat glands. Myxomata, sarcomata, and even carcinomata are found in connection with nerves. The latter are commonly the result of extension by contiguity, infiltrating the connective tissue between the fibres. The nervous tissue represented in neuroma is usually the fibrous, but very rarely ganglionic nerve cells are found, and in such event the tumor may be regarded as either dislocated brain substance or a glioma whose cells closely resemble true nerve cells. The nervous tissue may be of the medullated or non-medullated variety, myelinic or non-myelinic. Connective tissue varying in quantity is associated with both, producing various degrees of hardness, which is most striking in the multiple neuroma.

An interesting variety is the plexiform neuroma, nodular and tortuous in appearance to the naked eye, whose internal structure is composed also of interlacing nodular and tortuous nervous cords made up of connective tissue and nerve fibres. It is most frequently found in connection with the 5th pair of nerves in the orbit, upper eyelid, or on the temporal bone, but is met with also in connection with any of the spinal and even sympathetic nerves. It grows slowly, and probably begins in fœtal life.

Neuromata are usually small, but may be three or four inches (7.5 or 10 cm.) in diameter and even larger. They are usually found in connection with nerve trunks or at their end, are often multiple, and their number is sometimes enormous, counted even by thousands.

Etiology.—Nerve tumors which are not congenital are probably always traumatic. More than one member of a family has been found affected. Their growth seems stimulated by perversion in the healing process, since they are found on the ends of nerves in cicatrices after amputation.

Symptoms.—Neuromata may be totally without symptoms. At other times they are very painful, the *pain* being aggravated by pressure. There may be *numbness* and *formication* and even *loss of sensation* on the one hand, *muscular twitching and paralysis* on the other, the latter especially when the tumor is in the course of the nerve.

Neuroma of the cauda equina may cause paraplegia. Reflex spasm in adjacent or distant muscles, and even epileptiform convulsions, are occasionally present. Neuroma may give rise to visible swelling, or it may be beneath the surface out of sight and touch.

Diagnosis.—Except in the case of plexiform neuroma, which has a characteristic form described, the exact diagnosis can for the most part

only be made by microscopic examination after removal, since all the symptoms occasioned by true neuroma may be caused by pressure on nerves by any form of morbid growth. The multiple neuromata are usually true neuromata.

Prognosis.—Neuromata rarely cause death. The extreme pain which is so characteristic may in time wear a patient out, but the course of the disease is always long.

Treatment.—Excision is the proper treatment for neuroma if it can be reached and the symptoms demand active treatment. Often they do not. If syphilitic origin be suspected, syphilitic treatment should be adopted. In operations involving section of nerve trunk, the possibility of loss of function is to be remembered.

Local anodyne applications may be used to palliate in mild cases, but they are useless in severe ones. Cocaine in doses of $\frac{1}{6}$ to $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.011 to 0.0165 to 0.033 gm.) may be injected hypodermically, but morphine should not be used, as the conditions are especially favorable to the production of morphinism.

AFFECTIONS OF THE SPINAL CORD.

Anatomical.—The spinal cord, covered by its membranes, the dura and pia-arachnoid, hangs loosely in the spinal canal from the atlas to the 2d lumbar vertebra. It is, therefore, much shorter than the spinal canal itself. The remainder of the canal is occupied by the cauda equina. The nerve roots arise each pair above the foramen of exit, and descend to the latter within the canal. The part of the cord whence each pair arises is known as the segment of that particular pair of nerves. In the cervical region, the end of each cervical spine is nearly opposite the roots of the nerve below. The vertebra prominens, or 7th cervical, is opposite the 1st dorsal roots, and from the 3d to the 10th dorsal the spines correspond to the 2d root below. The 11th spine corresponds to the 1st and 2d lumbar, the 12th to the 3d, 4th, and 5th. The 1st lumbar to the 1st, 2d, and 3d sacral nerves, while the end of the cord is opposite the upper part of the 2d lumbar.

In transverse section the cord is easily seen by the naked eye to be made up of central grey matter and external white substance. The former is composed mainly of cells, the latter of fibres. The grey matter, roughly comparable to two crescents placed back to back, reaches the surface only by its posterior horns at the two points whence arise the posterior roots of the spinal nerves. The broad, blunt anterior cornua do not reach the surface, but the white fibres of the anterior roots are seen perforating the white matter to enter the grey. The cord is separated into halves by the anterior median fissure, which is broad and shallow, and the posterior median septum, deep and narrow, composed of connective tissue.

At the bottom of the anterior median fissure is the transverse commissure of white matter penetrated by the central spinal canal. A short

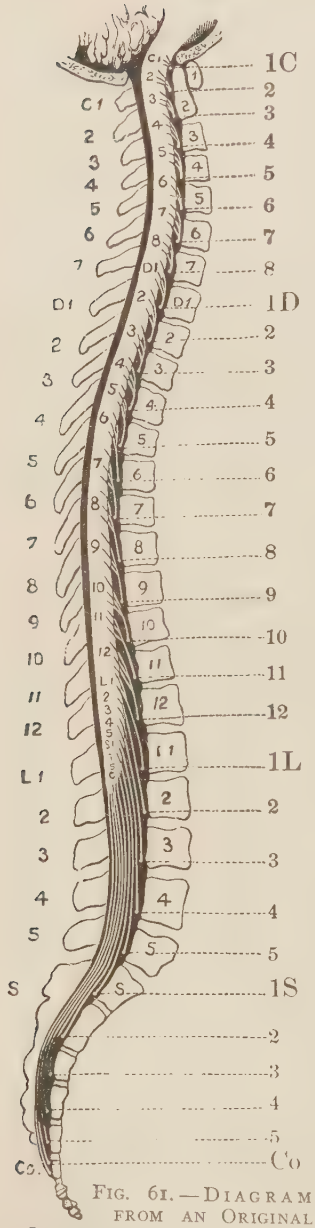


FIG. 61.—DIAGRAM FROM AN ORIGINAL INVESTIGATION BY W. R. GOWERS, SHOWING RELATION OF VERTEBRAL SPINES TO THEIR BODIES AND TO THE NERVE ROOTS.

The ends of the vertebral spines are opposite the middle of their own bodies only in the lumbar region. They correspond to the lower edge of their own bodies in the cervical and last two dorsal, and to the upper part of the body below in the rest of the dorsal region. (See also text.)

distance to the outside of the posterior median fissure is another less distinct septum, the posterior intermediate septum, which bounds the posterior median column, or column of Goll. Outside of this again, bounded by the posterior root, is the posterior external column, or column of Burdach. The part of the horns of grey matter at which enter the posterior roots is called the posterior-root zone, because many fibres of the posterior root pass through it. The remainder of the white substance of half the cord has no natural divisions, and is, therefore, known as the antero-lateral column, but artificially it is divided by a line coinciding with the outermost of the anterior nerve roots, and thus is made an anterior and middle column, which, with the posterior column, makes three columns for each half cord. The white matter is composed of the usual medullated nerve fibres unprovided with neurilemma and of neuroglia cementing the fibres. The further divisions of the cord in transverse section are clearly indicated by the appended drawing of a transverse section of the cord in the cervical region with description. Of these parts, the anterior pyramidal tract, the lateral pyramidal tract, and anterior cornua may be characterized, generally speaking, as motor, while the posterior columns, the direct cerebellar tract, the antero-lateral ascending tract of Gowers, antero-lateral ground bundles, and posterior cornua, as sensory.

The white matter gradually diminishes as the cord is descended. The grey matter also varies in extent and shape at different levels, which will be appreciated by the examination of Figure 63, which explains itself. In addition should be mentioned Clark's column, a group of nerve cells in the inner part of the neck of the posterior horn, from the 8th dorsal to the 2d lumbar, also known as the lateral fascicular column. In the upper dorsal and lower cervical regions a group of cells projects outward from the grey matter into the lateral column, called by Lockhart Clark the intermedio-lateral process, but well named also as the lateral horn. The lateral or crossed pyramidal tracts, representing about ¾ of the motor fibres, passing

down from the cortex decussate in the anterior pyramids and pass over into the lateral columns. The remaining fibres, which do not decussate, pass down their own side of the cord in the inner part of the antero-lateral column, constituting the anterior or direct pyramidal tract, also known as Türck's column. At every level of the spinal cord axis cylinders leave the crossed pyramidal tract to enter the anterior horns and end about the cell bodies of the lower motor neurons. The tract extends down to the end of the cord, but becomes smaller and smaller. The fibres of the direct pyramidal tract also cross at different levels in the anterior white commissure and end about cells in the anterior horns on the opposite side of the cord. If primarily small, it may not extend beyond the middle of the cervical enlargement. If originally large, it may be traced as far as the lumbar enlargement. Through the greater part

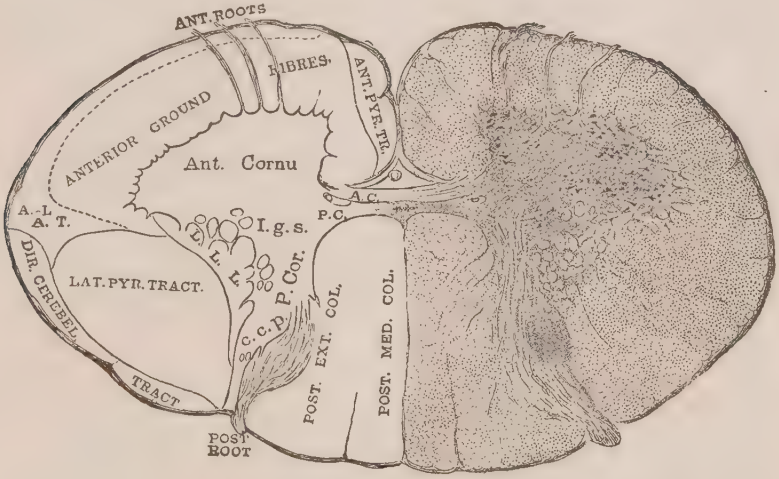


FIG. 62.—SECTION OF SPINAL CORD IN THE CERVICAL REGION.

A. C. Anterior commissure. P. C. Posterior commissure. I. g. s. Intermediary grey substance, the grey matter between the two horns. P. Cor. Posterior cornu. c. c. p. Caput cornu posterioris. L. L. L. Lateral limiting layer. A.-L. A. T. Antero-lateral ascending tract of Gowers, which extends along the periphery of the cord.—(*After Gowers.*)

of the cervical and dorsal regions the lateral pyramidal tract is separated from the surface by a narrow layer of fibres, the direct cerebellar tract, which in the upper cervical region lies further forward, so that the pyramidal tract comes up to the surface close to the posterior horn.

The axis cylinder processes of the anterior roots start from the nerve cells in the segments where these roots arise, but the relation of the axis cylinders of the posterior roots after they enter the cord is not so simple. It has already been said (p. 795) that the single process which leaves the cell on the posterior roots of the spinal nerves divides in a T-shaped manner, one limb running in the spinal nerve to the periphery of the body, the other into the central nervous system as an axis cylinder process. After entering the cord, each axis cylinder process divides into an ascending and descending limb, which run in the posterior columns.

The descending branch runs a short distance and ends in the grey matter of the same side of the cord. The ascending branch may end in the grey matter soon after entering it, or it may run in the posterior columns to the medulla, ending in the nuclei situated in the posterior columns of the medulla, remaining up to this point on the same side of the middle line.

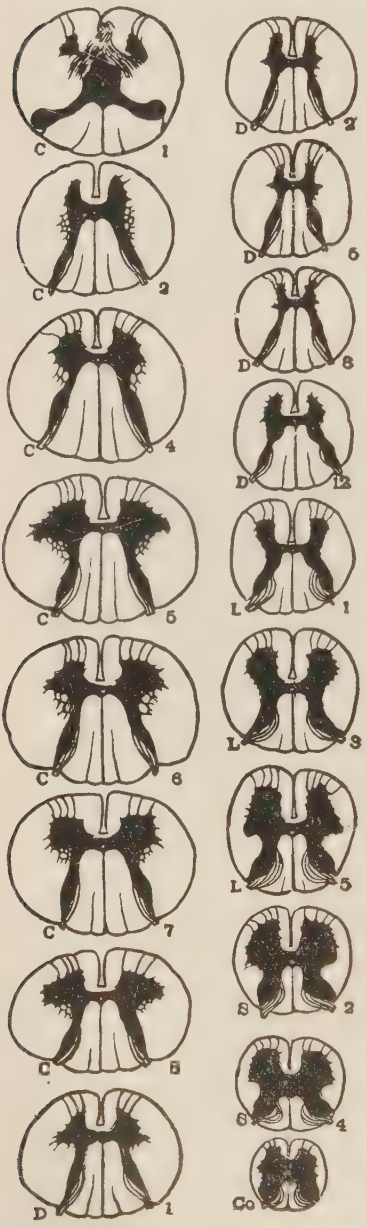


FIG. 63.—DIAGRAM SHOWING RELATIVE SIZE AND SHAPE OF THE CORD AND GREY MATTER AT DIFFERENT LEVELS.—(*After Gowers.*)

Cells about which the axis cylinder processes and their collaterals of the lower sensitive neuron end are of several kinds. In the first place, they include cell bodies of the lower motor neurons, forming paths for reflexes. They also end about cells whose axis cylinder processes cross the middle line and run to the opposite side of the brain. In the spinal cord these cells are found in the different parts of the grey matter, and their axis cylinder processes run in the opposite anterolateral ascending tract. From the nuclei of the posterior columns of the medulla the axis cylinder processes, after crossing, run toward the brain, enter the fillet, into which enter also the fibres of the lateral ascending tract containing the crossed fibres of the upper sensory neuron. The exact termination of sensory processes in the cerebral hemispheres is not known. The position of the tract in the crus and internal capsule is posterior. The lower sensory neurons also have endings in the cells or about the cells in Clark's column, from which cells the axis cylinders run in the direct cerebellar tract of the same side; also about cells whose axis cylinder processes run but a short distance in the cord to end in the grey matter on a different level. Thus the possible paths of sensory conduction are probably many and not altogether determined, whence disturbances of sensation do not give us as much help in topical diagnosis as those of motion. It may, however, be said in summary that cutaneous sensory impressions in man are conducted toward the brain on the

opposite side of the cord, the crossing taking place partly in the central grey matter soon after the path enters the cord and partly after the processes leave the nuclei in the posterior columns of the medulla oblongata. The muscular sense only is conducted on the same side of the cord in that part of the posterior column known as the column of Goll to cross in the medulla.

Spinal Cord Localization.—It has already been said that the areas of distribution of spinal nerves, sensory and motor, are not sharply defined for each nerve as it emanates from the spinal cord, but that the regions thus supplied overlap. At the same time physiologists and clinicians have been able to map out with approximate accuracy the motor and sensory areas corresponding to the distribution of each pair of nerves emanating from different segments of the cord. Among those who have especially devoted themselves to this subject are M. Allen Starr, Charles K. Mills, and Charles L. Dana in America, and William Thorburn and Henry Head in England.

The results of various observers differ in detail but agree in essentials. The appended table is that originally devised by Starr, further modified by C. L. Dana and C. K. Mills.

It must not be forgotten that these areas of distribution correspond to a nerve constituted as it is when it emanates from a corresponding segment of the cord, and not as it is constituted immediately before it begins to spread.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.

Segment.	Muscles.	Reflex and Centres.	Sensation.
I C.	Rectus laterales. Rectus capitis. Anticus and posticus. Sterno-hyoid. Sterno-thyroid.		
II and III C.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	*Hypochondrium* (*?*). Sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex and neck. (Occipitalis major, occipitalis minor, auricularis magnus, superficialis colli, and supraclavicular.)
IV C.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	*Pupillary* (4th cervical to 2d dorsal). Dilatation of the pupil produced by irritation of neck.	Neck. Shoulder, anterior surface. Outer arm. (Supraclavicular, circumflex, external musculo-cutaneous, cutaneous.)
V C.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Deep muscles of shoulder-blade. Rhomboid. Teres minor. Pectoralis (clavicular part) Serratus magnus.	*Scapular* (5th cervical to 1st dorsal). Irritation of skin over the scapula produces contraction of scapular muscles. *Supinator longus.* Tapping the tendon of the supinator longus produces flexion of forearm.	Back of shoulder and arm. Outer side of arm and forearm to the wrist. (Supraclavicular, circumflex, external cutaneous, internal cutaneous, posterior spinal branches.)

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.—(*Continued.*)

Segment.	Muscles.	Reflex and Centres.	Sensation.
VI C.	Deltoid. Biceps. Brachialis anticus. Subscapular. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators. Rhomboid. Latissimus dorsi.	*Triceps* (5th to 6th cervical). Tapping elbow tendon produces extension of forearm. *Posterior wrist* (6th to 8th cervical). Tapping tendons causes extension of hand.	Outer side and front of forearm. Back of hand, radial distribution. (Chiefly external cutaneous, internal cutaneous, radial.)
VII C.	Triceps (long head). Extensors of wrist and fingers. Pronators of wrist. Flexors of wrist. Subscapular. Pectoralis (costal part). Serratus magnus. Latissimus dorsi. Teres major.	*Anterior wrist* (7th to 8th cervical). Tapping anterior tendons causes flexion of wrist. *Palmar* (7th cervical to 1st dorsal). Stroking palm causes closure of fingers.	Radial distribution in the hand. Median distribution in the palm, thumb, index, and one-half middle finger. (External cutaneous, internal cutaneous, radial, median, posterior spinal branches.)
VIII C.	Triceps (long head). Flexors of wrist and fingers. Intrinsic hand muscles.	. . . . .	Ulnar area of hand, back, and palm, inner border of forearm. (Internal cutaneous, ulnar.)
I D.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar muscles.	. . . . .	Chiefly inner side of forearm and arm to near the axilla. (Chiefly internal cutaneous and nerve of Wrisberg or lesser internal cutaneous.)
II D.	. . . . .	. . . . .	Inner side of arm near and in axilla. (Intercosto-humeral.)
II to XII D.	Muscles of back and abdomen. Erectores spinæ.	*Epigastric* (4th to 7th dorsal). Tickling mammary region causes retraction of the epigastrium. *Abdominal* (7th to 11th dorsal). Stroking side of abdomen causes retraction of belly. Vasomotor centres. Second dorsal to 2d lumbar.	Skin of chest and abdomen, in bands running around and downward, corresponding to spinal nerves. Upper gluteal region. (Intercostals and dorsal posterior nerves.)
I L.	None.	*Cremasteric* (1st to 3d lumbar). Stroking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum. (Ilio-hypogastric, ilio-inguinal.)
II L.	Vastus internus.	*Patellar.* Striking patellar tendon causes extension of leg.	Outer side and upper front of thigh. Lumbar region. (Genito-crural, external cutaneous.)

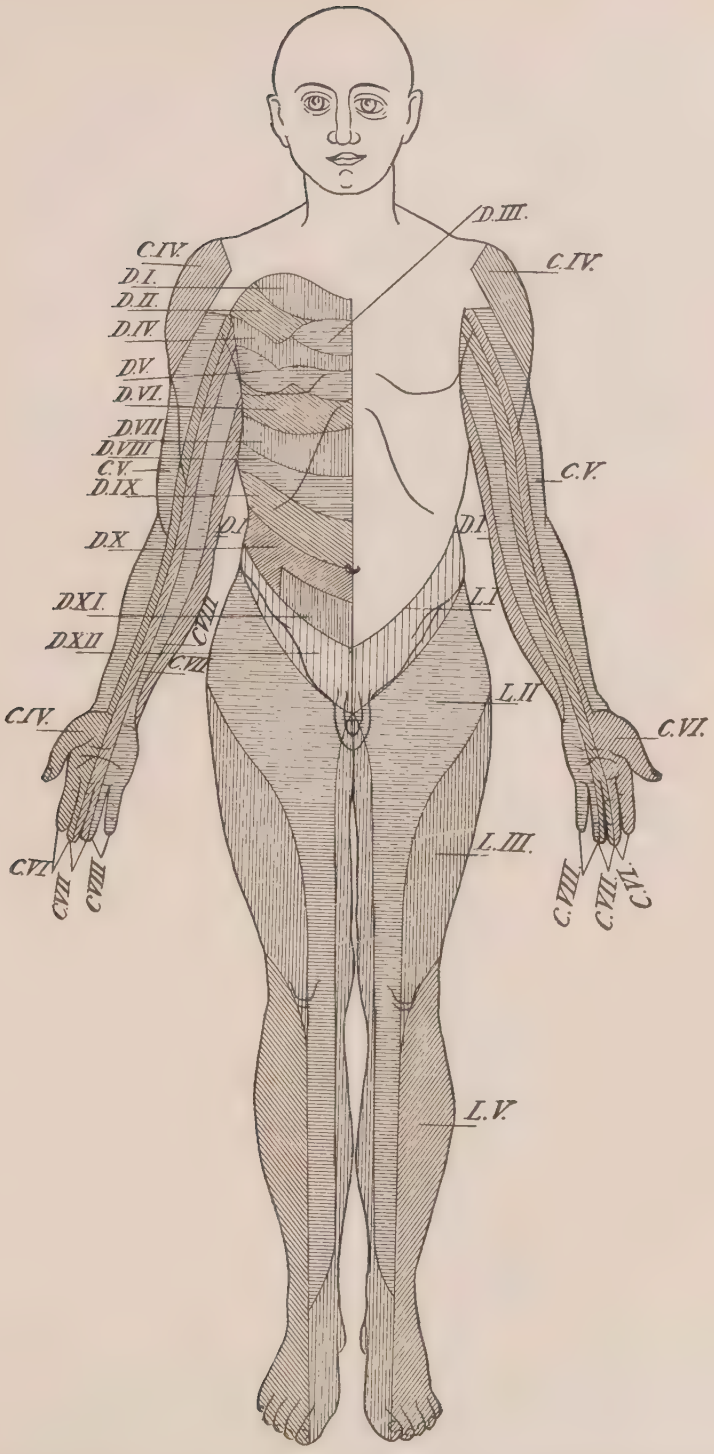


FIG. 64.—DIAGRAM OF SKIN AREAS CORRESPONDING TO DIFFERENT SPINAL SEGMENTS.—(*Chiefly after Starr. Trunk areas from head.*)

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.—(*Continued.*)

SEGMENT.	MUSCLES.	REFLEX AND CENTRES.	SENSATION.
III L.	Sartorius; adductors of thigh. Flexors of thigh.	. . . . .	Front and outer side of thigh. Inner side of leg and foot.
IV L.	Extensors of knee. Abductors of thigh.	*Gluteal* (4th to 5th lumbar). Stroking buttock causes dimpling in fold of buttock.	Inner side of thigh, leg, and foot. (Internal cutaneous, long saphenous, obturator.)
V L.	Outward rotators. Flexors of knee. Flexors of ankle. Peronei. Extensors of toes.	*Achilles tendon*. Over-extension causes rapid flexion of ankle, called ankle clonus.	Back of thigh and outer side of leg and ankle; sole; dorsum of foot. (External popliteal, external saphenous, musculo-cutaneous, plantar.)
I and II S.	Calf muscles. Glutei. Peronei. Extensors of ankle. Small muscles of foot.	*Plantar* (5th lumbar to 2d sacral). Tickling sole of foot causes flexion of toes and retraction of leg.	Back of buttock and thigh, side of leg and ankle; sole; dorsum of foot.
III to V S.	Perineal. Muscles of bladder, rectum, and external genitals.	Genital centre. Vesical centre. Anal centre.	Circum-anal region, anus, rectum, penis, urethra, vagina, perineum. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)

The preceding table includes only the distribution of spinal nerves.

The following table includes the localization of function of the nuclei residing in the pons and medulla, so far as these are concerned with motion :—

Nuclei.	*Muscles.*
III Cr.	Sphincter iris. Ciliary muscles. Levator palpebræ superioris. Rectus internus in convergence. Rectus superior. Rectus inferior.
IV Cr.	Obliquus inferior. Obliquus superior.
VI Cr.	(Upper facial group.) Rectus externus. Rectus internus of opposite side in lateral movements.
V Cr.	Associated movement of levator palpebræ. Muscles of the lower jaw.
VII Cr.	Facial muscles.
XII Cr.	Lower facial group. Muscles of tongue.
IX Cr. X Cr. XI Cr.	Muscles of pharynx. Muscles of œsophagus. Muscles of larynx.

The study of the sensory areas is facilitated by the use of diagrams in which the areas are mapped out and indicated by color or a shading which will permit one to separate them easily one from another like those annexed, in which, too, the areas corresponding to each spinal segment are indicated by suitable lettering.

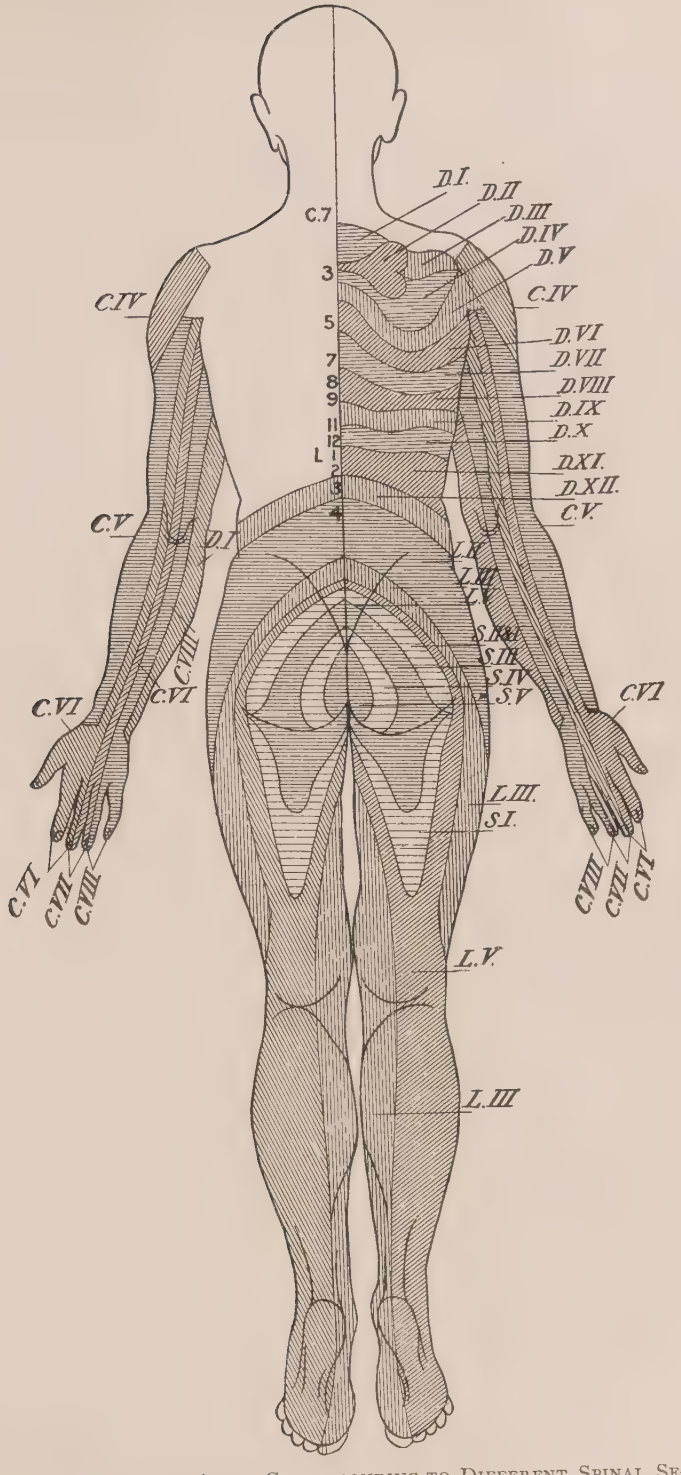


FIG. 65.—DIAGRAM OF SKIN AREAS CORRESPONDING TO DIFFERENT SPINAL SEGMENTS.—(*Chiefly after Starr. Trunk areas from head.*)

Interpreting by the data contained in tables and diagrams such motor or sensory derangements as may be present, one may deduce with more or less accuracy the seat in the cord of the lesions producing them. It has been mentioned that motor localization, being more exact, permits more correct inference than sensory derangements.

The union of both adds further facility. Results vary also according as a lesion involves only one-half or a complete section of the cord. Recalling the distribution of the two tracts as given on pages 832–3, it is evident that an injury involving the entire transverse section of the cord must produce, first, motor paralysis in all parts supplied with nerves emanating from segments below it. In less complete lesions correspondingly less degrees and extent of motor paralysis succeed. Such paralysis may extend to the bladder and rectum. After such section the muscles are flaccid and the deep reflexes absent. There is no atrophy, and the muscles respond normally to electricity. No satisfactory explanation has as yet been offered of the abolition of the reflexes. Second, there is impaired sensibility in the parts supplied by sensory nerves from corresponding segments below the lesion, but the sensory symptoms are not so decided. Moreover, the anæsthesia does not reach quite to the level of the lesion, because of the overlapping of sensory areas by nerves which come off from above the section. Thus, if the lesion be in the segment of the 6th dorsal the anæsthesia may extend only as high as the area supplied by the 7th. Moreover, above the anæsthetic area there is also at times an area of increased sensibility; the effect of the section being to increase the sensitiveness of the cord above it by increasing its vascularity due to section of vaso-motor nerves by the lesion. Finally, notwithstanding the less conspicuousness of the sensory effects, we may be enabled through them to ascertain the level of the disease because of the difficulty of demarking the upper limits of the motor paralysis. There may also be upper segment paralysis in those muscles whose centres are below the lesion by the involvement of the upper segment, so far as it contains fibres in the pyramidal tract. Muscular sense is lost. Reflex excitability, at first slightly impaired, is subsequently normal or slightly increased.

The phenomena are modified if the lesion of the cord be a *hemi*-lesion or half the cord. In such an event there is, first, motor paralysis in the half of the body on the same side, with upper segment paralysis on the same side. Cutaneous sensation is impaired, not on the same side, but on the *opposite* side, because of the fact already mentioned that one of the many routes of sensory impressions crosses the cord soon after it enters it. More than this, the sensibility on the same side, below the segment of lesion, so far from being diminished as to touch, pain, and temperature, may even be slightly increased, owing to the vaso-motor paralysis caused by the lesion, in consequence of which, too, there may be a slight rise of temperature on the same side. In the area corresponding exactly with the segment involved there is anæsthesia, while just above it on the paralyzed side, again, there is a small zone of hyperæsthesia. The muscular sense on the same side is impaired. Reflex excitability, at first diminished on the side of lesion, is subsequently increased.

On the opposite side muscular power is intact, sensibility is impaired, and the derangement may include the sense of pain, touch, and temperature, or any one or two; there is no elevation of temperature, the muscular sense is intact, and reflex action is normal.

All of these results, as described, may be produced by the experiments on the spinal cord originally suggested by Brown-Séquard, which included also section along the median line of the spinal cord, which affected sensation on both sides, impairing it, but left motion intact. Excepting the last, also, we meet them clinically in the so-called Brown-Séquard's paralysis, which will be considered later, but they are less definite and less constant because of the absence of the sharpness in the morbid lesion as contrasted with the experimenting knife. So far as completed, minute anatomical studies furnish results quite consistent with the derangements of motion produced by diseased states, and, to a less extent, also with the morbid phenomena of sensation as illustrated by disease. Thus anatomy, experiment, and pathology contribute to the same conclusion.

More circumscribed lesions produce more limited results. Thus a local lesion may produce paralysis only in a few groups of muscles. Destructive lesion of the anterior cornua in the lower segment produces lower segment paralysis, with secondary degeneration and muscular atrophy, the reaction of degeneration, diminished reflexes, and diminished muscular tension, while irritative lesions in this and other parts of the motor tract cause spastic conditions, including exaggerated tendon reflexes, all of which have been described.

It is also a matter of importance to know whether a lesion lies in a nerve or in the cord itself. Many times this is at once apparent. At others it is more difficult to settle. It has already been said that hemiplegias are almost invariably central in their origin, while paraplegias are spinal. It is chiefly with localized palsies that difficulties arise. Etiology aids us somewhat. Thus localized palsies succeeding localized exposure to cold are apt to be peripheral. Traumatic causes produce peripheral palsies. Some assistance is rendered if there be an associated anæsthesia. Thus if a part be anæsthetic and motorily palsied, and if the same nerve supplies sensory and motor fibres to the muscles, the lesion is in that nerve. If, on the other hand, the muscles are supplied by several nerves from a given segment of the cord, and the anæsthesia corresponds with the area of distribution of nerves from the same segment of the cord, the lesion is probably in the cord or the nerves at their origin from it.

Affections of the Membranes of the Cord.

As in the case of the brain, the dura mater and pia-arachnoid are separate seats of disease, chiefly inflammatory, not quite so well understood nor quite so definitely separated in their clinical features. As in the case of the brain, we call inflammation of the dura mater, pachymeningitis; of the pia mater, leptomeningitis.

SPINAL PACHYMENINGITIS.

The dura mater is separated by a loose connective tissue from the bony canal which surrounds it, and an inflammation may invade this outer or the inner layer, affording a pachymeningitis externa and interna.

External Pachymeningitis.—This is always secondary to disease of the vertebræ or similar morbid processes or aneurismal erosion. While an acute condition may thus supervene, it is much more commonly chronic. Perhaps its most frequent cause is tuberculosis of the spine, with its pathological cheesy product and its traumatic results—the spinal curvature known as Pott's disease. It may be confined to quite a limited area, corresponding to the primary seat of the disease, or it may extend over a large area of the cord, corresponding to six or eight vertebræ.

Internal Pachymeningitis.—This occurs in two forms, first as an inflammation of the internal layer of the dura, confined, primarily, at least, to the cervical part of the cord, first fully described by Charcot in 1871, and later by his pupil, Joffroy, under the name of "pachymeningitis cervicalis hypertrophica." The second is a pachymeningitis interna hemorrhagica, in every way identical with the same disease to be described in connection with the dura of the brain.

Symptoms.—To the subjective symptoms of inflammation itself are naturally added compression symptoms, which, in fact, overshadow the former. The former include *pain*, not merely at the seat of inflammation in the back, but also in the area of distribution of the spinal nerves, the roots of which are involved in the process. They do not differ in the two forms.

Cervical hypertrophic pachymeningitis, ascribed to exposure to cold and the abuse of alcohol, is a chronic process, consisting in an accumulation on the inner surface of the dura of concentric layers of a firm, fibrinous growth, covering either a small extent or a considerable portion of the cervical enlargement. The result is a compression of the cord and of the nerve roots which are involved in the growth, producing symptoms divisible into three stages :—

(*a*) The painful stage. In this there is pain in the region supplied by the nerves whose roots are thus compressed, viz., that of the arms, cervical region, and occiput,—pain at times of great severity. In addition are observed paræsthesia, numbness, and tingling, rarely herpes.

(*b*) The stage of paralysis. After two or three months the second period, or stage of paralysis, sets in,—an atrophic paralysis, in which there is weakness of the arms, resulting from pressure on the anterior nerve

roots. The wasting affects certain muscular groups, as the flexors of the hands, supplied by the ulnar and median nerves, while the distribution of the radial to the antagonistic extensors remains free. The result is the very striking claw-hand, or *main en griffe*. In extreme cases the atrophy of the arms and shoulders becomes very great. There may be anæsthesia of the skin at this stage.

(*c*) Stage of spastic paralysis in the lower extremities. If the compression of the cord continues we reach the third stage of the disease. The motor fibres to the lower extremities which pass through the cervical cord become involved, and the result is a spastic paralysis of the lower extremities—a paresis with increased reflexes, but without wasting of the muscles, because the trophic centres for the muscles of the lower extremities in the anterior cornua of the lumbar cord remain intact. In cases of long duration, however, the compression of the cervical cord may lead to anæsthesia of the lower extremities, paralysis of the bladder, and bedsores.

Hemorrhagic pachymeningitis, or hæmatoma, as stated, is identical in nature with that of the dura mater of the brain and usually occurs at the same time. It has generally been observed in the same class of persons, general paralytics and drunkards. It may occur at any part of the cord or it may be limited to the cervical region, producing the symptoms just described, but it is rarely recognized.

Diagnosis.—The disease is to be distinguished from amyotrophic lateral sclerosis, syringo-myelia, and tumors. From the first it can be differentiated by the presence of the characteristic severe pain in the neck and arms, by the absence of bulbar symptoms; from syringo-myelia, by the absence of the sensory changes peculiar to that disease, but from tumors in the same locality it is often distinguished with difficulty because the pressure symptoms in both are the same.

Prognosis.—Cases are related in which decided improvement has taken place, if not recovery.

Treatment.—There is no directly curative treatment. The symptoms are to be relieved by appropriate measures. Baths, iodide of potassium, counter-irritation, and electricity have been recommended. The first three are reasonable. Joffroy recommends the application of the hot iron to the neck. Paquelin's cautery should answer the purpose as well.

SPINAL LEPTOMENINGITIS.

Acute Leptomeningitis.

As a separate and distinct disease from epidemic cerebro-spinal meningitis, described under infectious diseases, acute leptomeningitis may occur :—

(*a*) As the result of tuberculosis, its most common cause.

(*b*) From localization of the poison of the infectious diseases.

(*c*) As the result of extension by contiguity; and

(*d*) Possibly as the result of exposure to cold. All of these are very rare.

Morbid Anatomy.—The pathological changes are similar to those of epidemic cerebro-spinal meningitis. Injection, accumulation of fluid in the pia-arachnoid space, either a sero-fibrinous or purulent exudate, and finally thickening of the membrane are all more or less present. On account of the position of the body the fluid exudate tends to gravitate downward or toward the posterior aspect. In rare instances the morbid process may even invade the cord itself, producing a meningo-myelitis.

Symptoms.—The symptoms are those of the disease with which the meningitis is associated, in addition to fever and such other symptoms as are the result of vascular derangement and mechanical interference. These have already been detailed under cerebro-spinal meningitis, including *pain in the back* of varying severity, *stiffness*, *sensitiveness of the spine*, symptoms of irritation of nerve trunks, *disturbances of sensation.* The reflexes may be increased.

Paralytic symptoms are a late and also a rare development. The reflexes are sometimes diminished or abolished on account of the involvement of nerve roots. The urinary and bowel functions are sometimes deranged.

Diagnosis.—The diagnosis of simple acute meningitis in association with the infectious diseases should not be too hastily made because of its simulation by these diseases. This is, however, less common with spinal meningitis than with cerebral. Here, as in cerebral meningitis, the etiological factor may help us out. While, on the other hand, given the disease, the special variety present cannot always be told. The tubercular form is easiest recognized because of possible preëxisting symptoms of the disease. Not so much should stiffness and pain in the back be regarded, as hyperæsthesia and pain in distant parts supplied by nerves from the seat of special spinal tenderness. Again, cases of spinal meningitis have been found on the autopsy table where no symptoms were recognized during life.

Prognosis.—This is generally unfavorable. Except in the cerebro-spinal form the recovery is scarcely possible, though its possibility must be admitted, especially where the disease is secondary to the infectious diseases.

Treatment.—This is mainly symptomatic, and the details are those given under the head of cerebro-spinal meningitis.

Chronic Leptomeningitis.

So rare is primary chronic meningitis that its existence as a separate disease may be doubted. It may, however, remain as a remnant of an acute inflammation, especially of epidemic meningitis. Its possibility as secondary to chronic disease of the cord may be admitted, such as locomotor ataxia, but it almost never gives rise to symptoms. It is regarded as a possible consequence of syphilis and alcoholism.

Morbid Anatomy.—This would be a thickening and opacity of the membrane, adhesions between the dura and arachnoid, localized or general, and possible blood pigmentation. Certain white cartilaginous plates

sometimes found on the posterior surface of the spinal arachnoid are not to be regarded as inflammatory.

Symptoms.—The symptoms would be those described in connection with the acute form, milder in degree and less definite. In fact, the diagnosis is rarely made. A long continued, otherwise unexplainable stiffness, such as could be produced in the trunk and extremities, would justify suspicion. The *reflexes* would be *increased.*

Treatment.—This is symptomatic.

HEMORRHAGE INTO THE SPINAL MEMBRANES.

Synonyms.—*Hæmatorrhachis; Meningeal Apoplexy.*

Hemorrhage may take place between the dura mater and its bony collar, extra-meningeal, or within the dura mater, intra-meningeal.

Extra-meningeal hemorrhage is usually the result of trauma, such as concussion or fracture of the spinal column, punctured or gunshot wound. The blood comes from the rich plexus of veins that surround the dura. A considerable amount of blood may be thus effused without compressing the cord, because of the free communication of the subdural space in the brain and cord. An aneurism may burst into the spinal canal with fatal consequences.

Intra-meningeal hemorrhage is more common, but is naturally more limited, as are the sources of the hemorrhage. Punctiform hemorrhages, such as occur in cerebro-spinal meningitis, are of no significance. Intra-meningeal hemorrhages occur sometimes in connection with the infectious diseases, and Osler has met with two such cases in malignant small-pox, while they have been found after death from convulsive diseases, such as epilepsy, tetanus, and strychnine poisoning. So, also, the same observer has found in ventricular apoplexy the blood pass down from the 4th ventricle into the meninges. Aneurism of the basilar or vertebral arteries is, however, the most frequent cause of this form of hemorrhage.

Symptoms.—These are those of pressure on the cord, and may be slight and scarcely recognizable or decided, with resulting paralysis and pain on the one hand or anæsthesia on the other. The symptoms are as sudden as is usually the event which causes them. Sometimes, however, the extravasation is slower and the symptoms correspondingly gradual in their appearance. The distinctive negative symptoms are the retention of consciousness and the absence of all cerebral symptoms.

The area of the paralysis and nervous symptoms depends on the seat of the hemorrhage. If in the lumbar region, the legs are alone involved, the reflexes may be absent, and the functions of bladder and rectum impaired. If in the dorsal, there may be complete paraplegia, while the reflexes are retained; there may be thoracic symptoms, such as girdle pains. Herpes may be present. If in the cervical region, arms and legs are paralyzed, and there may be pain or anæsthesia in the upper extremities and neck. Embarrassed breathing, stiffness of the muscles of the neck, and even pupil symptoms may be added when the hemorrhage is thus situated.

Diagnosis.—The diagnosis is based on the absence of brain symptoms in connection with the suddenness of the symptoms due to the disease and the history of possible cause.

Prognosis.—In certain cases, where the hemorrhage is small, contraction and absorption of the clot take place, and the symptoms may pass away. In others the hemorrhage is fulminating, and death follows early from involvement of the medulla in the pressure. In intermediate states there is intermediate improvement.

Treatment.—Conditions favoring the arrest of hemorrhage and absorption of blood should be secured. Absolute rest and the application of cold cover these. If symptoms remain permanent, without aggravation, iodide of potassium may be used to promote absorption, and the usual measures intended to restore muscular and nervous power, as massage, baths, and electricity.

AFFECTIONS OF THE SUBSTANCE OF THE CORD.

General Considerations—Topical Diagnosis.

Two separate sets of pathological changes invade the substance of the spinal cord. In one they are confined with marked constancy to certain definite areas which have precise functions, residing in "systems of fibres," so that the clinical phenomena of the disease are exactly defined. These diseases are called *system-* or *systemic-* diseases. They include such as anterior poliomyelitis, confined almost exclusively to the anterior cornua of the grey matter; tabes dorsalis, confined to the posterior columns; amyotrophic lateral sclerosis, to the lateral columns. Why certain definite areas of the cord are specially involved and why this peculiar selective systemic implication, we do not know any more than we know why certain poisons, such as curare, strychnine, and lead, select certain tissues for their operation.

In the second group there is no such limitation of tissue invaded, but the cord in its entire transverse section is involved in one large focus, or several combined foci separated by areas of sound tissue are invaded. In this group are included acute and chronic diffuse inflammations, the hemorrhages and traumatic lesions, multiple sclerosis, etc. These are the *non-systemic diseases.* Since in the diffuse affections all the parts involved in the systemic diseases are also affected, the symptoms of the latter are found associated with those growing out of the diffuse lesion. The diagnosis arrived at by a study of these symptoms is still, however, mainly a "topical" one, for it is an important fact growing out of the functions of the cord that all diseases involving certain areas produce the same symptoms, whence we infer the seat of the lesion rather than its nature or exact cause. This may, however, be determined with a varying degree of certainty from other symptoms. A further peculiarity of all disease of the substance of the cord is that its symptoms are commonly bilateral. This depends upon two causes: first, the fact that the two halves of the cord are in such close proximity that almost any cause of a violent kind, such as hemorrhage, affecting one-half must also extend its

influence to the other; and, second, the causes of system-diseases commonly select corresponding parts in the two halves of the cord for their operation. Again, symptoms vary according as the lesion affects the conducting path to and from the brain, as it attacks parts functionally independent, or the nerve roots. The symptoms are accordingly known as "conducting" or "central," and as "root" symptoms.

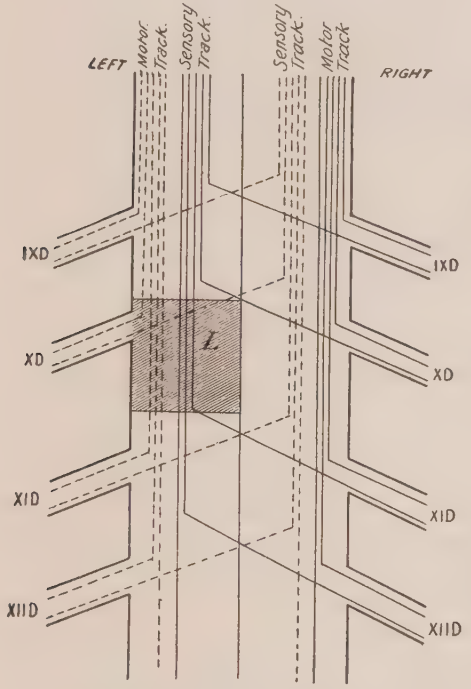


FIG. 66.—DIAGRAM OF LESION SHOWING BROWN-SÉQUARD'S PARALYSIS.—(*After Starr.*)

L. Lesion in left half of cord cuts off motor impulses to left leg, sensory impulses from right leg, and sensory impulses from XI Dorsal nerve.

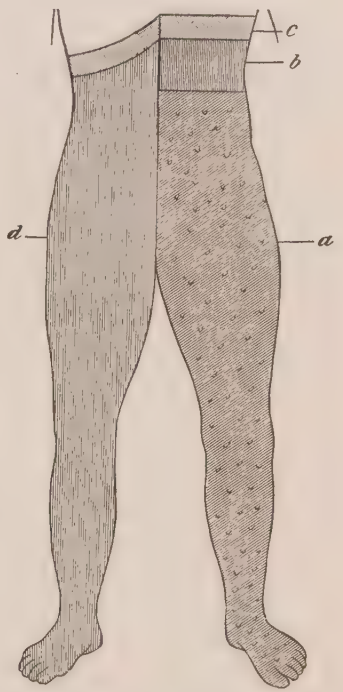


FIG. 67.—SCHEMA SHOWING CHIEF SYMPTOMS IN LEFT UNILATERAL LESION OF THE DORSAL CORD.—(*After Erb.*)

Oblique shading signifies motor and vasomotor paralysis; vertical, cutaneous anæsthesia; dots, cutaneous hyperæsthesia. *b.* Small anæsthetic zone. *c.* Small hyperæsthetic zone.

UNILATERAL LESION OF THE SPINAL CORD.

SYNONYM.—*Brown-Séquard's Spinal Paralysis.*

Etiology.—A sharply defined lesion of a lateral half of the spinal cord occurs as the result of knife cuts or sword stabs, though inflammatory processes, compression, especially by tumors of the cord, may also cause similar symptoms. The phenomena of this condition were first studied clinically and experimentally by Brown-Séquard.

Symptoms.—The effect of such injury is to interrupt the continuity of motor fibres below their point of crossing in the medulla oblongata and the sensory fibres, in part, at least, after they have crossed, since many of these cross immediately after entrance into the cord. This being under-

stood, there is *paralysis of motion on the same side* and *loss of sensation on the opposite side*, varying, however, with the seat of lesion. If the lesion is in the cervical cord the paralysis is of the arm and leg on the same side—spinal hemiplegia and loss of sensation in the arm and leg of the opposite side. The anæsthesia may be to painful and thermic sensation only, the tactile sense being unimpaired. The muscular sense is diminished on the same side as the lesion, where also the skin is hyperæsthetic and irritation is more keenly felt, slight pricks being very painful and tickling of the soles of the feet being felt unusually. The diminution of the muscular sense is explained by Brown-Séquard himself on the ground that its fibres run their course in the spinal cord uncrossed until the medulla is reached, as do the motor fibres. This, as we have seen, is generally conceded.

Above the hyperæsthetic level on the same side with the lesion there is usually a small anæsthetic zone corresponding closely with the level of the lesion itself in the spinal cord, and above this again another small hyperæsthetic strip. The anæsthesia is due to the fact that the sensory fibres coming from the same side are cut just as they enter the cord. The anæsthetic zone, as explained on p. 840, is somewhat lower down than the exact seat of the disease, because of the overlapping of the upper sensory area. The hyperæsthesia on the paralyzed side has been said to be unexplainable, but may it not be due to an increased vascularity, due to section of the vaso-constrictor nerves? It may be for this reason also that the temperature is higher on the side of the lesion from one to two degrees F. (0.5° to 1° C.). The upper hyperæsthetic zone may be similarly explained. There is sometimes, also, a small hyperæsthetic zone above the anæsthetic area on the paralyzed side.

The tendon reflexes are usually increased on the paralyzed side and there is often a good ankle clonus, explainable by the interruption of the inhibiting influences coming from above.

Diagnosis.—These symptoms are rarely as sharply defined as the above description would indicate, but the lesion may be generally recognized by the prominence of a few. The **prognosis** depends upon the cause of the lesion and the **treatment** as well.

SECONDARY SYSTEMIC DEGENERATIONS OF THE SPINAL CORD.

These succeed cerebral lesions and lesions in the spinal cord itself. They depend upon the fact, several times referred to, that a trophic influence is exerted by ganglion cells upon the fibres originating from them, so that the latter degenerate when the conduction of the trophic influence is interrupted or when the trophic ganglion cells are destroyed. For motor fibres such ganglion cells exist in two situations—in the motor portion of the cortex cerebri and in the anterior cornua of the spinal cord. The former exert a trophic influence on the motor fibres arising from them which extends as far as the latter. For sensory fibres in the cord the trophic influence resides in ganglion cells, the exact seat of which is not known—probably spinal root ganglia, possibly posterior cornua. The fibres of the lateral cerebellar column in the

periphery of the cord are connected trophically with the cells of the column of Clarke, or posterior vesicular column—the group of cells in the inner part of the neck of the posterior horn.

Secondary Degeneration in the Spinal Cord after Cerebral Lesions.—Hence, if there be disease in the motor area of the cortex or in any part of the motor tract in the brain,—that is, in the motor fibres of the corona radiata, the internal capsule, the crus, or the pons,—interrupting conduction, a secondary degeneration of the motor fibres takes place below in the related pyramidal tracts, anterior on the same side, lateral on the opposite side, as far as the anterior cornua of the grey matter. In many cases there is slight degeneration in the lateral tracts on the same side as far as the lumbar region, showing that some fibres of each anterior pyramid find their way to the lateral tract on the same side. The relative proportion of the crossed lateral fibres and the anterior fibres that remain uncrossed varies within limits. In cases where no anterior pyramidal tracts exist—that is, where all the fibres pass over to the lateral column of the opposite side—there is no descending degeneration of the anterior column.

Secondary Degeneration of the Spinal Cord after Transverse Lesion of the Cord Itself.—If a lesion be seated in any part of the cord affecting more or less its transverse section, the interruption of conduction in these fibres is also followed by secondary degeneration, which may be traced in two directions, upward and downward, ascending and descending. Such lesions may be transverse myelitis, compression of the spinal cord, and tumors—any lesion, in fact, involving the half of the cord. The descending degeneration affects the pyramidal tract exactly as the descending degeneration after cerebral lesions, but as the transverse disease affects the pyramidal tract on the two sides, the secondary descending degeneration will affect both lateral pyramidal tracts, if these exist below the seat of lesion.

The ascending secondary degeneration developing upward from the seat of lesion affects two systems: the columns of Goll—*i. e.*, the posterior median column—and the lateral cerebellar tracts on the periphery of the lateral columns, because the conduction in these parts which receive their trophic influence from the more peripheral ganglionic cells is interrupted. Where these ganglionic cells are which thus act trophically on the fibres of Goll, as has been said, is not precisely known. In addition, the con-

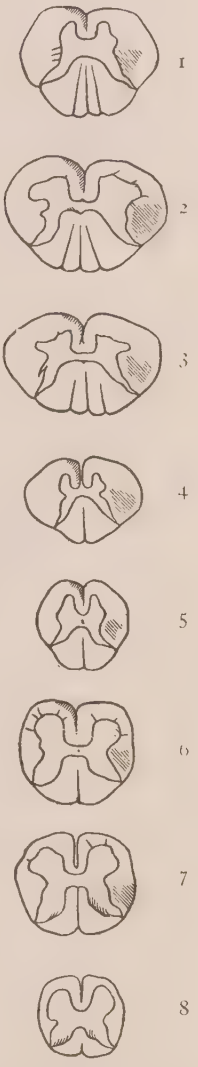


FIG. 68.—SECONDARY DESCENDING DEGENERATION OF THE PYRAMIDAL TRACTS IN A PRIMARY LESION OF THE LEFT HALF OF THE CEREBRUM.—(*After Erb.*)

The lateral pyramidal tract of *right* half is degenerated down to lowest part of lumbar region. 1–8. The anterior pyramidal tract at left half is degenerated to beginning of lumbar enlargement.

nection of the lateral cerebellar tracts with the cells of the column of Clarke is cut off, and when this occurs or these cells are destroyed an ascending degeneration of the lateral cerebellar tract ensues, which may be traced upward into the restiform body.

Clinical Significance of the Secondary Degeneration.—This is disputed, Charcot and the French clinicians generally ascribing to them the contractures and increase of the tendon reflexes in the paralyzed limbs of hemiplegia, while Strümpell and others think they have no clinical import.

Secondary Degeneration in the Spinal Cord after Injuries of the Cauda Equina.—After fractures, caries, or other injury to the lower lumbar vertebræ or sacrum, producing injury to the cauda equina, or as

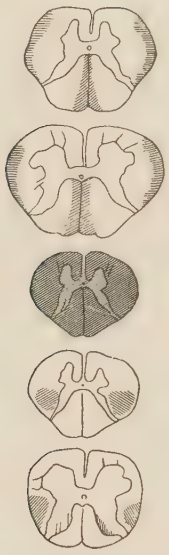


FIG. 69.—SECONDARY ASCENDING AND DESCENDING DEGENERATION IN A TRANSVERSE SECTION OF THE UPPER DORSAL REGION.

The columns of Goll and the direct cerebellar tracts are degenerated upward, the lateral pyramidal tracts downward. —(*After Strümpell.*)

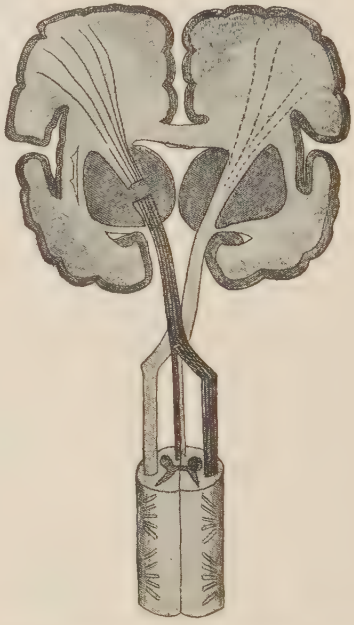


FIG. 70.—DIAGRAM OF DESCENDING DEGENERATION OF A PYRAMID WITH A LESION IN THE LEFT INTERNAL CAPSULE.—(*After Edinger.*)

the result of new growths in this region, a secondary ascending degeneration takes place in the cord after the ruptured continuity has existed for some time. This is due to involvement of the posterior nerve roots; whence the degeneration is confined to the posterior columns of the spinal cord, and in its distribution it resembles closely the state of the cord in tabes dorsalis. In the lumbar cord all of the posterior column is degenerated except a little median zone and the most anterior portion. The descending degeneration grows smaller as we ascend, and finally is confined in the cervical cord to the region of the columns of Goll, which include, in part at least, the prolongation of the fibres from the root zones of the lumbar cord.

Acute Affections of the Spinal Cord.

DISTURBANCES OF THE CIRCULATION OF THE SPINAL CORD.

Congestion.—Nothing is known of the phenomena of congestion of the cord as separated from inflammation from the standpoint either of clinical observation or post-mortem examination. The well-known fact that active hyperæmia disappears post mortem, and that even when inflamed the white matter is rarely found congested after death, renders such examination unavailable.

Anæmia of the cord has been studied clinically and experimentally. The phenomena of paraplegia which succeed profuse hemorrhages, as of the uterus post partum and from the stomach, are fairly ascribable to anæmia of the cord.

This is confirmed by some experiments of Stenson, who compressed the abdominal aorta of an animal with the effect of causing almost immediate paralysis of the extremities; and of C. A. Herter, at Johns Hopkins Hospital, in which paraplegia supervened a few minutes after the application of a ligature to the aorta, followed more slowly by paralysis of the sphincters. Within thirty-six hours there were marked changes in the ganglion cells of the anterior horns in the lumbar segment, and later signs of myelitis. Within fourteen days contracture of the limbs set in with atrophy of the muscles and with fibrillar twitchings. Obstruction of the aorta by thrombi and emboli has been followed by similar clinical phenomena. On the other hand, the intensest degrees of general anæmia observed in acquired disease, such as is found in pernicious anæmia, in which the cord may reasonably be expected to share, are unattended by any symptoms referable to such a state of the cord. At the same time observations are daily multiplying to go to show that the conclusions of Lichtheim as to degeneration of the posterior columns of the cord in pernicious anæmia are correct.

Embolism and *thrombosis* of the spinal arteries have been produced experimentally with resulting choreiform movements. Thrombosis of the smaller vessels occurs in connection with endocarditis. *Endarteritis* or its results are frequently found post mortem in persons over fifty as a nodular periarteritis or endarteritis in syphilis, sometimes associated with gummy tumors of the meninges; and as an *endarteritis obliterans* with thickening of the intima and narrowing of the lumen, involving chiefly the arteries of medium and larger size. Miliary aneurism and aneurism of the larger vessels of the spinal cord are very rare.

Hemorrhage into the Substance of the Cord.

Etiology.—Hæmatomyelic hemorrhage is at most a rare event. Whether it ever occurs primarily independent of disease is reasonably questioned. Its possibility must, however, be admitted, at least, as the result of traumatic causes such as falls. Great physical exertion is another

admitted possible primary cause, so are cold and exposure, tetanic and other convulsions. Repeated coitus is mentioned by Gowers as having been followed by hemorrhage in the grey substance at the top of the bulbar enlargement, and this cause may have operated in a patient of my own who was suddenly seized with a paraplegia during one of repeated acts and while in vigorous health.

Secondary hemorrhage is more frequent and cavities in the cord, such as are due to congenital defects and arrests of development.

Morbid Anatomy.—The cord may be distended, infiltrated, or lacerated by the hemorrhage escaping into the meninges; if not too copious it may be limited to the grey matter and extend up and down the cord to considerable extent. The blood undergoes the usual changes after effusion, coagulates, becomes darker hued, then yellow, and, finally, at times, the seat of the hemorrhage becomes occupied by a cyst, while numerous hæmatoidin crystals will be found in the residue. The blood may remain liquid for a long time.

Symptoms.—The effect is, as a rule, sudden paraplegia,—paraplegia, because of its seat in the lumbar enlargement. If the cervical part of the cord, the arms as well as legs are involved, and there may be embarrassed respiration, often sharp pain in the extremities supplied by the nerves given off at the seat of the effusion. Loss of sensation succeeds later, while the reflexes also disappear. Myelitis is often developed as a consequence of the irritative presence of the clot, and there follow its usual symptoms, including trophic changes and fever.

Diagnosis.—This is based upon the suddenness of the consequent events—acute pain and paraplegia—under the etiological conditions described, trauma or coitus. Hemorrhage into the spinal meninges is, of course, equally sudden or nearly so, but the symptoms of injury to the cord are less prominent and there is little or no fever. In meningeal hemorrhage the pain is more severe and symptoms of irritation are more apt to precede the paralysis, while the paralytic symptoms are less persistent.

Prognosis.—The accident is rapidly fatal in the severest cases. In others paralysis may exist for a long time or be permanent. In others there is slow but persistent improvement and the patient may even recover. Hemorrhage into the cervical region of the cord is more serious because the respiratory centre is apt to be invaded. The presence of trophic changes renders the prognosis as to recovery more unfavorable.

Treatment.—This is identical with that for hemorrhage into the membranes. Absolute rest is the primary essential condition. Ice may be applied to the spine over the seat of the hemorrhage and leeches or wet cups may be applied to the same locality with reasonable expectation of benefit.

If the case is not immediately fatal, improvement is apt to follow the contraction of the clot, as in cerebral hemorrhage. Then, from the theoretical standpoint, ergot is indicated. Full doses, ½ drachm (two c. c.) to one drachm (four c. c.), of the fluid extract, or three to five grains (0.2 to 0.32 gm.) of ergotin, repeated in two or three hours with a view to produce a profound effect early, should be given. Muscular nutrition should be kept up by massage and electricity.

CAISSON DISEASE.

SYNONYM.—*Diver's Paralysis.*

Definition.—A paraplegia and sometimes general paralytic affection which happens to workers in caissons after their return to the surface from the compressed atmosphere of the caisson.

Pathology.—This is not thoroughly determined. That it is, however, a spinal lesion a study of the symptoms goes to show. It is ascertained that the pressure about the body required to produce it must exceed three atmospheres, that the symptoms do not come on until the patient returns to the surface, and they are more prone to occur the longer has been the exposure. Under these circumstances the most reasonable explanation is that the symptoms are due to the escape from the blood of gases with which it has become charged during the exposure to the high pressure, gases which under ordinary circumstances escape gradually by the lungs. Gas escaping in the more rapid manner exerts a pressure which arrests the function of the nervous system for the time being. At one time local hemorrhages were supposed to be the cause, but in the few autopsies which have been made nothing has been found which would explain the symptoms from that standpoint. Leyden found in a case dying on the fifteenth day small, irregular fissures in the mid-dorsal region, chiefly within the posterior and hinder part of the lateral column, filled with round cells, but containing no blood corpuscles. It has been suggested that the fissures were produced by the sudden escape of gas, and were finally occupied by wandered-out round cells. Schultzer found in a case dying at two and a half months these fissures together with signs of myelitis. Leyden suggests that the dorsal region suffers because of its relative softness as compared with other parts of the nervous system.

Symptoms.—These come on, as stated, not during exposure to the abnormal conditions, but on return to the normal atmospheric pressure, often immediately, always within a half hour. *Paraplegia* or *hemiplegia* is characteristic and essential, although the onset is usually preceded by *pain in the ears and joints*, especially the larger joints. The latter may be affected without paraplegia in shorter periods of exposure, and there may be *tenderness in the limbs.* There may also be *dizziness* and *headache.* The onset of the paraplegia is, however, sudden. Sensation may also be impaired, though the impairment is imperfect and irregular. In severe cases the sphincters are affected. Monoplegia and hemiplegia are rare, and when present they are apt to be transient. The cerebral symptoms may be more severe, amounting to sudden loss of consciousness and continuing as coma. Such cases are usually fatal in a few hours. Those who are less accustomed to work are the most liable to suffer, but the power of resistance also varies in different individuals.

Diagnosis.—The diagnosis is not usually difficult, the history of the case furnishing the conditions and explaining the symptoms.

Prognosis.—The prognosis is mostly favorable, the symptoms generally passing away in the course of a few days, although in severe

cases they may last for weeks or months, and may even be permanent. In fatal cases death may occur in a week or as late as the end of two or three months, with the symptoms of myelitis.

Treatment.—None of an active character is usually required. Morphine may be needed to relieve pain. The prophylactic treatment is of greatest importance. This consists in avoiding too long continuance of work under pressure, and care should be taken to make the transition from the higher to the lower pressure gradually. Dr. Andrew H. Smith, of New York City, who has paid much attention to the subject, with large opportunities of study during the building of the Brooklyn Bridge, advises that in passing through the lock from the low to the high pressure at least five minutes should be taken.

MYELITIS (Acute and Chronic).

Synonyms.—*Diffuse Myelitis; Transverse Myelitis.*

Definition.—The line of demarcation between acute, sub-acute, and chronic myelitis is not sharp, but the term *acute* is applied to that form of inflammation in which the symptoms come on suddenly. When requiring two to six weeks for their development, it is called *sub-acute.* When a still longer time elapses before the symptoms reach a decided degree of intensity, it is *chronic.* At the same time it is plain that no very sharp line of demarcation can be drawn between these forms. When the whole thickness of the cord is involved in a small vertical extent—a common form—it is said to be *transverse;* if an extensive area, *diffuse;* when one small area, *focal;* when many foci, contiguous or distant, it is *disseminated.* Inflammation of the grey matter around the central canal, extending into the intermediate grey substance, is called *central* myelitis —parenchymatous and interstitial.

Etiology.—The cause is often undiscoverable. There is an occasional hereditary tendency to it. It may occur at any age, but is more common in adult males. It may result:—

(1) From repeated exposure to wet, cold, or from over-exertion, or both combined.

(2) Rarely from the infectious diseases, as small-pox, typhoid fever, or puerperal fever.

(3) From syphilis, either as the direct result of the infection, or secondarily from invasion of the cord by syphilitic tumors; the former appears a year or eighteen months after the primary inoculation, the latter as a late manifestation.

(4) From tumors other than syphilitic.

(5) From injuries to the spinal column, especially fractures, and from caries of the vertebræ. Concussion alone may produce it. The former causes are more apt to produce local inflammations, and, in fact, it is extremely difficult, nay, often impossible, to distinguish their inflammatory results from the effects of compression.

Morbid Anatomy.—Considerable experience is necessary to be able to recognize the changes in many cases of myelitis. The separation of

the process into different stages is difficult or impossible. To the untrained naked eye the cord often appears quite normal. Adhesions and opacities of the pia, easily recognized, have generally no significance. The expert examiner may recognize by the touch that the cord over a certain extent may be softer and more flexible or harder and firmer. On section the substance of the cord rises up more than in the normal state, the outline of grey matter is less distinct, and sometimes has a hyperæmic, reddish coloring, while the white matter is of a reddish-grey color. Sometimes there may be minute hemorrhages.

These changes become much more apparent if the cord is incised at intervals of one-fourth inch, but allowed to remain continuous at one part, like the leaves of a book, and hardened eight to ten weeks in Müller's fluid.* The normal parts of the cord assume a greenish color from the staining of the medullary sheaths, while the parts that have lost this covering from disease do not take the stain. Similar but less distinct differences are found in the elements of the grey matter.

In like manner, microscopic examination of the fresh cord affords little definite information. There may be numerous granular fatty cells (compound granule cells), but these simply indicate a diseased state and nothing more. Corpora amylacea may be present, also blood discs and leucocytes. Thin sections stained by carmine give a very different picture even to the naked eye, the diseased tissues taking on the darker staining because of its greater richness in connective tissue. To the microscope it is found that in these portions the normal tubular nerve tissue has partly or almost wholly disappeared. In some places axis cylinders remain, having lost only their medullary sheath, in others the whole fibrous nervous matter has gone. The changes in the ganglion cells are less definite, but they have lost their processes and are rounder, while the increase of connective tissue goes on *pari passu* with the destruction of the proper nervous matter. The former occupies the enlarged meshes caused by the disappearance of the latter. The nuclei of the connective tissue increase and also Deiter's spider cells. The fatty granular cells may also be recognized, provided no alcohol has been used in hardening. The blood-vessels are dilated and distended, and there may be hemorrhagic extravasation, thickening, and hyaline degeneration of the vessel walls, at times numerous corpora amylacea.

The part invaded varies, the upper half of the dorsal cord being most frequently involved, but there may be cervical myelitis and rarely lumbar myelitis. There may be a central focus and numerous small foci in the vicinity. The extent varies at different levels. The softer reddish conditions indicate the more acute stage, the harder, greyer, and more contracted the chronic stage—sclerosis.

In localized acute myelitis affecting the white and grey matter after injury, the cord is swollen, the pia injected and soft, and on cutting the membrane an almost diffluent fluid may escape. In less degree the appearances first described are present. It is in the cases which arise particularly by invasion from without or from compression in which the

* Potassium bichromate 2½ parts, sodium sulphate one part, water 100 parts.

white matter is involved. In extensive cases the membranes are often involved, producing myelo-meningitis.

Localized areas of softening with blood accumulation constitute *red softening*. *Abscess* of the substance of the cord scarcely exists, but one or two cases have been reported. Pus only forms in the cord in considerable quantity in purulent meningitis.

Symptoms.—The distinctive symptoms of myelitis may be preceded by constitutional disturbance, including *headache* and general *malaise*, and even *chill*, *fever*, and *delirium*. Temperature of 107° and 108° F. (41.7° to 42.2° C.) has been noted. These symptoms are, however, unusual.

The characteristic symptoms vary greatly with the part of the cord involved, and no picture can be drawn to suit such differences of locality. I prefer, therefore, following Strümpell, to describe the symptoms more or less common to all localities, and after this such modifications or peculiarities of these as enable us to locate more precisely the process. The former include :—

(1) *Symptoms of Motor Paralysis.*—They are the most conspicuous and commonly first-recognized sign of developing transverse myelitis. Beginning with tired feeling in one or both legs, followed by evident weakness and then dragging, the paresis continues to grow until the patient is totally unable to make any active movement with his legs. This implies that the lateral columns of the cord and especially the posterior part of the lateral columns, carrying the lateral part of the pyramidal tract, are involved, cutting off the motor impulses. This motor paraplegia can occur in every form of myelitis, lumbar, dorsal, or cervical, but in the first two the upper extremities are intact. In the cervical, however, paralysis of the upper extremities also takes place. If one side of the body is involved more than another it implies that one-half of the cord is more intensely affected.

(2) *Symptoms of Motor Irritation.*—These consist in spontaneous twitchings of the muscles of the paretic limbs, either rapid and short or slow and persistent. They occur at the beginning and during the course of the disease, and are variously severe. It is not always easy to distinguish them from increased reflexes, whence their diagnostic value is not great. Ataxia and intention tremor may occur in connection with involvement of the upper extremities and in the convalescence of acute cases, but they are very rare.

(3) *Disturbances of Sensibility.*—These in marked degree occur much later in the disease than the motor phenomena. At the beginning there may be numbness, formication, tingling, and even girdle sensations, but severe pain is rarely present. When pain is present it is an evidence of involvement of the vertebræ or meninges. There may be slight impairment of sensibility, but, on the whole, the *absence of perverted sensibility* in all except advanced degrees is a characteristic symptom. In such advanced degrees, in addition to anæsthesia, there may be paræsthesia and even striking hyperæsthesia. The involvement of sensibility means that the posterior columns or cornua are both invaded, the latter always where there is marked anæsthesia.

Disturbances of sensibility are useful in determining the seat of the

lesion in the cord, because the lesion corresponds very nearly, or sufficiently so for practical purposes, to the level of the seat of the modified sensibility. Thus in myelitis of the lumbar region the impairment extends to the umbilicus or slightly higher, in the lower dorsal to the ensiform cartilage, in the upper dorsal to the level of the axillæ, while in the cervical the sensibility of the upper extremities is impaired, but total anæsthesia is very rare.

(4) *The Reflexes.*—The effect of myelitis on the reflexes varies greatly with the seat of the disease and the degree and extent of the lesion. In the very incipiency of an inflammation of the cord we may expect all the reflexes, cutaneous and tendon, centring in the part involved to be increased, but with the progress of the disease the effect varies greatly and must be discussed in detail.

(*a*) *Skin Reflexes.*—The reflex arcs of the cutaneous reflexes are at about the same level as the distributions of the corresponding sensory and motor spinal nerves. Their connection with reflex inhibitory fibres from above is to be remembered, irritation of which diminishes and interruption of which increases the sensitiveness of the reaction. In extensive lumbar myelitis the reflex path is broken and the cutaneous reflexes of the lower extremities are diminished, running about *pari passu* with altered sensibility. In dorsal and cervical myelitis the arc for the lumbar reflexes is intact, and if the reflex inhibitory influence is removed they may even be increased. Experience shows, however, that the skin reflexes in the legs may be diminished even in dorsal or cervical myelitis, in which event there must be loss of irritability in the fibres. The cremaster reflex has its arc at about the level of the 1st lumbar nerve, hence its loss means disease there. The lower abdominal reflex corresponds to the lower dorsal cord and the upper to the level of the 4th to the 7th dorsal vertebræ.

(*b*) *Tendon Reflexes.*—The reflex arc of the patellar reflex lies at about the level of the 2d to the 4th lumbar nerve. It fails with disease of the middle part of the posterior columns and the anterior cornua of the grey matter of the lumbar cord. The ankle clonus has its reflex arc at the level of the 1st sacral nerve. It is always absent in extensive disease of the posterior columns and grey matter of the lumbar cord in this vicinity. The absence of reflexes of the lower extremities is, therefore, one of the most valuable signs of myelitis of the lumbar cord. In almost all inflammations above the lumbar cord, that is, of the dorsal and cervical portions, there is a decided increase in the tendon reflexes of the lower extremity, because these lesions destroy the reflex inhibitory influence. When, therefore, alongside of this it is remembered that the fibres which influence the condition of the tendon reflexes run chiefly in the *lateral* columns of the cord but are not identical with the pyramidal fibres, we may conclude that the lateral columns are involved. In cervical myelitis the tendon reflexes of the upper extremities are often increased.

(5) *Disturbances of the Bladder and Rectum.*—These are common in myelitis. There is, first, delay in micturition, finally accomplished by extra straining, but later all power to empty the bladder is lost—the *detrusor urinæ* is paralyzed. Still later the *sphincter vesicæ* is paralyzed, and then

there is incontinence of urine. These symptoms occur in connection with paralysis in any part of the cord, but they point to involvement of the posterior columns. The ultimate effect is almost invariably a cystitis, the result partly of decomposition, induced by germs introduced through repeated catheterization, even when most cautiously conducted, partly by the entrance of germs through the patulous sphincter. Such cystitis has also been ascribed to trophic influence. To the cystitis may succeed pyelitis and purulent pyelo-nephritis.

In myelitis there is at first obstinate constipation, followed by paralysis and incontinence of fæces. This symptom does not give any information as to the seat of the myelitis. Defecation and micturition are sometimes reflexly aroused in abnormal degree when there is increased reflex irritability. Sexual functions residing probably in the upper lumbar cord are also often deranged in myelitis.

(6) *Trophic Disturbances.*—These are most important symptoms, and valuable also in diagnosis. In cervical and dorsal myelitis the trophic centres in the lumbar cord are intact. The paralyzed muscles, therefore, do not atrophy, though they may be a little soft from want of use. They retain their normal electrical excitability. On the other hand, genuine atrophy and the presence of the reaction of degeneration show the anterior grey cornua or the fibres of the anterior roots of the lumbar cord to be involved; in the upper extremities they show that the same portions of the cervical cord are involved. Bed-sores are among the trophic phenomena, the possibility of the occurrence of which should be always vividly present. They are among the most unpleasant and most unmanageable symptoms, yet they may be guarded against, for, although favored by the deranged trophic influence, they require an exciting cause, such as pressure, the irritation of secretions, or foreign substances to originate them. They occur over the sacral and gluteal regions, more rarely on the feet and inner side of the knees. The total anæsthesia often associated with advanced stages of the disease coöperates to permit the action of the exciting causes.

Other trophic effects often met are drying and hardening of the skin; also thick and brittle nails. Vaso-motor disturbances also occur, producing congestion and mottling, and there may be slight œdema of the paralyzed parts; also sweating, which may be localized. The temperature of the affected limbs may be lowered and multiple arthritis may occur.

(7) Disturbances in the area of distribution of the cranial nerves almost never occurs, though bulbar symptoms have been met with in rare cases of cervical myelitis, where the process has extended to the medulla. Optic neuritis and pupillary changes, also vomiting, hiccough, slow pulse, sinking to 20 and 30, dysphagia, dyspnœa, and syncope, have been observed in cervical myelitis.

By uniting the symptoms detailed and their mode of manifestation, we may draw conclusions with a certain degree of positiveness as to the portion of the cord involved. Thus in *cervical myelitis* there is paraplegia of the legs, along with various degrees of deranged function in the

arms, especially if the lesion is at the level of the 6th or 7th cervical nerves, when the arm paralysis is more or less complete. Ultimately there may be loss of sensation. Sometimes the muscles of the shoulder are spared. In rare instances the arms only are involved, the so-called cervical paraplegia. There may be atrophy of separate muscle regions in the arms, but the muscles of the legs are not affected. The tendon reflexes are increased in the legs and often in the arms, while there may be spastic symptoms in each. Cutaneous reflexes are retained in the legs, often increased. Disturbances in the bladder and rectum are present.

In *dorsal myelitis* the arms are intact, but there is motor and ultimately sensory paraplegia of the legs without degenerative atrophy. In some cases a transverse myelitis of the region involves the anterior horns above and below it, producing flaccid muscles, wasting, fibrillar contractions, and reactions of degeneration. Tendon reflexes are increased, especially in myelitis of the upper cord; skin reflexes present, rarely increased; also disturbances of the bladder and rectum. The pyramidal fibres below the lesion may undergo the secondary degeneration, and there may be ascending degeneration of the posterior median columns.

In *lumbar myelitis*, arms are intact; legs as in dorsal myelitis; tendon and skin reflexes diminished or absent; sometimes degenerative muscular atrophy with reaction of degeneration; disturbances of bladder and rectum.

Diagnosis.—The difficulty of diagnosis is sometimes very great, because identical symptoms may be produced by other diseases, especially pressure diseases of the cord such as are caused by tumors or hemorrhages, possible conditions which must be carefully sought.

Landry's acute ascending paralysis and multiple neuritis present some striking resemblances. Both of these present rapidly progressive motor paralysis, but though sensory derangement may be a late development in myelitis, it is still a symptom belonging to it, rather than to Landry's paralysis, which is a motor affection, while the trophic symptoms, the paralysis of the bladder and rectum, rapid wasting, electrical disturbances, and fever, pertain to myelitis. The resemblance to multiple neuritis is closer. In this, however, anæsthesia is less complete, the wasting less rapid, bladder and rectum involvement almost never present, and there are never trophic changes.

Duration and Course.—Almost all cases of myelitis are chronic, seldom lasting less than a year, often two or three years and even longer. The term acute is not, therefore, applied in its ordinary meaning, implying rapid course and early termination, but is used to indicate cases which develop rapidly to their acme as contrasted with those that are slow. Even these cases become chronic. The possibility also of a rapidly developing case passing to a fatal termination in five to ten days may, however, be admitted. Moreover, recoveries are not impossible, though very rare. Certain cases, after reaching a given stage, remain as to symptoms in *statu quo*, by which it is understood that the local lesion has healed while function is not regained because of the impossibility of restoring the normal structure of the cord. Remissions and improve-

ments are less infrequent. Death is usually the result of exhaustion, although it may be from intercurrent disease.

Treatment.—This is for the most part to be directed to the relief of symptoms, no curative means existing beyond what nature herself provides. In cases with acute onset and pain, cups may be applied to the back. Even in syphilis it is thought by some useless to give the usual remedies, but it is safer in cases of suspected disease to give iodide of potassium in ascending doses to the extent permitted by the stomach, while the mercurial effect should be brought about by inunctions, 30 grains (two gm.) to a drachm (four gm.) of mercurial ointment being rubbed in daily in the arm-pits or inner surface of the thigh. Tonics, such as iron, quinine, and strychnine, are useful in this as in other prolonged affections.

The most painstaking attention must be given to the skin by bathing with alcohol and thoroughly drying after all washing, in order to prevent the excoriations which are often the beginnings of bed-sores, while the irritating effects of the excretions must be carefully watched, and if catheterization is necessary it must be practised with the closest attention to antisepsis. It may even be desirable to keep a soft catheter permanently in the bladder, to which a long tube is attached and the bladder thus kept drained. Should cystitis supervene the bladder is to be washed out as directed on p. 705. The rectum should be emptied when possible by enemata rather than by purgatives, which should be cautiously used.

Electricity is elaborately directed by the German authors, although they admit that in the majority of instances it is principally a diversion to the patient. In the later stages, however, of the forms in which there is atrophy of the muscles, some advantage may be expected. The constant current is most recommended by large electrodes placed over the vertebral column, and a moderate stabile current or slowly labile current is passed for four or five minutes through the supposed seat of the disease. Peripheral galvanization or faradization of the paralyzed muscles and nerves should also be employed. Massage is also useful, perhaps more so than electricity. The bladder may also be treated by electricity.

The bath treatment is carried out to various degrees of elaboration. The simple tub bath with warm water furnishes the easiest form and may be quite useful in the cases with spastic symptoms, at a temperature of 85° or 90° (24° to 26° C.). The baths should be at first limited to ten or fifteen minutes three or four times a week, and if well borne may be increased to an hour daily. The water may be impregnated with sodium chloride, using either the sea salt or common salt, four to six pounds of the former or five to ten pounds of the latter, to the bath. Where the patient is able to travel and avail himself of the actual sea baths they may be expected to be beneficial. The Hot Springs of Arkansas and Virginia in this country may be resorted to. In Europe the thermal waters at Rehme and Nauheim in Hesse, Ragatz in Switzerland, Teplitz in Austria, Wildbad in Würtemberg, Gastein in Salzburg in Austria, and Wiesbaden in Baden are among those recommended; also the mud baths of Marienbad in Bohemia and Elster in Southern Saxony.

MYELITIS OF THE ANTERIOR HORNS.

SYNONYMS.—*Acute Anterior Poliomyelitis of Children; Spinal Paralysis of Children; Atrophic Spinal Paralysis; Infantile Palsy; Essential Paralysis of Children.*

Definition.—A febrile disease of children usually under three years of age, in which there is paralysis with rapid wasting of certain muscles.

Historical.—The clinical phenomena of this disease were described as early as 1840 by Jacob von Heine. The same physician twenty years later suggested that a spinal malady lay at the bottom of it, but it was reserved for Prevost and Velpeau, Charcot and Joffroy, to demonstrate the lesion which justifies the term "spinal paralysis of children" for the older "essential paralysis of children."

Etiology.—Especially a disease of later infancy; it may occur at all periods of life, but is ten times more frequent in the first decade than in all the rest of life. It is more common in boys than in girls, and in the warm months, as pointed out by Wharton Sinkler, who ascertained that in Philadelphia four-fifths of the cases commenced in May to September. There seems to be a slight family tendency. It has occurred in epidemic form in Stockholm, where Medin reports 29 cases from August 9th to September 23d. A very remarkable epidemic in this country, occurring at Rutland, Vermont, was reported by Caverly in the New York Medical Record, Vol. II, 1894, in which 190 cases occurred during the summer, of which 85 were under six years of age, and 18 died. It has been ascribed without foundation to dentition, to cold and over-exertion, and very frequently erroneously attributed by patients to falls due to the carelessness of nurses. Most children are attacked while in perfect health, although previous exhausting diseases may reasonably be held responsible.

Morbid Anatomy.—The disease occupies more frequently the cervical or lumbar enlargement. The usual lesion found in old cases, which are those which commonly come to autopsy, is an atrophy of one anterior cornu which is changed to a dense sclerosed tissue, whence the ganglion cells have almost or totally disappeared, often pierced by thickened and dilated vessels. A similar condition is found in the corresponding cornua of the cervical enlargement if it is in the arm, and of the lumbar if in the leg. If bilateral, both cornua would be involved. The corresponding anterior nerve roots are atrophied and the muscles supplied by them are wasted, undergoing a fatty and sclerotic change. The white matter of the cord in the vicinity may be involved to some extent, resulting in sclerosis and slight reduction in size. Opportunities for autopsy in the earlier stages are rare, but a condition of acute hemorrhagic myelitis, with degeneration and rapid destruction of the ganglion cells, has been found by David Drummond and Charlewood Turner.

Symptoms.—*Sudden onset* is characteristic of the disease, although there may be, and probably oftener than is noticed, slight previous indisposition with feverishness. A child apparently perfectly well and lively may be suddenly taken with *fever*, 101° to 103° F. (38.3° to 38.9° C.), sometimes reaching 105° to 106° F. (40° to 41° C.); *headache* and

sometimes *pain* in the *loins* and *limbs* and *aching* in the *joints, drowsiness,* and even *stupor*. Rarely there may be *convulsions* and *loss of consciousness*. These initial symptoms rarely last longer than a couple of days, when *paralysis* sets in abruptly, reaching its climax usually in twenty-four hours. The extent varies, involving one or both arms, one or both legs, more rarely one arm and one leg, or there may be crossed paralysis, as of the right arm and left leg. Groups only of muscles may be affected, as those of the upper or lower arm, upper or lower leg, corresponding to the nerve cells involved. More rarely the paralysis comes on more slowly, taking from three to five days to develop. It commonly also diminishes in extent after its first invasion, in rare cases even totally disappearing, but usually withdraws itself to a definite set of muscles, which remain more or less permanently affected. In the legs the tibialis anticus and extensor groups of muscles are more commonly affected than the glutei and the hamstrings, in the arm the deltoid. The child in the meantime has regained its health in other respects.

At first the affected limbs remain natural, and hence the child, too, appears vigorous, especially as the face is not involved. This may be the case for some time, but usually in a few weeks *atrophy* sets in and progresses rapidly, producing a soft, flaccid, wasted limb. The changes in the electrical reaction of the nerve and muscle appear at the same time, commonly more rapidly than the visible atrophy. Usually as early as the first week the reaction of degeneration is present. Faradic excitability of both nerve and muscle is gone. To galvanism there is at first an increased excitability in the muscles, with a predominance of slow anodic contraction (An Cl C), while after two or three months even galvanic excitability falls decidedly, while the muscular contraction maintains its characteristic quality of slow vermicular motion.

Sometimes even the growth of bone of the affected limb is delayed and even arrested and a stunted development results. After a time there also ensues relaxation of the joints and deformity from secondary muscular contraction. In the legs the paralytic club foot (talipes varo-equinus) develops, resulting from the paralysis of peroneal muscles and the tibialis anticus, which permits the point of the foot to drop, while a contracture develops in the antagonistic muscles of the calf. In paralysis of muscles of the calf there results, on the other hand, moderate deformity of the calcaneum from contraction of the antagonistic muscles. In the arms and vertebral column, in paralysis of the spinal muscles, numerous contractures and deformities arise varying in degree, mainly due to the contracture of unparalyzed antagonistic muscles and to external mechanical conditions such as weight and pressure.

Sensation remains intact, as also do, fortunately, bladder and rectum control. At the onset micturition is sometimes a little deranged, but subsequently this disappears. The tendon and cutaneous reflexes, and almost always the skin reflexes, are lost. The skin sometimes exhibits trophic disturbances, being cool and cyanotic.

Diagnosis.—This is usually easy. There are few diseases in which one can reason so soundly from characteristic symptoms to morbid states causing them. The paralysis of one or more limbs, the flaccidity, the

rapid wasting, the reaction of degeneration, and the absence of reflexes, with integrity of sensibility and undisturbed mental state, point only to disease of anterior cornua. The pseudo-paresis of rickets presents some similarity in paretic and muscle phenomena. There is not, however, in rickets true paralysis,—simply pain on motion, to which are added the peculiar head-sweating and hyperæsthesia, together with rachitic symptoms elsewhere.

Prognosis.—This is always unfavorable so far as recovery is concerned, but improvement, at first general, and afterward in groups of muscles, is often decided, so much so, indeed, as to be sometimes delusive. In protracted cases we must expect the superaddition of contractures, while deformities must be pictured to parents as possible. The initial period passed, there is no danger to life, and subjects may live to old age.

Treatment.—Notwithstanding the unfavorable prognosis, treatment should not be ignored. Paralysis is established, of course, before the diagnosis is made, and atrophy nearly as soon. The early symptoms can, therefore, only be treated symptomatically. An aperient should be given and febrifuges ordered. Should an opportune circumstance favor an early diagnosis, cold applications should be made to the spine. Paralysis supervening, the little patient must immediately be put at rest in bed and wrapped in cotton. No active measures should be taken at this stage.

The acute stage passed, electricity is the most important therapeutic measure to be employed. It is used both for curative purposes and to keep up the nutrition of the muscles. In attaining the former, galvanism is preferred, a broad electrode being placed on the vertebral column over the spot supposed to be diseased—on the cervical region, if the upper extremity is paralyzed, and over the lumbar if the lower, while the other pole is placed over the paralyzed muscles and nerves. The latter is moved about, the current being at times reversed. Interruptions may also be made. While the galvanic current is commonly employed, faradization may be used.

The second purpose of the electrical treatment, keeping up the nutrition of the muscles, is more likely to be effectual. For this purpose massage and baths, after the method laid down under treatment of myelitis, are also useful. The electrical treatment must be persisted in for months and even years in order that the muscles may be in a condition to resume their function should the integrity of the cord be restored. Both massage and electrical treatment may be carried out by members of the family after a little instruction.

Tonics are employed for the usual purposes and orthopædic appliances may be necessary to overcome the effect of muscular relaxation on the one hand and of contractures on the other.

ACUTE POLIOMYELITIS IN ADULTS.

SYNONYM.—*Acute Atrophic Spinal Paralysis of Adults.*

The existence in adults of a disease with all the clinical manifestations of the one just described as comparatively frequent in children must be admitted, since an undoubted anatomical lesion of the same kind has been found by F. S. Schultz associated with such manifestations. On the other hand, it must be conceded that the disease is very rare and that many of the cases so diagnosticated were really cases of multiple neuritis. In view of the fact that symptomatology is almost the same as that of the infantile form, no separate description is necessary. Among points of difference may be mentioned the possible involvement of all four extremities or a paraplegia; that in paralysis of the crural region the sartorius muscle often remains free while the peronei and extensor digitorum on the other hand may be separately affected; that in the forearm the supinator longus supplied by the radial nerve may remain free while all the other muscles on the extensor side of the forearm are paralyzed, furnishing "the forearm type" of E. Remak; and that the supinators may be paralyzed alone or together with the biceps, brachialis anticus, and deltoid, furnishing the upper arm type of Remak. The latter form is said to correspond to a lesion at the level of the 4th and 5th cervical roots, and the former to a lesion of the 8th cervical and 1st dorsal roots.

Diagnosis, prognosis, and treatment are much the same as in the same affection of children, except that the prognosis is rather more favorable, recovery being reported, though such cases may have been multiple neuritis.

SUBACUTE AND CHRONIC POLIOMYELITIS.

SYNONYMS.—*Subacute and Atrophic Spinal Paralysis; Paralysie Générale Spinale Anterieure Subœgue of Duchenne.*

All that was said of the probable confounding of acute poliomyelitis of adults with acute multiple neuritis may be said of the subacute and chronic form. Yet it would seem an undoubted case was studied by Oppenheim, in which the anterior cornua of the cord were found markedly diseased at necropsy. There were, clinically, paralysis and atrophy of all four extremities without sensory disturbance. The cases differed from the acute form in the absence of the severe initial symptoms, fever, headache, somnolence, delirium, and vomiting.

ACUTE ASCENDING SPINAL PARALYSIS.

SYNONYM.—*Landry's Paralysis.*

Historical and Definition.—A disease first described by Landry in 1859, characterized by an advancing paralysis, beginning in the lower extremities, passing upward to the trunk and arms, and finally to muscles supplied from the medulla, including those of respiration, sensibility and bladder and rectum control remaining intact.

Etiology and Pathology.—It is most common in men between twenty and thirty years old, and usually those who are strong and healthy. Cases have, however, been seen in children and old persons. No separate anatomical lesions have, however, been shown to be associated with it. Hence attempts have been made to classify it elsewhere, and James Ross, Neuwerk, and Barth regard it as a form of peripheral neuritis, the first having found an interstitial form confined to nerve roots, and the second and third a case confined to peripheral nerves. Other carefully studied cases failed to furnish such lesion. A toxic cause seems not unlikely. It is quite consistent with such cause that it should leave no local lesion as well as that it should always seek the same spot. Gowers is especially disposed to ascribe the disease to such cause. Some cases have, however, followed trauma.

Symptoms.—The characteristic symptoms are commonly preceded by a prodrome, in which *loss of appetite, general malaise, moderate fever, headache, backache*, and *tingling in the extremities* are conspicuous. These symptoms vary in severity and last from a few days to several weeks, when suddenly a *paresis* sets in, first of one leg and then of another, increasing rapidly, so that in a few days, sometimes in a few hours, an almost total motor paraplegia is developed. The paresis next extends to the trunk; in a few days or even less the arms are paralyzed. The muscles of the neck are next involved, and finally those of respiration, deglutition, and articulation, producing bulbar symptoms. Finally, facial paralysis and other disturbance of facial muscles ensue. The paralysis is a flaccid one and there is no tendency to spasm or resistance to passive motion. There is not usually a change in electrical reaction, although there is sometimes a rapid loss of faradic muscular excitability. The reflexes are diminished or absent, but the muscles do not waste.

There is no definite loss of sensation, but in addition to the primary tingling referred to there is sometimes hyperæsthesia and muscular tenderness. In other characteristic cases sensation is intact. More rarely there is a blunted and delayed sensation. The special senses are not affected, nor are the bladder and rectum. Sometimes there are vaso-motor œdema and sweating. The spleen has been found enlarged and slight albuminuria present.

Diagnosis.—This is not always easy, the disease being stimulated by multiple neuritis, acute atrophic paralysis (anterior poliomyelitis), and ascending myelitis. The first for the most part alone gives difficulty, and sometimes diagnosis is impossible. The rapid motor paralysis advancing from below, in the hands and feet, instead of from above, the absence of anæsthesia, of wasting, and of electrical changes, are more characteristic of Landry's paralysis. It differs, too, from acute central myelitis in the less serious derangement of cord functions.

Prognosis.—This is grave, and the possibility of a rapidly fatal termination, even in a couple of days, is to be remembered, the danger being from interference with the cardiac and respiratory functions of the medulla. Other cases terminate similarly in three or four weeks. If, on the other hand, the acute stage passes off, the symptoms of paralysis may cease to extend, and recovery is possible.

Treatment.—The patient should be immediately put to bed, and counter-irritation applied to the back by dry cups, and maintained by gentler means, as a mustard plaster. The thermo-cautery has been recommended, but is of doubtful value. Paquelin's cautery is, however, harmless.

Of internal remedies the apparent results from ergotin and mercury justify their further use. Gowers relates a remarkable case of recovery under the use of the former drug, 20 grains (1.32 gm.) having been given in the course of a night in divided hourly doses. Likewise cases of syphilitic origin have been reported, in which the iodide of mercury has seemingly proved of service. The biniodide may be given in doses of $\frac{1}{30}$ to $\frac{1}{50}$ grain (0.003 to 0.006 gm.). The salicylates have been recommended. Both remedies are indicated from the view of its toxic origin. Perchloride of iron is recommended in traumatic cases, especially where there is evidence of septic poisoning.

If swallowing is difficult the patient must be nourished by the rectum or through the nasal tube, and if symptoms of respiratory failure come on, electrical stimulation of the phrenic nerve and respiratory muscles may be used. If the acute symptoms pass away and paralysis persists, the usual application of galvanism and faradization may be made for restoring muscular and nervous power.

Chronic Affections of the Spinal Cord.

SPASTIC SPINAL PARALYSIS.

Synonyms.—*Primary Lateral Sclerosis; Spasmodic tabes dorsalis.*

Historical and Definition.—In 1875 Erb, and independently Charcot, called attention to a form of paralysis characterized "by a gradually increasing paresis and paralysis, usually advancing from below upward, with muscular tension, reflex contractions and contractures, with marked increase of the tendon reflexes and complete absence of sensory and trophic disturbances, of vesical and sexual weakness, and of any cerebral disturbance." Both observers agree on a "primary symmetrical sclerosis of the lateral columns" as the anatomical condition of the disease. Since then numerous cases have been observed corresponding in clinical features, but while the anatomical features described as essential have been found, they have, with one or two exceptions, been accompanied by other lesions. On the other hand, the symptoms have been found associated with quite different lesions. A single case of true primary lateral sclerosis seems to have been described by Dreschfeld in 1881, another by Minkowsky more lately with involvement of the cerebellar and pyramidal tracts in the lateral columns.

Symptoms.—Owing to the absence of uniformity in the lesions responsible for the clinical picture, I will first describe such a picture in its typical character and then the morbid states which have been found more or less constantly associated with it.

The two essential and predominating symptoms are increase of the tendon reflexes and motor paresis. The first is the most unmistakable, constant, and characteristic. In decided degrees of this increase the contractions come on even with that amount of tension on the tendons which is produced by the weight of the limbs or any active or passive movements, while the reflex muscular tension or rigidity opposes any attempt at motion. The muscles feel rigid and firm and the legs are found in almost permanent extension, while the feet are in plantar flexion. Any attempt, especially if sudden, to flex the leg at the knee or the foot dorsally meets with resistance. Yet if slow effort is made flexion may generally be accomplished, the leg remaining while undisturbed in the position assumed, whence the graphic term of S. Weir Mitchell of "lead-pipe contraction." If the thigh be placed over the edge of the bed the traction of the leg on the quadriceps extensor may be sufficient to excite vigorous extensor tetanus and a convulsive tremor of the whole leg, like that of ankle clonus. If the patient is examined in a bath the spasms are less, because the effect of the weight of the legs is diminished. The superficial reflexes are also increased.

Walking is interfered with in two ways, first by the stiffness in the legs and second by the paresis. The legs are only partially, if at all, flexed at the knee and the foot is not raised, but is pushed along the floor in short, difficult steps. Owing to the contraction of the calf muscles the toes are brought to the ground, and thus he walks on his toes; sometimes an ankle clonus is developed by contact of the toes with the ground. The legs are kept close together, the knees touch, and in certain cases adductor spasm may cause cross-legged progression. Stiffness is not always so marked. The effect is the so-called *spastic gait.* In some cases there is no paresis and the peculiarity of the gait depends purely on the muscular spasm. The effect is what Strümpell calls *pseudo-paresis*, or spastic pseudo-paralysis. The absence of actual paresis is shown by the fact that notwithstanding the stiffness in the gait the patient can still walk some distance, even miles.

To these symptoms is added weariness or stiffness, worse in the morning. Yet with all this the patient is well nourished and the muscles may even be hypertrophied, and outside of these symptoms he may enjoy excellent health. Ocular symptoms are rare. The tendency is to grow gradually worse, but very gradually; finally the patient cannot walk at all, nor can he stand. Rarely the muscles of the trunk and arms become involved, presenting also a paresis with decided increase in the tendon reflexes without disturbance of sensibility or muscular atrophy. Such is the picture of primary spastic palsy, rarely, perhaps, seen in an uncomplicated form.

As intimated, the symptoms are sensory as well as motor. The onset is sudden, with numbness in the extremities, progressive loss of strength, and emaciation. Spastic symptoms, with increased knee jerk, appear, followed by gradually developing paraplegia. The arms are affected, but less than the legs. The course of the disease is rapid, and mental symptoms similar to those of dementia paralytica may be present at the close. The disease resembles multiple neuritis, in which, however, the knee jerk is unaltered or diminished.

Etiology.—The etiology is not always apparent, although syphilis is said to be a cause. The cases are chiefly among the middle-aged. Resembling amyotrophic lateral sclerosis in some of its symptoms, it differs in the absence of muscular atrophy.

The causal conditions are neuropathic inheritance, possibly, according to Putnam, lead and arsenic, but in many cases they are unknown. Seven out of 19 cases collected by Putnam were in women more or less enfeebled and anæmic, in age from forty-five to sixty-four.

Morbid Anatomical Conditions under which it Occurs.—(1) Every kind of transverse lesion of the spinal cord above the lumbar enlargement, whether myelitis, slow compression as in caries or by a tumor, meningo-myelitis, or hydromyelus. All of these causes have produced paralysis of the legs with greatly increased tendon reflexes without sensory disturbance.

(2) Multiple sclerosis. This frequently results in such localization of its nodules as to be followed by paresis and spastic symptoms without sensory disturbance. Such was a case as determined by autopsy previously diagnosticated by Charcot as "tabes dorsalis spasmodique."

(3) Acute illness, unverified, however, by autopsy.

(4) Certain cases of ataxic paraplegia, described by Strümpell and Gowers, characterized by a combination of ataxia and spastic paraplegia, and by anatomical lesions affecting the posterior and lateral columns; or, as more specifically stated by Strümpell, "combined systemic disease of the pyramidal tract, the lateral cerebral tract, and the columns of Goll (posterior median columns) in adults." In these there is gradually increasing paralysis of the arms and legs, with weakness and increased tendon reflexes, spastic symptoms, and sensibility almost normal. Later there is vesical disturbance, probably due to disease of the columns of Goll. While the occurrence of true ataxia of the legs is not unusual, lancinating pains, marked disturbance of sensibility, and pupillary changes are always absent, in which respects it differs from tabes dorsalis.

It occurs most frequently between the ages of twenty and forty, in women about as often as men, and follows syphilis in a way to suggest a causal relation. Spinal injury, such as concussion, often preceding by two or three years the disease, has been assigned as a cause, as has also exposure to wet, cold, and rarely acute illness.

(5) The spastic paraplegia of infants—paraplegia cerebralis spastica—presents also the symptoms described as characteristic; but as this affection is generally conceded to be cerebral in origin, its further consideration is deferred.

(6) Hysterical spastic paraplegia presents in the most striking manner the symptoms detailed. Every symptom mentioned may repeat itself more or less identically. It is, therefore, not necessary to repeat them. Moderate wasting is sometimes added. It occurs, with very rare exceptions, in women only.

(7) Primary combined sclerosis, a condition studied by J. J. Putnam and Charles L. Dana, presents the motor symptoms characteristic of spastic paraplegia, but in addition also sensory symptoms, owing to the invasion of the posterior columns as well as the anterior by a relatively chronic sclerosis, including especially the pyramidal and cerebellar tracts.

To these are commonly added more acute conditions in adjacent areas, diffuse or systemic, some degree of degeneration of the grey matter, and disease of the posterior roots.

Diagnosis.—The symptomatic diagnosis is easy with attention to the facts above mentioned, but caution is demanded in inferring a causal anatomical condition, the result of autopsies failing to find a constant undeviating lesion.

Prognosis.—Spastic paraplegia of all forms except the hysterical is of long duration with little prospect of recovery, although marked improvement is reported and some cases of recovery. The upper extremities are tolerably free from derangement, the patient does not suffer much pain, while the mind is mostly clear. Hysterical spastic paraplegia almost always ends in recovery if properly managed. Where the cause is transient, also, recovery may be expected with removal of pressure, as in caries.

Treatment.—If caries is present, mechanical measures should be used to remove pressure. If syphilis is suspected, treatment by iodides and mercurials should be persevered in. Mercurial inunction is the most ready way of bringing about mercurialism. Galvanism and faradization are less useful in spastic conditions of the muscle than in those in which nutritional changes are more decided, but in hysterical spastic disease they are of signal use for their normal effect. The electrical brush is here the most useful instrument. It should be associated with massage and passive motion and early attempt at locomotion should be encouraged and a positively favorable prognosis made.

In any case friction, massage, and forcible flexion may be expected to be useful as tending to defer the immovable stage. The prolonged effect of the warm bath at 90° to 95° F. (32.2° to 35° C.) is often an amelioration of the spastic symptoms. The bath should be kept up for half an hour and manipulation practised during it. Ergot and nitrate of silver are prescribed.

LOCOMOTOR ATAXIA.

SYNONYMS.—*Tabes dorsalis; Posterior Spinal Sclerosis; Grey Degeneration of the Spinal Cord; Duchenne's Disease; Peripheral Neuro-Tabes; Consumption of the Spinal Cord.*

Definition.—A disease especially characterized clinically by loss of coördinating power, by sensory and trophic symptoms; anatomically by degeneration of the posterior columns of the cord, by foci of degeneration in the basal ganglia and sometimes in the cortex of the brain.

Historical.—Incoördination of movement in cases of spinal-cord disease was noticed in the first third of this century, but the cases in which it was present were not separated from those with loss of power. The association of this distinctive symptom with disease of the posterior columns was first announced by Stanley in 1840, and the first accurate account of the disease was published by R. Bentley Todd in 1847. He

distinguished incoördination without weakness from paraplegia, inferred involvement of the posterior columns, and confirmed his inference by autopsy. In 1851 Romberg described the disease and the lesion in the posterior columns, but failed to eliminate loss of power from the symptoms. In 1855 Russel Reynolds accurately described the symptoms and ascribed ataxia to muscular anæthesia, giving thus the first true explanation of this symptom. Turck first recognized with the microscope the wasting of the fibres in the posterior columns of the cord. In 1858–9 Duchenne, without adding anything essential to the symptomatology or pathology of the disease, published a monograph which so attracted attention that the disease has come to be called by his name, although, as Gowers correctly says, if the name of any man should be associated with locomotor ataxia, it is that of Todd.

Etiology.—The etiology of tabes dorsalis is not a satisfactory chapter. The disease is more common in cities, affects ten men to one woman, is rare in the negro, and is preëminently a disease of middle life, about one-half the cases beginning between thirty and forty, one-fourth between forty and fifty, and less than one-fourth between twenty and thirty. It has been met with as early as ten and younger, and as late as sixty-six. Direct inheritance, independent of inherited syphilis, is almost unknown, but a slight tendency is found in neurotic families.

Of the direct causes syphilis is the most frequent. From 50 to 90 per cent. of cases have been ascribed to it by different authors, Erb and Strümpell leading with the latter figure. Yet there are difficulties in tracing the relation, growing out of the fact, first, that the pathological product is not anatomically a syphilitic one, and second, that it does not respond to the treatment of syphilis. A reasonable explanation ascribes it to a toxic cause analogous to that of the paralysis which follows diphtheria, acting especially on the centripetal sensory fibres.

Prolonged exposure to cold and wet, such as belongs to certain occupations, as lumbering, is a commonly admitted cause, but simple over-exertion, physical and mental, especially sexual excesses, formerly held responsible, are not acknowledged as causes by modern authorities. Alcoholism is also held responsible less commonly than formerly. On the other hand, Tuczek has shown that in chronic ergot poisoning, symptoms like those of tabes are developed, and with them a lesion in the posterior columns of the cord.

Traumatism affecting the spine, and especially concussion, such as is caused by falling from a height, seems to be an undoubted cause in a few instances. In some cases it succeeds a primary paraplegia, while in others it is preceded by the more trifling effect of such accident.

In this connection should be mentioned secondary tabes, especially emphasized by Gowers as succeeding other diseases of the spinal cord, especially in syphilitic subjects. Myelitis and syphilitic gummata are named as such.

Morbid Anatomy.—Tabes dorsalis is preëminently a systemic disease, certain systems of fibres being always affected, fibres associated from an anatomical and physiological standpoint. Since, however, fibres with different functions are also diseased, as evidenced by the sympto-

matology, it is not a simple, but a combined systemic disease, including at the same time cerebral nerves and symmetrical parts of the spinal cord with their corresponding spinal nerves.

Directing our attention to the spinal cord, in which are found the most manifest changes, we find that at times, even when enclosed in the membranes, its smallness and thinness are noticeable, while through the pia we may see the posterior columns distinct as a grey band throughout the length of the cord. The pia is, however, commonly thickened and opaque, especially on the posterior surface, sometimes more firmly adherent than is natural, while the blood-vessels also show signs of arterial sclerosis. The contraction of the posterior columns is more conspicuous on section. They are flattened instead of convex, while the grey translucent appearance is also evident, being due to the fact that the nerve fibres have been substituted by connective tissue. Hence, also, the name, "grey degeneration." In the hardened cord the difference in hue is even more striking than in the fresh state. The posterior nerve roots share the atrophic process and are small and grey.

On minute examination, in transverse sections stained by carmine or other staining fluid, the affected areas are more conspicuous, because of

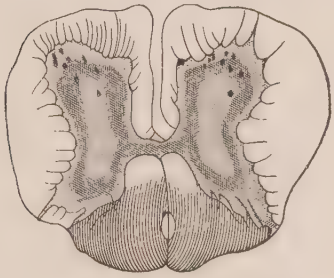


FIG. 71.—TRANSVERSE SECTION THROUGH THE LUMBAR REGION IN LOCOMOTOR ATAXIA. The diseased portions of the posterior columns are shaded.—(*After Strümpell.*)

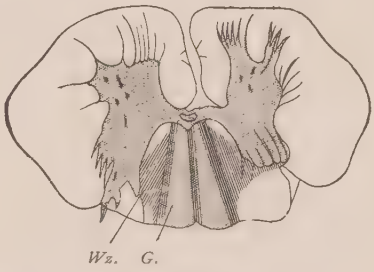


FIG. 72.—TRANSVERSE SECTION THROUGH THE CERVICAL REGION IN LOCOMOTOR ATAXIA. *G.* Columns of Goll. *Wz.* Root zones.—(*After Strümpell.*)

the deeper staining of the sclerosed tissue, while all parts of the posterior columns are not equally affected. In the lumbar cord, which is at once the most frequently and intensely involved, the form in which the legs only are affected, the change affects chiefly the middle and posterior parts of the columns, while the anterior portion remains intact. The sclerosis is commonly most intense in the part adjacent to the posterior cornua into which the posterior roots run, also near the surface of the cord. Ascending into the dorsal cord, the intensity of the disease gradually lessens in the external parts of the posterior columns and becomes greater in their median portion. It presents, also, the distribution of an ascending degeneration, which, in fact, it is, receding from the commissure in the upper cervical region.

In the cervical cord, the so-called columns of Goll are chiefly affected, together with the fibres prolonged from the root zone of the

lumbar cord, and also the lateral root zones, that is, those portions of the columns of Burdach where fibres enter directly from the posterior nerve roots, and from which fibres may be traced further into the grey matter of the posterior cornua; but the so-called posterior external areas and also two little antero-lateral areas remain free from disease, at least for a long time. Figure 73 shows how the first beginnings of the disease are localized in the posterior columns. In the earlier stages the "posterior root-zone" of Charcot is first involved, often at the same time with the fibres of the posterior root, so that it has been thought to begin as a neuritis of these roots within the vertebral canal.

It is in consequence of this involvement of the posterior roots that the corresponding cornua into which they enter are also affected. The same is true of the medullated fibres of Clarke's columns, which are also direct processes of the posterior roots, while the cells of the columns remain normal. Lissauer's * tract, a narrow strip between the pyramidal tract and the posterior cornu, is early involved. Finally, a single observation of Charcot and Joffroy goes to show that the anterior horn is the true seat of the central lesion upon which the spinal arthropathies of this disease depends.

In advanced cases, in the larger peripheral nerve trunks, such as the sciatic, and in the finer branches of the sensory nerves, many de-

Dorsal Region. Lumbar Region.

Fig. 73.—Transverse Section Through the Posterior Columns of the Cord in Beginning Locomotor Ataxia.—(*After Strümpell.*)

generated fibres can be recognized. Some of these atrophies may be secondary, but modern clinicians are disposed to regard the peripheral degenerations of tabes as independent and primary, especially as, in addition to these, decided degenerative processes sometimes occur in the trunks of certain cranial nerves, such as the optic and oculo-motor, and more rarely the vagus and auditory. They will be alluded to in treating the diseases of special nerves.

Finally, there are even cerebral changes, consisting in sclerosis of the restiform bodies, of the inferior peduncles of the cerebellum, of the cranial nerves alluded to, and cortical changes constituting a diffuse meningo-encephalitis.

Symptoms.—The characteristic symptoms of locomotor ataxia are easily divisible into three sets, *motor*, *sensory*, and *reflex*. In addition to these there are others not essential, but striking, including modifications

* A narrow strip of fine fibres from the posterior root, which pass upward for a short distance and then enter the posterior horn.

of special sense function, especially that of the eyes, and certain visceral symptoms chiefly characterized by pain and known as "crises," of which the gastric is the most striking.

The *motor phenomena* are the most prominent, whence, indeed, the disease takes the name adopted. The distinctive symptom is a *loss of coördinating power* in the legs, having its simplest illustration in the unsteady gait of the drunken man. It is intensified when the patient attempts to walk with his eyes closed, that is, when the guiding sense is removed, and, indeed, in its early development does not appear except when the eyes are closed. It is unaccompanied by a loss of power or muscular wasting. On the other hand, incoördination is by no means always the earliest symptom, and it may, indeed, never be developed. It may also be shown sometimes before otherwise evident by directing the patient to place the heels and toes together and then to close the eyes, when a swaying appears as though the patient were going to fall—Romberg's symptom.* In health a slight unsteadiness under these circumstances is present which varies with different persons. Higher degrees develop the "sway" even when the eyes are open. The symptom often exists for a long time before being recognized by the patient, but sometimes is discovered quite early through an accidental production of the favoring conditions. Soon the gait is noticeable and becomes peculiar. The foot is raised too high, thrust forward too far, and brought down suddenly, with the whole sole flat on the ground at once, with a stamp. The walk is also straddling. Finally, the patient can walk only with the aid of a stick or by keeping the eyes fixed upon the floor. He rises from the sitting posture with difficulty, often after three or four efforts. The loss of coördinating power may also be shown in the recumbent posture when the patient attempts to touch the knee with his heel, when he will carry it around and in front and behind without accomplishing his purpose.

Incoördination also develops in the hands, but much more rarely and late in the disease, though it may appear first in them. It is shown in connection with more delicate acts, such as picking up a pin, buttoning, and writing. It may be demonstrated also by asking the patient to bring the ends of two of his fingers together with his eyes closed, or to touch the end of his nose with one, which he may not be able to do. With all this ataxia the muscular power remains intact. The patient lying in bed can kick out with great force, and resist successfully any effort to flex the extended leg, and the grip of the hand is strong.

The *sensory symptoms* are less distinctive, especially at first. The most frequent of these—indeed, the most frequent of all symptoms—are pains of a darting, shooting, or stabbing character, whence they are called *lightning pains*. They appear in nine-tenths of all the cases. They are very like those of neuralgia, lasting but a second or two. They are most common in the legs, and are often accompanied by burning or tingling, espe-

* This symptom is classed by Strümpell among those of sensibility, which is impaired in the soles of the feet and the muscles, whence follows defective control of muscular movements necessary to equilibrium.

cially in the feet. They may be felt in the trunk, arms, and even head. They do not commonly correspond with nerves or affect joints. *A sensation of cold* is felt also, a feeling as though the limb was in cold water. The pains are induced by fatigue or excesses or temporary ill-health from other causes, and are apt to come on at night. They may last hours or a day or two. There may be areas of *hyperæsthesia* and *anæsthesia*. A very curious sensation is felt in the soles of the feet when walking, a feeling as though *soft carpet or cotton is interposed* between them and the floor. A painful sense of constriction about the limbs or waist or entire trunk—*girdle pains*—is regarded as characteristic.

There are other disturbances of sensation, such as retardation of tactile sensation, wherein the prick of a pin instead of being instantaneously felt is *delayed* for several seconds. Another sensory symptom is a difficulty in localization, manifested, for example, in referring a pin prick to the right foot when it is made on the left—allochiria—or it may be felt in both feet—polyæsthesia. In advanced stages the *muscular sense* is also impaired, and the patient is unable to indicate correctly the position of a limb. There may be other perversions of sensibility. The sense of pain may be lost or perverted; also the temperature sense—that, too, without derangement of the pain-sense or common sensibility. All sensations may be lost in the most diverse parts of the body and most irregularly.

Visceral pains, known as tabetic crises, among which the gastric is the most common, are also among the sensory phenomena. They may be laryngeal, rectal, nephritic, urethral, and are sometimes exceedingly severe. The gastric crises are sometimes accompanied by vomiting of highly acid gastric secretion. The laryngeal crises may be associated with spasm and dyspnœa, with noisy breathing. Death is a possible termination from this cause. Rectal crises consist in paroxysmal pain and tenesmus, with a sensation as of a foreign body in the rectum.

The *reflex symptoms* consist in *impairment in reflexes*, both tendon and cutaneous. The loss of the knee jerk is one of the most frequent and early of these, occurring sometimes years before ataxia appears. Of itself it is not diagnostic, as it may be absent in healthy persons, but in association with lightning pains and ocular symptoms it is almost conclusive evidence of the disease. The skin reflexes fail *pari passu* with the loss of tactile sensibility, and it is doubtful whether they are ever present without this. The plantar skin reflex is that most frequently impaired, and after this are successively involved the gluteal, cremasteric, and abdominal. It rarely happens that in the early stages of the disease the skin reflex is increased, sometimes considerably, but even then the knee jerk continues absent or diminished.

Of the remaining symptoms the *ocular* are the most important. They include *ptosis* of one or both eyelids, producing a very striking appearance. It may be alone or associated with *external strabismus* and *double vision*. Rarely there may be paralysis of all the external muscles of the eye, producing *ophthalmoplegia externa*.

The most remarkable eye symptom is, however, the *Argyll-Robertson pupil*, in which there is loss of reflex contraction of the iris in response to light, while the contraction of accommodation remains. According to

Gowers, the loss of this reflex occurs in five-sixths of all cases. The contraction on accommodation is, however, not always maintained. Very rarely the reverse of the Argyll-Robertson pupil exists. Often the dilatation of the pupil which takes place in health when the skin of the neck is pinched cannot be produced, and coincident with this is often an unnatural smallness of the pupil—spinal myosis.

Finally, there is sometimes *atrophy of the optic nerve.* When it occurs it is an early symptom, usually commencing before incoördination appears, and what is more singular, the ataxia often does not supervene, that is, there seems a tendency for the spinal malady to become stationary when the optic nerve suffers. The failure of vision usually begins with peripheral limitation and progresses slowly to total blindness, sometimes to a considerable extent before the patient notices it. Occasionally it ceases, and there may be even slight impairment. *Hemianopia* may occur from disease at the optic chasm.

Deafness may be present from disease of the auditory nerve; also more rarely *anosmia*, from atrophy of the olfactory. Attacks of *vertigo* are common in these cases. Abnormalities in function of other cranial nerves may be due to similar involvement. Among these may be mentioned pain at one time and anæsthesia at another in the area of the 5th nerve; also unilateral atrophy of the tongue.

There may be delayed micturition from weakness of the detrusor muscle of the bladder, or incontinence from paralysis of its sphincter, with partial evacuation of the bladder, and cystitis. The anal sphincter is less frequently affected.

Vaso-motor and trophic phenomena also occur late in the disease, but may be predominating symptoms. They include local sweating of the palms and soles, or of half the head, œdema, skin ecchymoses, herpes, and modified hair growth, loss of pigment from hair and skin, thickening of the epidermis of the sole, succeeded by blisters under it. Alteration in the nails and onychia with ulceration may occur; also decay of the teeth and the so-called perforating ulcer of the foot, which is almost peculiar to the disease. Only late in the disease may atrophy of muscles, sometimes associated with neuritis or involvement of the anterior cornua, occur. Paroxysmal diarrhœa occurs and has been regarded as vaso-motor in origin.

The so-called *arthropathies* are an interesting trophic symptom which are directly the result of the disease. The most common is that known as Charcot's joint, anatomically similar to chronic affections in which the disease begins in the bone as contrasted with the synovial membrane, resulting in atrophy and in the destruction of bone and cartilage, while brittleness of bones, attended with spontaneous fracture or luxation, may occur. If union takes place there is a superabundance of callus, with ossification or calcification of adjacent structures and in any new-formed inflammatory tissue. The large joints are those commonly affected and are painless. There may be effusion and even pus in the joints. The arthropathies may even occur in the preataxic stage. They may be excited by injury. The joints may also become much relaxed, while changes in the tarsal bones and articulations may cause the foot to become

flat, with projection backward or inward of the tarso-metatarsal articulations and of the tarsal bones, producing the "tabetic club-foot."

Cerebral symptoms also occur, but are rare. They include hemiplegia, usually occurring late in the disease, and due to hemorrhagic softening or thrombosis, or syphilitic cortical changes; hemianæsthesia, convulsions, melancholia, dementia, and dementia paralytica. It is not always easy to decide whether the dementia or the tabes is primary. The final stage of the disease, in which the patient is bed-ridden, is spoken of as the paralytic stage.

Diagnosis.—The diagnosis, commonly easy when the characteristic symptoms are developed, may demand critical judgment in the early stage. The combined presence of lightning pains, absence of knee jerk, early ocular palsies, including the Argyll-Robertson pupil, ptosis or squint, and ataxia are conclusive. Lightning pains and ocular palsies should always stimulate to thorough examination. The same is true of severe attacks of gastralgia in middle-aged men.

Differential Diagnosis.—Disease of the vertebral column with resulting compression of the spinal nerves is also associated with lancinating pain and absence of the patellar reflex, but the later symptoms are widely different. The same is true of deep-seated tumors impinging on the spinal cord.

Peripheral alcoholic and arsenical neuritis are also associated with diminished knee jerk, a pseudo-tabetic gait, and sharp pains, but the gait differs from the true tabetic gait, the leg being lifted high in order that the toes may clear the floor. The pains also follow the course of the nerves, which are tender on pressure, and there is none of the shooting character. Nor is there reflex immobility of the pupils or bladder disturbance, while atrophic paralysis, always absent in tabes, also develops. Multiple sclerosis in rare instances presents similar symptoms, but defective speech, nystagmus, mental weakness, and ultimate apoplectiform seizures serve to distinguish it. In diphtheritic palsy and ocular palsies there is absence of knee jerk, but the history of the case, the throat palsy, and all absence of pain are distinctive. Ataxic paraplegia has also ataxia, but here again eye symptoms and pain are absent. In cerebellar disease there is also loss of coördination, but the knee jerk is present, there may be headache, optic neuritis, and vomiting, but no lightning pains or sensory disturbance.

General paresis and tabes sometimes merge, the latter developing on the former and the former on the latter toward the end. Rapidly developed ataxia with mental symptoms often resolves itself into general paresis. Yet acute involvement of the posterior columns is also possible, producing ataxia. Osler refers to such a case developing in three or four months, during which there was the characteristic gait and Romberg's symptom—swaying; but the knee jerk was retained and there were no ocular symptoms. There was a syphilitic history, and under large doses of iodide of potassium the symptoms disappeared.

Finally, there is the *nicotine* tabes of Strümpell, who has twice met with in men long working in tobacco factories a set of symptoms consisting in painful sensation, absence of patellar reflex, contracted pupil,

with reflex immobility and uncertain gait, differing, however, from tabes in the presence of tremor and marked increase in the skin reflexes, especially in the lower extremities.

Course and Prognosis.—It is generally conceded that no case of thoroughly developed tabes has ever recovered. It may, however, be arrested. This especially happens after optic-nerve atrophy has set in, after which ataxia rarely develops further, while the other symptoms subside and the optic atrophy itself may be checked. In most cases of the disease, however, the advance is slow but irresistible. The duration of the first stage, characterized by absence of knee jerk, by the presence of the Argyll-Robertson pupil, and lancinating pains, lasts from a few months to twenty years. The second stage—that of ataxia—from which, indeed, the patient often dates the disease if the initial symptoms were slight, may then supervene gradually or suddenly. Finally, the paralytic stage intervenes, to be soon followed by death.

Treatment.—While recovery from locomotor ataxia probably never occurs, much may be accomplished by treatment in arresting progress and relieving symptoms. There is no specific treatment, although this effect has been claimed for more than one remedy. Nitrate of silver, first recommended by Wunderlich, has probably had most reputation, but has latterly fallen into comparative disuse, and with it the number of cases of chronic argyria. The dose administered is $\frac{1}{6}$ to $\frac{1}{4}$ grain (0.011 to 0.0165 gm.) three times a day. A proper question as to the remedy is the length of time it may be used without danger of producing this unfortunate result. Prof. E. Harnock asserts that in no recorded case of argyria were less than 30 grams (450 grains) of the salt taken before the discoloration appeared. To consume this much in quarter-grain doses three times a day would take six hundred days. If, therefore, it is given in the usual doses for a month and then suspended for one week, as commonly directed, it does not seem possible that any unpleasant effect can result. In this manner, then, it may be kept up indefinitely. Or it may be alternated with arsenic, for which Gowers at least claims that it more frequently than any other remedy does distinct good. The favorite form in this country is five minims (0.3 c. c.) of Fowler's solution three times a day for an adult. The œdema below the eyes, which results from its accumulated effect, is a sign that the dose should be reduced or the drug temporarily suspended. Arsenious acid in doses of $\frac{1}{30}$ to $\frac{1}{20}$ grain (0.0022 to 0.0033 gm.) or sodium arsenate in doses of $\frac{1}{30}$ to $\frac{1}{10}$ grain (0.002 to 0.006 gm.) may be substituted. Sometimes a smaller dose only is borne. Gowers has also found the chloride of aluminium useful in doses of two to four grains (0.132 to 0.264 gm.) three and four times a day. The supposed frequent causal relation between syphilis and tabes renders the anti-syphilitic treatment appropriate in all cases in which such relation can be traced. To this end mercurials are to be administered until the specific effect is produced. This is best accomplished by inunction, a drachm to a drachm and a half being rubbed into the arm-pit or inner surface of the thigh daily, to be discontinued when the gums are affected. After this the bichloride may be given in doses of $\frac{1}{24}$ grain (0.0027 gm.) three times a day in association with

the iodide of potassium in ascending doses if well borne. Or the biniodide of mercury may be given in doses of $\frac{1}{24}$ grain (0.0027 gm.) three times a day. If this treatment is found effectual, the iodide should be continued in the minimum doses, which will keep up the effect. Calabar bean in doses of $\frac{1}{10}$ to $\frac{1}{5}$ grain (0.0064 to 0.0128 gm.) three times a day and ergot in doses of five to 30 minims (0.3 to 1.6 c.c.) or more are recommended, but the results have not been such as to give them a permanent reputation. Iodide of potassium may be tried apart from the indications of syphilis. The rest treatment, originally suggested by S. Weir Mitchell, has been found useful in arresting the disease, but I do not know that it has been followed by permanent results. Extension of the spinal column and presumably of the cord by suspension of the body for one to three minutes daily was suggested by Charcot and used for a time, but it has been discontinued.

In Germany electricity is still a popular remedy, and failure with it in this country may be due to imperfect and too brief trial. Erb's directions for galvanism are to place a moderate-sized anode in the vicinity of the sympathetic in the neck, and a large kathode on the side of the vertebral column for four or five minutes, moving it at intervals from above downward. Severe pain and vesical weakness are treated by galvanization and the faradic brush. The latter, as recommended by Rumpf, should be brushed over the skin of the back and extremities for five or ten minutes, using a strong current. Counter-irritation by blisters is of no use, although simple rubefacients may relieve slight degrees of pain.

Hydrotherapy likewise maintains its popularity in Germany, although claimed by some authorities to be sometimes harmful, especially hot and vapor baths and wet packs. The tepid bath is entirely safe and often symptomatically useful. Its temperature should be 80° to 90° F. (26.6° to 32.1° C.), accompanied by gentle rubbing. Wet compresses upon the abdomen or legs at night sometimes relieve the pains. In Germany, too, there are numerous water-cure establishments in the hands of experienced directors, to which patients may be advantageously sent, but, unfortunately, there is nothing of the kind in this country which can be recommended. Oeynhausen-Rehme in Minden has the best reputation for its carbonic acid thermal salt baths, but the baths at Neuheim in Hesse are similar. Mud and iron baths are found at Pyrmont, near Brunswick; Driburg, in Westphalia, Prussia; Elster, pleasantly situated in Saxony, Karlsbad and Franzenbad in Bohemia.

The painful attacks are often not relieved by the measures thus far suggested, and require more powerful measures. The first to be used should be phenacetin, acetanilid, exalgine, and antipyrin, while morphine should be deferred as long as possible. It may, however, be necessary, when it should be used hypodermically. Cocaine used in the same manner in doses of $\frac{1}{6}$ to $\frac{1}{4}$ grain (0.011 to 0.165 gm.) is also sometimes efficient, while cannabis indica in doses of $\frac{1}{4}$ to $\frac{1}{2}$ grain (0.0165 to 0.033 gm.) of the extract may also be tried.

Fatigue of all kinds as well as anxiety of mind should be avoided, while moderate exercise may be encouraged. Excesses in smoking, and especially in the use of alcohol, are harmful, as is also sexual indulgence.

Over-eating and the use of indigestible articles of food should be avoided, as gastric crises are invited by them.

HEREDITARY ATAXIA.

SYNONYMS.—*Hereditary Ataxic Paraplegia; Friedreich's Disease.*

Definition.—The disease depends anatomically upon combined posterior and lateral sclerosis, occurring in families and at an age much earlier than ordinary tabes, from which it differs also in the addition of peculiar symptoms.

Historical and Etiology.—Friedreich reported in 1861 six cases of this disease and a further number in 1870, whence the association of his name with it, but as the name Friedreich's disease is also applied to *paramyoclonus multiplex*, confusion results. On the other hand, the term hereditary ataxia is scarcely correct, because, while sometimes it is hereditary and even congenital, it is not always so. It does, however, always occur in families, several brothers and sisters being, as a rule, affected. Yet isolated cases do occur. In one case of William Osler's three generations were involved. A neurotic tendency is sometimes noted. Alcoholism and syphilis were present in parents in a few instances, while consanguinity of parents only in a very few. Otherwise its etiology is unknown. It is more common in males than in females, affecting 86 males and 57 females out of 143 cases collected by J. P. C. Griffith. Strümpell makes the opposite statement as to sexes, but other observers agree with Griffith. Of Griffith's cases 15 occurred before the age of two, 39 before the age of six, 45 between six and ten, 20 between eleven and fifteen, 18 between sixteen and twenty, and six between twenty and twenty-four.

Morbid Anatomy.—There is decided degeneration of the posterior columns, extending to the lateral and often also the anterior columns, the posterior degeneration being at once the more intense and extensive, especially in the lumbar region, though it may extend throughout the cord, except a narrow band near the neck of the cornu. In the lumbar region it usually extends to the head of the horn and the posterior surface of the cord. The lesions are thus those of tabes and the ataxic paraplegia of Gowers—common involvement of the lateral and posterior columns. As in tabes, the posterior nerve roots are also usually affected. The degeneration of the lateral columns always involves the pyramidal tracts and is most intense in them, but is not limited to them, extending to the surface even when the latter do not, involving thus the cerebellar tract. It sometimes extends forward toward the surface so as to form a band of annular sclerosis, varying in thickness in different parts of the cord. As yet no changes have been found in the cells of the posterior horns. The pia mater over the posterior columns is sometimes thickened.

The disease seems to consist of a double morbid process, consisting in early degeneration of nerve elements, associated with a tendency to overgrowth of interstitial or neurogliar tissue,—a gliosis of the posterior columns, due to defect in development.

Symptoms.—The essential symptoms are *ataxia with paraplegia.* Initial pains are rare. The ataxia is, however, peculiar. As in tabes, it begins in the legs, but it is swaying and irregular, more like that of drunkenness. The feet are not often raised too high, and there is not the stamping as in true tabes. The ataxia of the arms occurs early and is striking, the movements being choreiform, jerky, irregular, and swaying. The hand first moves about an object in its efforts to secure it and then pounces upon it. There seems to be a superabundance of effort in voluntary movements, action is over-done, and prehension is claw-like. Again, the fingers may be spread out or over-extended. The first manifestation of the disease in children is often a tendency to fall.

As the disease advances, irregular, *jerky movements* affect the *head* and shoulders, sometimes tremor-like. In most cases there is *nystagmus* when the eyes are moved laterally or upward, usually a late, sometimes an early, symptom. Atrophy of the optic nerve is rare and the pupils are normal. *Speech* is sometimes impaired, generally as a late symptom —three, five, or ten years after the initial symptoms. Syllables are elided—the speech is scanning—with occasional movements of the tongue, but no twitching of the lips.

The *paresis* is at first slight, indeed, the power of the muscles is at first unimpaired, while there is rarely ever total paralysis. Some patients, however, never walk. The nutrition of the muscles is good, but myotatic irritability is lost. The knee jerk mostly disappears early, or is at least absent when the cases come under observation. In a few this symptom appears late, while in a few atypical cases this reflex has been reported increased. Sensory symptoms are not usually conspicuous. There may be none, even in bad cases. At times there is delayed sensation or impairment of sensibility to pain and temperature. Increased sensitiveness may be present. No visceral crises occur.

While trophic lesions of the usual kind are rare, there occur peculiar deformities, especially of the feet. There is talipes equinus or equinovarus, and the patient walks on the outer edge of the foot. The great toe is flexed. These are probably the results of muscular weakness developing under the influence of position. There may be lateral curvature of the spine.

Diagnosis.—This is not difficult, though sometimes the disease is confounded with chorea, with the hereditary form of which it has certain points in common. The ataxia in early life, the club foot, flexed great toe, spinal curvature, nystagmus, and scanning speech form a complex of symptoms not found in any other disease.

It resembles ataxic paraplegia in more than its symptomatology, but the increased knee jerk, foot clonus, and spasm of the latter disease are wanting. In cases of combined lateral sclerosis and posterior sclerosis where the knee jerk is absent the family history and youth of the subject can alone settle the question. The loss of iris reflex in children points to tabes, the result of inherited syphilis. Disseminated sclerosis presents incoördination, nystagmus, and defective articulation, but there is little or no unsteadiness of the legs, while the ataxia of the arms is more violent and extreme.

Prognosis.—This is invariably bad, although the disease lasts many years.

Treatment.—There is no treatment except such as will overcome tendency to deformity. The remedies used in locomotor ataxia may be tried.

SYRINGO-MYELIA.

Definition.—A term applied to all cavities in the spinal cord, most of which are, however, surrounded by an overgrowth of embryonal tissue, whence they receive a congenital stamp.

Pathology and Morbid Anatomy.—The cavities are formed partly by defective closure of the central spinal canal and partly by the breaking down of the residual embryonal tissue. The cavity of a syringo-myelia is usually in the posterior part of the cord, extending toward the posterior cornua. It may prevail throughout the entire extent of the cord, but in most cases involves only the cervical or dorsal regions or more limited areas. The transverse section is oval or circular, but it may be fissure-like or quadrilateral, even irregular. The embryonal neurogliar tissue about the cavity may be the seat of hemorrhage, followed by degeneration and cavity formation, or the degeneration may take place directly in the gliomatous tissue without intermediate hemorrhage. The cavities may be duplicate or even triplex. Gowers says of the term hydromyelia, applied to the simple forms in which the cavity is merely the dilated central canal, that it is falling into disuse and there is no real difference between this and the other varieties.

Symptoms.—The slighter degrees are without symptoms and are often overlooked. Symptoms usually make their appearance about the period of adolescence. They are mostly gradual in development, the result of the secondary processes of distention, which derange natural function and vary greatly in degree and to a less extent in character. These variations are influenced also by the situation of the cavity, which is found most frequently in the cervical region, whence the arms and neck are mostly frequently affected.

The essential symptoms are *loss of sensibility*, chiefly to *pain*, *temperature*, and to a *less degree* simple *tactile sense;* also muscular *atrophy*, the latter progressive in development. The sensory symptoms are the earlier and more constant. The sense of tactile impression is lost by implication of its path, which, as has been said, is not precisely known after it enters the posterior roots, though it is probably partly in the posterior columns. The comparative rarity of this involvement may be said to be due to the difficulty in destroying this path completely. There may be irregular pain, chiefly in the cervical region. Derangement of the sense of pain and thermal sense is probably due to implication of the central grey matter, since it is through it that these impressions probably radiate to the white conducting tracts of the opposite side. There may not only be a loss of thermal sense, but it may be reversed in that heat is felt as cold, and *vice versa*. So also subjective sensations are felt, including heat and cold, or in their absence of pain, which may be neuralgic in char-

acter. The involvement of all forms of sensibility probably takes place when the postero-internal columns are extensively involved. The muscular atrophy, which may ultimately finish the picture of a typical amyotrophic lateral sclerosis, is the result of injury to the motor cells of the anterior cornua in the compression to which they are subjected in the dilatation. This causes degeneration of the nerves and wasting of the muscles, and along with this is a lowered electrical irritability. There is also *muscular weakness*, involving the trunk muscles, whence results sometimes lateral curvature. If the legs suffer, it is generally from simple spastic paralysis from pressure on the pyramidal tracts. Wasting of the legs indicates lumbar enlargement, and the presence of ataxic symptoms evidences involvement of the posterior columns.

The remaining symptoms are not essential, but may be incidentally present from the action of the causes which usually produce them. The *reflexes* may or may not be increased, and *myotatic irritability* may in rare cases be lost, while *tremor* of the limbs has been noted in some cases.

Trophic symptoms are not rare in the parts affected by sensory loss. The skin may be glossy and thin, or thick and horny, while there may be eczema, herpes, bullæ, even ulceration and gangrene. The nails may become fissured and drop off. *Vaso-motor* disturbances are more common, including coldness, lividity, or redness with swelling and heat. There may be sweating or its absence, brittleness of bone, and joint changes like those of tabes.

The area of the cranial nerves may be invaded where there is involvement of the medulla and pons. The phenomena include paralysis of one vocal cord, the tongue and face, difficulty in swallowing, of breathing, and embarrassed heart's action. The eyes may be disordered, pupils unequal and smaller on the side where most severity of symptoms exists, but the other special senses escape and the sphincters are unaffected.

Diagnosis.—This is based upon the sensory symptoms, and of these, thermo-anæsthesia and analgesia rather than tactile sensibility, together with muscular atrophy succeeding after some interval. Cervical pachymeningitis causes similar symptoms similarly distributed. J. Hendric Lloyd in an important paper * has also called attention to certain traumatic affections of the cervical region of the cord simulating syringomyelia. Cervical pachymeningitis runs a more rapid course; the anæsthesia includes all varieties of sensation and corresponds more nearly in its distribution with that of the muscular atrophy, pain is more conspicuous, and the reaction of degeneration is commonly present in the wasting muscles.

The symptoms of syringo-myelia are sometimes simulated by the anæsthesia and wasting of anæsthetic leprosy, but in the latter disease the trophic changes are more marked, the phalanges often drop off, while the sensory symptoms include all varieties of sensation.

Progressive muscular atrophy differs in the absence of altered sensation. A spinal tumor in the same situation as a syringo-myelia furnishes almost identical symptoms, and may have an identical origin

* Read before the Philadelphia Neurological Society, March 26, 1894.

if it starts from the neuroglia, but the symptoms are more rapid in their development.

Prognosis.—This is ultimately fatal, although the course is slow, extending over a period of fifteen to twenty years. Toward the end the course is more rapid, death resulting from exhaustion, or interference with the functions of the medulla.

Treatment.—This can only consist in measure to combat symptoms and tendencies to them, such as cystitis, bed-sores, and the like.

Morvan's Disease.

Synonyms.—*Analgic Panaritium; Analgesic Paresis with Panaritium; Painless Whitlows.*

Definition.—This term is applied to a chronic affection described in 1883 by a Breton physician named Morvan, which is characterized by neuralgic pains, tactile and thermal anæsthesia, analgesia, and destructive felons (panaritia). The tactile anæsthesia is considered the distinctive symptom between it and syringo-myelia, but Joffroy considers the two diseases identical. An autopsy by Gombault discovered neuritis, whence it has been suggested that the disease is syringo-myelia associated with neuritis, but the condition of the cord in this case did not permit the recognition of dilatation. Twenty cases were recognized in a population of 50,000 in Brittany, whence, too, it has been suggested that it is a peripheral neuritis of infectious origin. One or two cases have been reported in America.

COMPRESSION OF THE SPINAL CORD.

Synonyms.—*Compression Myelitis; Pressure Paralysis of the Spinal Cord.*

Definition.—Under this head are included all forms of paralysis due to gradual compression of the cord from whatever cause.

Etiology.—A large number of causes may operate in the way indicated, among which are tumors or inflammatory new formations, including syphilitic products, either in the membranes or outside of them, caries of the vertebræ, especially the form known as Pott's disease or tuberculosis of the vertebræ, cancer of the vertebræ, echinococci and cysticerci in the vertebral canal. Extra-spinal causes may also produce erosion of the vertebræ and compression of the cord; among these are aneurism of the aorta, retro-peritoneal sarcoma, lymphadenoid growths, and suppurating kidney; also retro-pharyngeal abscess. Pott's disease is by far the most frequent cause.

Morbid Anatomy.—The changes in the cord as the result of compression are best studied in the compressions due to dislocation of the vertebræ owing to the breaking down of the bodies of one or more of them after tubercular infiltration, or as the result of intrusion into the spinal canal of foci of cheesy pus on the posterior surface of the bodies of the vertebræ. Macroscopically, the cord is often smaller, softer, and sometimes bent. In old cases it may be harder. The term myelitis has been applied to the changes thus produced in the cord, but careful

examination fails to find any of the usual histological products of inflammation. On the other hand are noted, rather, remnants of disintegrating nerve tubules—a few fatty granular cells, wandering cells which have taken up the disintegrated nerve medulla—to which may be added sometimes small traumatic hemorrhages. At a later stage may be seen a secondary overgrowth of neuroglia, replacing the destroyed nervous tissue, first loose, later firm and fibrillated. After a certain duration there may be ascending and descending secondary degeneration of certain systems of fibres in the spinal cord.

Symptoms.—Where tubercular disease of the spine is the cause, the resulting deformity—kyphosis—is usually seen long before the symptoms of compression of the cord are present. On the other hand, where the erosion comes from aneurism or growths within the thorax or abdomen, the subjective symptoms appear before the deformity, or more frequently without external deformity. The first of these symptoms is usually *pain* at the seat of the compression, which often does not amount to more than a dull ache, while at another time it increases to extreme severity. It is also aggravated by motion, by bending or straightening the body. Again, the pain is distributed along the course of the nerves, when the compression is exerted on the nerve roots. Such pain radiates in every direction followed by the nerves themselves in the upper and lower extremities or over the trunk. Previous to such pain and associated with it are *paræsthesias* of various kinds, such as numbness, tingling, formication. More rarely there is *impaired sensibility*, the same degree of pressure which deranges the function of motor fibres having often no effect on the sensory. Marked anæsthesia is rare, and then only in the last stages.

With these soon become associated motor symptoms, which may consist in *stiffness*, giving rise to difficulty in moving arms or legs, with peculiarity of gait, or there may be simple weakness or *paresis*, increasing to complete motor *paralysis*, rarely, however, affecting both arms or legs at once, but rather first one, then the other.

The seat of the more pronounced sensory and motor symptoms varies with the segment compressed. Thus when the caries is in the upper cervical region, between the axis and the atlas, or between the latter and the occipital bone, there may be *spasm of the cervical muscles*, the head may be fixed, and movements may either be impossible or extremely painful. Retro-pharyngeal abscess may be the cause of such symptom, as in a case in the Montreal General Hospital mentioned by Osler, where movement was liable to be followed by transient instantaneous paralysis of all four extremities from the compression of the cord, the patient dying in one of the attacks.

If in the lower cervical region, there may be *dilatation of the pupils* from interference with the cilio-spinal centre. There may be flushing of the face and ear on one side or unilateral sweating, rigidity of the muscles of the neck, while the sensory and motor symptoms described, if present, will be found in the arms. The deformity of tubercular caries is not marked in this locality, but after recovery evidence of its presence may be found in a conspicuous callus, which may cause permanent

rigidity of the neck. The cortical inhibitory influence being suspended, both tendon and cutaneous reflexes are increased, sometimes so markedly as to produce in the lower extremities a pronounced type of the spastic paralysis, with increased patellar reflex and ankle clonus. The increase of the skin reflexes is less marked than that of the tendons.

When the dorsal and lumbar segments are involved, the lower extremities only suffer from the effect of compression; commonly the paresis is late, though rarely it may appear before the deformity of Pott's disease. When the lesion is confined to the dorsal region there may be girdle sensation and pain in the course of the intercostal nerves. Here, as elsewhere, motion is lost before sensation. As to the reflexes, since the reflex arc for the lower tendon reflexes is supposed to be in the lumbar region, compression of the *dorsal* cord should produce an increase in them, and this is the case. On the other hand, they are diminished when the *lumbar* cord is compressed. If the lower dorsal and lumbar region is affected, the sphincters are apt to be involved, and there is, first, difficulty in micturition, then retention, and finally incontinence with cystitis. Yet all these symptoms may disappear, and recovery take place after many months' duration of the disease.

Trophic symptoms may be present in the paralyzed parts, such as herpetic eruptions in the course of the nerves, at other times derangement of nutrition, manifested by bed-sores forming on slight irritative provocation and rapid shedding of the epidermis, while the nails become brittle. With the involvement of their trophic centre the muscles may waste.

Diagnosis.—This is easy where there are evident signs of caries of the spine, manifested by prominence of spinal processes of the vertebræ and by tenderness on pressure. Repeated examination of the spine should be made. Root symptoms are always significant, as is also stiffness on motion in separate parts of the spinal column. Root symptoms are said to be more common in cancer than in caries, but any of the symptoms named have increased diagnostic value if there has been a cancer elsewhere and if the age exceeds forty. There is much more pain attending the paraplegia of cancer—whence the term paraplegia-dolorosa. Such is the case whenever erosion is wrought from the abdomen outward, as by retroperitoneal growths or aneurism.

Prognosis.—This is unfavorable in all cases except tubercular spondylitis, which often terminates in cure, for, sooner or later, especially with suitable treatment, the tubercular process often ceases and the symptoms of paralysis disappear entirely, although, of course, the kyphosis remains. Some cases perish from miliary tuberculosis, others from the exhaustion incident to bed-sores, cystitis, and pyelo-nephritis.

Treatment.—Only when tubercular spondylitis is responsible is there hope of cure. The treatment is general by the usual measures found useful in tuberculosis, such as cod-liver oil, such tonics as iron and iodine, good food, fresh air, and mechanical appliances suggested by the orthopædic surgeon. These should be so adjusted as not to produce pain. Their object is to produce extension and thus relieve compression, and if this is not accomplished they are useless. The method of extension

by suspension, originally suggested by Dr. J. K. Mitchell and more recently revived by S. Weir Mitchell and extensively practised of late years, has again fallen into comparative disuse, partly because of the difficulties in carrying it out, and perhaps because there have been some unfortunate accidents, but good results have followed its use. Especially may good results be hoped for if it is used early, although they have followed even after paralysis had supervened.

Along with the extension, rest in bed is a most important measure, and many cases are arrested by its use. Local measures, like counter-irritation and the hot iron, are of no use, rather harmful than otherwise. The same may be said of electrical treatment, except so far as it is useful to keep up the nutrition in the paralyzed muscles. On the other hand, warm bathing is useful in relieving pain and allaying discomforts.

Operative treatment—laminectomy—has lately been practised with a good show of result, and it should be considered, at least, after other measures have failed. Treatment should be persevered in, as recovery takes place sometimes after paralysis has long persisted, and in no form of tuberculosis has the general treatment above recommended been so useful.

In the incurable forms anodynes must be resorted to to relieve pain, including even the hypodermic use of morphine, which should never be used without bearing in mind the possibility of the morphine habit.

LESIONS OF THE CAUDA EQUINA AND CONUS MEDULLARIS.

The cauda equina is the bundle of nerves coming off from the lower cord and occupying the spinal canal from the 2d lumbar vertebra downward. At this vertebra the cord itself terminates in the conus medullaris, prolonged into the thread-like filum terminale. Fractures and dislocations in the lumbo-sacral region may impinge on these parts, while the filaments of the nerves of the cauda equina may be invaded by tumors or compressed by cicatrices.

Symptoms.—Compression of the conus and of the last sacral nerve given off from it, such as may be caused by a dislocation of the 1st lumbar vertebra, produces paralysis of the bladder and rectum, whence it has been inferred that the ano-vesical centre is seated in this part of the cord. This paralysis may be the only symptom or may be associated with a spot of anæsthesia in the neighborhood of the coccyx or perineum.

When the lumbar nerve roots * are involved, from the 2d to the 4th inclusive, there is paralysis embracing all the muscles of the thigh and leg, except the outer rotators of the thigh, the flexors of the knee and of the ankles, the peroneal, the long flexors of the toes, and the small foot muscles. There is also loss of sensation in the front, inner, and outer parts of the thighs and the inner side of the leg and foot. Involve-

* Of the lumbar nerves the 1st root appears between the 1st and 2d vertebræ, the 5th between the last lumbar and the base of the sacrum. The four upper sacral nerves pass from the spinal canal through the sacral foramina, the 5th between the sacrum and coccyx.

ment of the 5th lumbar and 1st and 2d sacral produces paralysis of the muscles just excepted, and loss of sensation in the outer and posterior part of the leg and foot and sole of the foot. Lesion of the 3d, 4th, and 5th sacral and coccygeal nerves causes paralysis of the perineal muscles, the bladder, rectum, and of the external genitals, the coccygeus, with loss of sensation in the back of the thigh, anus, perineum, genital organs, and skin about the anus and coccyx.

SPINA BIFIDA.

SYNONYMS.—*Split Spine; Hydrorrhachis; Myelocele; Meningocele.*

Definition.—A name applied to a congenital defect in the closure of the spinal canal, through which protrudes a sac-like portion of the dura containing cerebro-spinal fluid at times, a part of the cord either normal or altered, and forming also, as a rule, an external prominence of tumor covered by skin.

Description.—The tumor is found commonly in the lumbar and sacral portions of the spine, rarely in more than one place, very rarely throughout the whole column. Its size ranges from that of a small nut to an orange, and occasionally it is so large as to interfere with the birth of the child. On section of the skin, beneath it is the protruded sac of the dura, and beneath this the arachnoid. Rarely is the dura cleft so that the sac is formed by the arachnoid only. There may be a dilatation of the central canal—hydromyelia—when the substance of the cord is found more or less atrophied, while the central canal communicates directly with the cavity of the spina bifida. At other times the cord is normal, while its lower end may be adherent to a spot in the sac.

Symptoms.—At first there are usually no clinical symptoms. By pressure the contents of the tumor can often be forced into the spinal canal, causing expansion of the fontanelles and increase of cerebral pressure with its consequences, viz., somnolence, with changes in the pulse and breathing which may be fatal if the pressure is continued. The absence of such symptoms goes to show that communication of the tumor with the spinal cord is cut off.

With the lapse of time the tumor usually grows slowly and the results of pressure on the spinal cord or cauda equina appear. These are paralysis, atrophy, anæsthesia, bed-sores, vesical derangements, talipes varus, and trophic phenomena, of which perforating ulcer of the foot is one. The sac may burst, or the walls become inflamed, converting the contents into pus.

Prognosis and Treatment.—Unless removed by surgical interference the child dies sooner or later of exhaustion. The tumor has been rarely obliterated by gradually increasing pressure or by injecting the cavity, after evacuation of the fluid, with iodine, producing obliteration through an inflammatory process. Other surgical measures may be found in surgical text-books.

TUMORS OF THE SPINAL CORD AND MEMBRANES.

Both the membranes and the substance of the cord may be seats of tumors, while the cord may also be invaded from the spinal column by enchondroma or sarcoma.

Varieties.—From the spinal column, enchondroma, sarcoma, and cancer may intrude into the canal. External to the dura mater in the extra-dural space occur fatty tumors early in life, malignant tumors late, while parasites are also found in this region. The extra-dural tumors, all rare, may spring from the membrane or from the tissue between it and the bone, or from the outside through the intervertebral foramina. Within the dura are found myxomata, fibromata, lipomata, and neuromata on the nerve roots. Subdural tumors may arise from the inner surface of the dura, the arachnoid, or from the pia, and may include sarcomata, syphilitic and tubercular and parasitic growths. The last two are rare, but both echinococci and cysticerci have been met with, developing in the meshes of the arachnoid. Leyden has collected about a dozen cases of hydatid disease usually in the loose adipose tissue between the dura and the bone and, it is said, they sometimes form in the substance of the bone. Fatty tumors are also rare, but have been found and are probably congenital because they were found associated with spina bifida. In the cord itself and attached to the pia occur tuberculous, syphilitic, and gliomatous tumors; sarcomata and myxomata have been found. Syphiloma and sarcoma are the most common. Most of these tumors spring from the pia mater into the cord, but tubercular growths also grow from the grey matter. Some tumors are compound, as myxo-sarcoma, etc.

The size attained by them is never large, as the growth is restricted by the limited space. The largest do not exceed two inches (five cm.) in diameter and many are very small, not larger than a pea. They are usually single, rarely multiple, as is the case with neuromata. Tumors developing within the cord may lead to syringo-myelia.

Symptoms.—These vary with the seat of the tumor and the degree of pressure exerted. When the latter increases slowly the growth may reach quite a large size before serious mischief is done. *Pain* is a frequent and conspicuous symptom and is apt to maintain itself by pressure on nerve roots which are in the way of the growth. The seat of pain varies with the course of the nerves impinged upon, and may be of every variety, such as "burning," "tearing," "stabbing," "aching," "girdle sensations," and the like. It may be unilateral or bilateral and is worse, according to Horsley, when the tumor presses forward. Sometimes the pain is in the spine itself, which may also in rare instances be tender to pressure. When the growth is in the lower lumbar region the pain may be referred to the soles of the feet, and may ascend from this seat. At others there is hyperæsthesia of the skin, which may be associated with pain felt at the level of the tumor, or pain may be felt in anæsthetic areas. Very rarely pain is absent, chiefly in extra-dural lipoma.

Muscular spasm is also frequent, especially when the tumor springs from the membranes, when it may be very decided. There may be

rigidity at the seat of the growth, most marked when the disease is at the more mobile parts of the spine, as in the cervical region. Then there is apt to be pain in the vicinity, increased by motion. Spasm in the abdominal muscles may be associated with girdle pains. Contractures may arise in the limbs, both those supplied by nerves directly irritated by the tumor and by those given off lower down. It is important to note the seat of the rigidity and its character, whence we may learn the seat of the tumor, whether it is in the nerve roots or conducting tracts of the cord. Thus a tumor in one half of the cord, in the cervical region, may cause persistent contraction of the arm and leg on the side of the growth, and in the early stage of dorsal tumors one leg only may be rigid at a time or one may be more so than the other. In the dorsal region the level of the pain is apt to correspond accurately with the level of the growth, and the reflexes centering at this level may be lost, but retained in the legs.

Paralysis occurs sooner or later, as constantly as pain, increasing gradually with the pressure. Paraplegia is more common, but all four limbs may be paralyzed by a tumor in the cervical region, one limb being usually affected before the other, though when the tumor is exactly central both sides are affected simultaneously. Loss of sensation follows paralysis sooner or later. It corresponds in distribution to the motor palsy when the tumor is below the middle of the dorsal region, but if higher and on one side, the sensory loss is greater on the opposite side; especially is this the case when the tumor is within the cord, when the symptoms may be those already described under the head of Brown-Séquard's paralysis.

Atrophy follows involvement of the anterior cornua and *vaso-motor* disturbances may be marked. In cases of prolonged interruption ascending and descending degenerations may occur. Tumors not infrequently cause subacute or acute myelitis, whose symptoms may mask the clinical picture.

Diagnosis.—The characteristic symptoms are slow development of severe and constant root symptoms at the level of the growth and a progressive paralysis, motor and sensory. The radiating pain is usually at the level of the tumor or below. Pain in the spine itself is an important sign. Rigidity of the muscles of the spine, muscular contractions in the limbs, early and marked exaggeration of reflex action, when the cord itself is involved, are also important signs, especially when associated with the history of syphilis or tubercular disease. Caries of the spine may produce the same symptoms, but the radiating pains are less severe and the effects of compression of the cord are more likely to be bilateral, either from the first or soon after their commencement. Tenderness of the spine may generally be elicited by careful examination, while irregularity of surface, from the breaking down of the bone, sooner or later makes its appearance. When the tumor is in the bone itself, the symptoms at first scarcely differ from those of caries, though the pain on motion is usually worse in the former.

The symptoms of cervical meningitis also closely resemble those of tumor. They are usually from the first bilateral, and have a very con-

siderable vertical extent. Central tumors covering a like area may produce identical symptoms. Pain and muscular atrophy in the arms without wasting occur in both, but are usually less severe in tumor, while the early and localized impairment of all forms of sensation is also less.

Chronic transverse myelitis also closely simulates tumor in the radiating pain, sense of constriction, and progressive paralysis, but the radiating pain is never so severe in myelitis, which invades also larger areas of the cord.

The nature of the tumor may be inferred only from the history of the case, syphilis and tuberculosis giving the most valuable assistance. Its seat is suggested by the level of the transverse symptoms. It is never below these, while it may be a distance of three or four vertebræ above the nerves corresponding to the highest level of anæsthesia or pain.

Prognosis.—Only where the tumor is syphilitic may any relief be expected from treatment. In all other forms the symptoms gradually increase until paralysis results unless operative interference produces a more favorable termination—a practice which modern methods is rendering more frequent and justifiable.

Treatment.—Where there is reason to believe syphilis is present the antisyphilitic treatment may be used with reasonable expectation of relief. Beyond this, symptoms must be met as they arise. Attempts made of late years to formulate the laws governing surgical operations in these cases have been more or less successful, but wider experience is necessary before they can be thoroughly relied upon. I may, however, close this subject by the advice of Victor Horsley, whose studies on surgery of the nervous system entitles his opinion to the highest respect: "If it is clear that the growth is not syphilitic, and that no good can be done by other treatment, delay in an operation can only cause harm—can only result in a less favorable state for the proceeding, less chance of recovery, longer and greater suffering, and should, on every ground, be avoided."

PROGRESSIVE BULBAR PALSY.

SYNONYM.—*Glosso-Labio-Laryngeal Paralysis.*

Definition.—Bulbar palsy is a progressive paralysis invading the tongue, the lips, the palate, pharynx and larynx, and in more advanced cases the lower face muscles, due to lesion of the motor nuclei in the medulla oblongata (or bulb), whence arise the nerves distributed to those parts.

Historical.—Bulbar palsy was first completely described in its clinical aspects by Duchenne in 1860, but the exact seat of the disease was not determined until 1870, when Charcot in France and E. Leyden in Germany confirmed the prior suggestion that it was a progressive degeneration and atrophy of the nuclei in the medulla oblongata.

Etiology.—Primary progressive bulbar palsy is difficult to account for. It has been ascribed to the over-use of the muscles of the mouth,

as in the blowing of wind-instruments; to a tumor in the medulla or vicinity; while syphilis, to which so many of the unaccountable lesions of the nervous system are ascribed, is less commonly held accountable for this affection than for some others. Cold, emotional excitement, and extreme fatigue have all been named as causes. Most frequently, however, no cause is traceable.

Pathology.—Most writers concede that the lesion starts in the medulla. Possibly, however, as claimed by Friedreich, the process may begin in the muscles and ascend along the motor nerves to the ganglia involved, the atrophy of which is thus the terminal rather than the initial act. Or it may be that the entire motor apparatus from the muscular fibre to the ganglionic cell is invaded simultaneously. Certain it is that bulbar paralysis is often associated both with progressive muscular atrophy and amyotrophic lateral sclerosis, the symptoms now of one and now of the other preceding. There can be no doubt that these three conditions are closely allied. The nature of the lesion is the same in each, the motor ganglia in each are involved, the muscles are wasted in each, though the particular ones involved vary as the situation of the motor ganglia is different.

The anatomical lesion is an atrophy of the motor ganglia of the medulla oblongata. The nucleus of the hypoglossus, to a less degree the common nucleus of the pneumogastric and spinal accessory, of the facial, and even sometimes that of the glosso-pharyngeal are all involved, while the sensory nuclei are intact. From these nuclei the degeneration extends to the nerves which have their origin in them, and thence to the muscles to which they are distributed.

The nature of the degeneration is a more or less complete destruction of the ganglion cells, which become fatty. In addition, there is an overgrowth of connective tissue and a thickening of the walls of the blood-vessels. The nerve trunks themselves are narrow and grey, owing to the destruction of the medullary sheaths.

Symptoms.—The symptoms of progressive bulbar paralysis are exceedingly gradual in their development. The first noticeable is usually a *difficulty in the pronunciation of words* containing letters which require the use of the tongue in their formation, such as E, R, L, S, G (hard), K, D, T, and N. Still later there is difficulty in pronunciation of words requiring the aid of the lips, as O, A (long), P, F, B, V, and the sound of O in tool, while whispering becomes impossible.

Concurrently with these symptoms the *tongue and lips* are both observed to *waste*, the tongue becomes thinner and narrower, the lips thin and compressed in appearance, the loss of power being commensurate with the degree of wasting. *Fibrillar* tremors may be seen in the tongue, and the mucous membrane may be thrown into transverse folds. Finally, the tongue cannot be protruded or can be brought only to the edge of the teeth, and the *mouth cannot be closed* because of complete paralysis of the orbicularis oris. In more advanced stages other muscles of the face become involved, the labio-nasal fold is increased, and the face becomes expressionless.

Before this degree has been attained, however, the muscles of the

palate have commenced to fail in their action, and thus a further difficulty in the articulation of words is added, and fluid begins to pass through the nose when swallowing is attempted. The difficulty in swallowing is increased by growing *paralysis of the pharyngeal muscles*, and is further aggravated by the inability of the tongue to carry the bolus of food backward. Feeding with the patient is a troublesome and disgusting process, the food being scattered all about and sometimes thrown to considerable distance by the act of coughing and absence of power of the lips to retain substances in the mouth. By this time, too, the *laryngeal muscles* are involved, and the patient's efforts to speak, to tell his name, result in mere grunts. Earlier than this the voice becomes nasal from paralysis of the palate.

Thus he cannot talk, he cannot swallow, he cannot close his mouth. He cannot expectorate, yet the saliva flows from his mouth because he can neither swallow nor close his lips, and the term "driveling idiot" well covers the impression caused by his appearance. Yet his mental powers are unimpaired, and remain so until the last. The motor electrical phenomena in the muscles involved are altered, although it is difficult to test them.

To these symptoms are to be added complications due to the paralysis. From the difficulty in swallowing, particles of food may enter the larynx, be insufflated to the deeper parts of the lungs, and there cause a pneumonia which may be fatal, or the fragment which enters the larynx may be so large as to cause death by suffocation.

In rare cases the entire distribution of the facial nerve is involved, producing *diplegia facialis;* or that of the ocular nerves to which it may be confined (anterior bulbar paralysis, or progressive ophthalmoplegia of Graefe), and even also the muscles supplied by the spinal accessory and the motor branch of the tri-facial. In all these instances the nuclei of the corresponding nerves are involved.

Diagnosis.—The diagnosis is generally easy, the symptoms are so characteristic and so evident. For a typical case they must be purely motor; they must be disassociated from other muscular involvements which would go to make them a part of a progressive muscular paralysis or an amyotrophic lateral sclerosis. If there are disturbances of sensation, invasion of the upper division of the facial, of nerves of special sense or ocular muscles, the disease is not true bulbar paralysis. There must be some more general involvement of the medulla, thrombosis or embolism, a tumor developing near it or diffuse sclerosis through it.

There is a glosso-labio-pharyngeal paralysis of cerebral origin known as "pseudo-bulbar paralysis," in which there is complete paralysis of the tongue and lips, due to bilateral and even unilateral cerebral disturbance in the lower part of the ascending frontal convolution. Close examination will, however, detect, sooner or later, deviations from the typical course, which will determine the correct diagnosis.

Prognosis.—The prognosis is invariably, sooner or later, fatal, although it is said that the progress of the disease may be delayed by treatment.

Treatment.—If there be any suspicion that syphilis is the cause,

iodide of potassium should be used, but the treatment which has been found most effectual is electrical. Galvanism is recommended, electrodes being applied to the two mastoid processes daily for two or three minutes, the current often reversed. The sympathetic nerve and the affected muscles of the lips and the tongue may be similarly treated, faradizations being also substituted for galvanism in the case of the muscles. Deglutition may even be excited by galvanism when it begins to be impaired. This is accomplished by placing the anode on the nape of the neck and the kathode on one side of the larynx. At every kathodal closure, or every time that the kathode is carried across the side of the larynx, there is a reflex act of deglutition. When deglutition becomes very difficult the stomach-tube should be used and nutrient substances thus introduced. Great care should be exercised in feeding the patient without the tube, lest the food pass into the trachea and cause suffocation. Hence, too, the use of the tube should not be too long deferred.

In addition to iodide of potassium, nitrate of silver and ergot are also recommended. The first should be given in such doses as the stomach will tolerate, while salivation may be controlled by atropine $\frac{1}{100}$ to $\frac{1}{60}$ gr. Silver should be given in the usual doses of $\frac{1}{6}$ to $\frac{1}{4}$ gr. (0.0106 to 0.016 gm.); ergot in usual doses.

Acute Bulbar Palsy.

Besides the chronic or progressive form of bulbar palsy, there is an acute variety which is caused by hemorrhage into the pons and medulla by embolism or thrombosis of the vessels supplying these centres, the anterior spinal, vertebral, and basilar, also by inflammation of the medulla oblongata. Emboli only occur in the vertebral artery, most frequently in the left. They are generally cardiac in origin. They may extend by thrombotic detachment into the basilar, but are never primary in the latter. Thrombosis may occur in any of the vessels and is commonly due to the atheromatous or syphilitic endarteritis. Inflammation is a rare affection, but does occasionally occur.

Etiology.—The etiology of hemorrhage, thrombosis, and embolism is that of this condition elsewhere in the brain, but the cause of the inflammatory form of acute bulbar palsy is unknown.

Symptoms.—In any event the symptoms are sudden. They are those already detailed in connection with progressive bulbar paralysis, but there are others added. There is usually no loss of consciousness, but there may be, in consequence of disturbances of cardiac action or of respiration. Among the latter are irregular and frequent pulse, vasomotor derangements, Cheyne-Stokes breathing. The temperature, normal at first, may rise to 105° to 107° F. (40.5° to 41.71° C.), and higher toward a fatal termination. Sensation is rarely affected. Most characteristic of all is the so-called *crossed paralysis*, described on p. 798, which attends most hemorrhages into the pons, in which there is paralysis of the face on one side and of the extremities on the other.

Diagnosis.—The diagnosis of these conditions depends on the suddenness of their occurrence and the presence of the bulbar symptoms,

while a crossed hemiplegia, that is, paralysis of the arm on one side and of the leg on the other, is conclusive of a lesion at the decussation. Where inflammation of the medulla is present the phenomena of bulbar paralysis do not occur quite so suddenly. They may be several days or even a few weeks in developing, and may be preceded by prodromal symptoms such as vertigo and painful sensations in the back of the neck.

Treatment.—The treatment is the same as for similar lesions elsewhere in the brain.

AMYOTROPHIC LATERAL SCLEROSIS.

Notwithstanding the similarity of the clinical phenomena and, to a certain extent, of the morbid anatomy of amyotrophic lateral sclerosis to those of the so-called progressive muscular atrophy, to be next considered, there appears to me sufficient difference to demand a separate consideration.

Definition and Historical.—Amyotrophic lateral sclerosis is a systemic degeneration of the pyramidal tracts of the spinal cord with atrophy of motor cells in the anterior cornua and medulla oblongata, and consequent wasting of muscles depending upon these cells for their trophic influence.

The confusion which has long existed between the disease and progressive muscular atrophy was first cleared up by Charcot and his pupil, Joffroy, who published a fairly accurate account of the disease in 1869 and a complete description in 1874. Such description became possible, however, only after Flechsig's descriptions of the paths of motor conduction in the spinal cord. That there are, however, certain common features in the two affections appears not only in the clinical history, but also in the morbid anatomy.

Etiology.—The causes of this condition are still essentially unknown. Severe muscular exertion has been assigned as a cause, as it has also of the allied affection, progressive muscular atrophy. As in it, too, the male sex suffers most. It is a disease of young adult life and middle age, from twenty-five to forty-five.

Morbid Anatomy.—A sclerosis of the crossed pyramidal tract in the two lateral columns and the direct pyramidal tract in the anterior columns is essential to the morbid anatomy in a typical case. As important is atrophy of the corresponding large ganglion cells in the anterior cornua and medulla. The degeneration has been traced by Charcot and Marie in the pyramidal tracts from the lowest part of the lumbar cord upward to the pyramids in the medulla, sometimes even through the pons and crura into the internal capsule and central convolutions, in which, too, the large ganglion cells have been found atrophied. The nerve nuclei which are affected in the medulla oblongata are especially the hypo-glossal nucleus, the vagus, and spinal accessory, into the trunks of which the degeneration extends toward the periphery, although it is not always easy to make out the atrophied fibres in these nerves.

The changes in the motor ganglion cells of the cord and the nerve

nuclei in the medulla are analogous and produce corresponding results in the muscles supplied by the motor nerves originating from them. These results are an atrophy present in various degrees, some disappearing almost entirely, others partially. The process is by fatty metamorphosis and absorption of resulting fat, leaving a residue of connective tissue. It is identical with that which occurs in the ganglion cells in progressive muscular atrophy.

In this disease, too, the process may extend to adjacent parts of the cord *per contiguum.* In this way may be involved to a slight extent the posterior columns and the lateral cerebellar tract.

Symptoms.—The clinical phenomena are in strict accord with what would be expected from the pathological lesions, consisting in *muscular wasting* and *corresponding paresis.* Before the muscular wasting appears, a *sense of fatigue* succeeding slight effort may be manifested, followed by a positive weakness, first almost always in the arms, first one and finally both. This is followed by wasting of the muscles of the upper extremity, usually first seen in the thenar and hypothenar eminences, the interossei and the muscles on the extensor side of the forearm, the flexors of the hand and fingers remaining longer uninvaded. The atrophy is particularly well seen in the deltoid, and to a less degree in the triceps, still less in the biceps and shoulder muscles.

Not until about six months after their appearance in the upper extremity do symptoms begin to appear in the lower ; then the patient tires easily in walking, the gait becomes unsteady and stiff, and rising from the chair becomes difficult. Tremor may appear in the legs. The paresis in both extremities is proportionate to the destruction of muscle, though first, at least, it is independent of the atrophy. Associated with muscular atrophy, sooner or later, is a *diminished electrical excitability,* which remains, however, as long as the muscles are intact, diminishing as their destruction diminishes. A reaction of degeneration may also develop in the muscular fibres still intact. The excitability remains for the most part intact in the nerve trunk, because in any event a large number of fibres are preserved in their normal state.

A distinctive feature of amyotrophic lateral sclerosis is found in the *reflexes,* which, in strong contrast to progressive muscular atrophy, are *markedly increased.* Even in the early stages of the disease vigorous contractions are obtained by gently tapping the tendons of almost any of the muscles in the extremities. Always most conspicuous is the patellar reflex, while more rarely ankle clonus may be obtained. The same is true of the masseter reflex. In the arms the biceps and triceps and the flexors of the hands may be excited to strong contraction.

Contractures may take place in the later stages of the disease in the arm and hands, but not always. In the lower extremities, where the atrophic symptoms develop some months later and are less marked, *spastic* symptoms are a more prominent feature. The legs become rigid and some strength is required to flex them, though the muscles themselves are paretic. A typical spastic paraplegia may be produced, which is due mainly to the increase of the tendon reflexes, and a spastic paretic gait is common—that is, at first. On starting to walk, the patient seems

glued to the ground, making ineffectual efforts to lift the toes; then four or five short, quick steps are taken on the toes with the body thrown forward. Finally he starts off at a rapid rate.

After the disease has lasted a couple of years, *bulbar symptoms* may present themselves, manifested first by defects of speech, difficulty in retaining the saliva, and in swallowing; and later the lips and tongue may be seen to be atrophied, and ultimately there is difficulty in taking food, whence nutrition is impaired, and the patient gradually sinks.

Throughout *sensibility* remains *normal* in the upper and lower extremities, and the superficial reflexes are not much altered. The *sphincters* are, as a rule, *unaffected*. Micturition is natural. Only in the event of involvement of the reflex centre in the lumbar cord may there be incontinence at night when the inhibitory power is in abeyance. There may be constipation but no actual paralysis of the bowel. Sexual power may be lost.

The successive involvement of the upper extremities, the lower extremities, and the bulbar centres marks pretty well-defined stages of the disease.

Death comes ultimately by exhaustion, or more frequently through an inspiration pneumonia, caused by entrance of foreign matter into the air passages as a result of defective deglutition.

Diagnosis.—The disease is distinguished from progressive muscular atrophy by the invariable increase in the tendon reflexes even in the early stages, as contrasted with their absence in the latter disease.

Prognosis and Treatment.—The prognosis is very unfavorable and the disease cannot be arrested. By rest in bed, massage, electricity, and hot bathing we may be able to defer the end somewhat. (See, also, treatment of Progressive Muscular Atrophy.)

PROGRESSIVE MUSCULAR ATROPHY.

Synonyms.—*Spinal Form of Progressive Muscular Atrophy; Wasting Palsy; Progressive Muscular Atrophy, type Duchenne-Aran; Cruveilhier's Atrophy; Chronic Anterior Polio-Myelitis; Chronic Degeneration of the Motor Nuclei.*

Definition.—Progressive muscular atrophy is a progressive wasting of more or less limited groups of voluntary muscles, associated with degenerative atrophy of that portion of the conducting nerve tract extending from the ganglion cells of the anterior cornua, inclusive, to the muscles, but *unaccompanied by disease of the pyramidal tracts.*

Historical.—The history of the development of our knowledge of this disease is very interesting. A few facts only can be given here. Although a number of isolated cases were described at an earlier date, Duchenne's memoir on "*Atrophie Musculaire avec Transformation Graisseuse,*" published in 1849, and Aran's "*Recherches sur une Maladie non encore decidé du Systeme Musculaire,*" published in the next year, contained the first accurate description of this malady. Cruveilhier's studies were

commenced in 1832, but not given out until 1853. All of these observers believed at first—Cruveilhier reluctantly—that the disease was purely muscular. In his third case, however, Cruveilhier found atrophy of the anterior roots of the spinal nerves, and in his fourth, lesion of the grey matter of the cord, whence the anterior roots take their origin, and first asserted the belief that the disease of the grey matter in the spinal cord was the special anatomical lesion of the disease. The researches of Lockhart Clarke in 1866 and 1867 and of Charcot in 1869 may be said to have established the spinal nature of the disease; while Friedreich in 1873 still maintained its muscular nature, and Gowers and Leyden regard it as identical with amyotrophic lateral sclerosis. Strümpell also separates the two diseases of progressive muscular paralysis and amyotrophic lateral sclerosis, and it appears to me there is quite enough reason for doing so.

Etiology.—In the majority of instances we fail to find a sufficient cause. Heredity has been regarded as playing an important rôle in its causation, but Strümpell considers the cases thus originating as instances of the juvenile myopathic variety of atrophy—that in which no nervous lesion is traceable. On the other hand, a habit of excessive muscular exertion seems to be more than an accidental coincidence. Exposure to cold, especially the action of very cold water, and the infectious diseases—typhoid fever, influenza, diphtheria, and syphilis—have all been held accountable, but it is likely that some of the atrophies thus resulting include other forms than the true progressive muscular atrophy.

It is a disease commonly of adult males, supposed cases among those who are younger being probably, as held by Erb, instances of the juvenile form of muscular atrophy or of pseudo-hypertrophic paralysis.

Morbid Anatomy.—The anterior horns of the grey matter are wasted and reduced in size; their ganglion cells wholly or partially destroyed; the neuroglia converted into fine fibrous tissue, intercalated in places with spider cells. The anterior nerve roots leading to the horns are atrophied, as are also the motor nerve filaments in the peripheral nerves. But the anterior pyramidal tracts in the lateral columns containing the crossed motor fibres descending from the brain to the cells in the anterior cornua are intact. The muscles seen to be wasted before death are found converted into fat and connective tissue, a remnant of true muscular tissue remaining. At times also they are the seat of waxy change, at others still simply narrowed but retaining their transverse striation.

As to the relation of the nervous changes to the muscular atrophy, the conspicuous symptom of the disease, there is more than one possible explanation. According to one, the muscular atrophy is primary, possibly due, Friedreich sought to prove, to a myositis, followed by fatty metamorphosis of the sarcous substance and subsequent absorption of fat, or a simple primary fatty metamorphosis. In such event it may be inferred that the nerves atrophy from want of use. According to a second view the atrophy of the anterior cornua is primary, the result of a chronic polio-myelitis anterior, and the degeneration of the peripheral nerves and muscles is secondary to it. According to a third, the degeneration begins

in the last terminal branches of the motor nerves and extends upward along them to the spinal cord. Finally, there may be a simultaneous degeneration of the whole motor system involved, including muscle and nerve. I cannot but regard it more in accord with the office of the spinal cord as a nutritive centre, as well as with its morbid anatomy, to suppose the disease is a chronic polio-myelitis anterior, the essential infantile paralysis of Relliet and Barthez being an acute form of the same disease. Very important is the anatomical fact that the lateral columns, and especially the pyramidal tracts, are quite normal. Changes in other parts of the cord, sometimes found associated with those of the anterior roots, may be the result of extension by continuity.

Symptoms.—One of the most striking features of the disease is its *slow development.* Like its congener, amyotrophic paralysis, it begins most frequently in the *upper* extremities, seven out of nine times in Aran's cases. Of the upper extremities, the right was first invaded in 37 out of 62 of Sandahl's cases, the left 14 times, while the involvement was simultaneous in 11. The disease may begin in the *lower* extremities, as shown by Friedreich's statistics, according to which these were first invaded 27 times out of 146, the upper extremities 111, the lumbar muscles in eight.

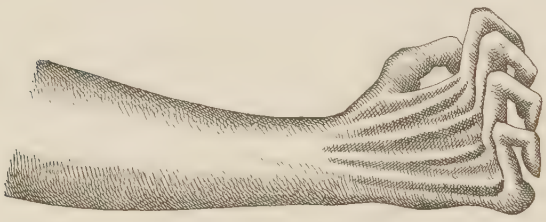


FIG. 74.—CLAWED HAND.—(*After Duchenne.*)

The atrophy usually begins with the short muscles of the thumb, the abductor pollicis brevis first, then the opponens and the adductor. The consequent flattening of the ball of the thumb and its persistent approximation to the 2d metacarpal bone, producing the so-called "ape-hand." Simultaneously, or almost simultaneously, the interossei begin to waste, producing conspicuous depressions between the metacarpal bones, associated with loss of power to extend completely the terminal phalanges of the fingers. Atrophy of the lumbricales follow, producing a flattening of the hollow of the hand. The ultimate result is the characteristic *main en griffe* of Duchenne, in which the extensor tendons on the dorsum of the hand, and the flexors in the palm, become as distinct as if dissected out.

From the hand the wasting creeps up the forearm and thence to the arm, or it may skip the forearm and pass into the arm, sparing usually the triceps extensor. In the forearm the muscles on the extensor side are usually first affected, the abductor and extensor longus pollicis, and later the supinators and flexors. It may come to a standstill at either of these stages or may involve the muscles of the shoulder, especially the deltoid, in which, indeed, it may begin, preferably in the right, passing thence to the scapular and trapezius muscles, the pectorals, the rhomboidei and latissimus dorsi, while a grotesqueness of effect is often produced by reason of certain adjacent muscles retaining their natural size

or being even seemingly hypertrophied. This is particularly the case with the inferior part of the trapezius and platysma myoides, which are almost never involved. The disease may arrest itself at almost any of these stages.

If the lower extremities are first affected the gluteal muscles, the vasti, and the anterior tibials are first attacked. The face muscles are invaded late or not at all, but ultimately even the intercostal and abdominal muscles may be involved. The result, then, is a veritable living skeleton, instances of which are sometimes exhibited. Deformities, including lordosis or anterior curvature of the spine, may result.

With all this, *sensibility* is unaffected in the vast majority of cases, but the patient may complain of a numbness and coldness of the affected limbs. Very rarely *pains* precede the wasting in the muscles, when they are sometimes regarded as rheumatic. The *galvanic and faradic irritability* of the muscles progressively diminishes and disappears with the complete destruction of the muscle, the galvanic persisting longer. The *reaction of degeneration* may, however, be elicited late in the disease in certain muscles, more especially in the modified form known as "partial reaction" of degeneration.* If the disease runs a rapid course, it may occur earlier and be more typical. *Fibrillary* muscular contractions may be present and idiopathic muscular contractions, brought out by a blow, or myoid tumors may be thus produced. The bladder and rectum remain intact, but sexual power may be lost.

Sweating and other *vaso-motor* disturbances may occur in the affected muscles, such as pemphigoid bullous eruptions, thickening and fissuring of the skin, and curving and grooving of nails. In certain places there is an over-accumulation of fat, producing an appearance of hypertrophy when there is actual atrophy.

Along with wasting there is a corresponding *paresis*, the result of the atrophy and not its cause. The arms are flaccid and toneless and hang loosely at the sides. The patient can no longer dress himself, and various devices are resorted to to accomplish acts. Especially characteristic is one of these—when the shoulders, being first affected, the arm and forearm retain their usefulness. Under these circumstances the power of lifting the arm from the side, and especially of raising it above the head, is lost, while that of the forearm remains. Hence, if the patient wishes to lay hold of anything he swings the arm forward with a jerk until the object is brought within reach of his fingers, when it may often be caught by the pathologically hooked terminations of these. As long as the neck muscles remain effective, objects may be grasped by the mouth.

In true progressive muscular atrophy the *reflexes* are entirely

* A peculiar electrical reaction, named by Remak diplegic, is said to be sometimes produced in muscular atrophy, as follows: When the cathode or negative pole of a galvanic battery is put below the 5th cervical vertebra, contractions can be produced in the atrophied muscles of the arm when the positive pole is placed in an irritable zone, which extends from the 1st to the 5th cervical vertebra, or in the fossa between the lower jaw and the external ear. The contractions take place on the side opposite that on which the anode is placed, but when it is placed in the median line they occur on both sides, being limited, however, when the current is weak to the muscles most seriously involved. Remak's statement is confirmed by Erb and others, but not by Eulenberg or Benedikt.

absent, at least in the wasted extremities, a natural result of the atrophy of the ganglion cells in the anterior cornua and of the centrifugal motor fibres of the reflex arc. Sensibility, the *special senses*, and the *sphincters* remain normal.

Toward the close of the disease sometimes, and then only after it has existed for a long time, the phenomena of *bulbar* paralysis may present themselves after invasion of the ganglia of the medulla oblongata. These have been detailed in the section on that subject. They are by no means always present even in advanced cases.

Diagnosis.—Muscular atrophy is not confined to the disease under consideration. It occurs in diffuse myelitis, in tumors of the cord and when cavities are formed in its interior, in multiple neuritis, and especially in amyotrophic lateral sclerosis. From all these named, except the last, it is easily distinguished by strict attention to the conditions and order of development of the symptoms, viz., insidious and progressive atrophy of groups of muscles to the exclusion of others, beginning usually in the hand or more rarely in the shoulder and upper arm ; accompanied by a corresponding loss of power in the affected muscles and partial or complete reaction of degeneration in the same, by diminished reflexes and fibrillar twitchings.

Differential Diagnosis.—From amyotrophic lateral sclerosis it is to be distinguished by its greatly slower course and absence of the reflexes and of spastic symptoms. It is also to be distinguished from the myopathic juvenile muscular atrophy of Erb, pseudo-hypertrophic muscular paralysis, and Duchenne's infantile hereditary palsy. In juvenile progressive muscular atrophy there is also slow, symmetrical, and intermittent wasting with weakness in certain groups of muscles, especially those of the shoulder, girdle, and upper arm, the pelvis, upper thigh, and back, associated, however, with true or false muscular hypertrophy and with a peculiar toughness of the atrophying muscles but unassociated with fibrillar contraction or reaction of degeneration. The average age, also, in the juvenile form is much less, Erb's cases ranging from seven to forty-six and one-half, or an average of twenty-six and one-half, while in the spinal form or true progressive muscular atrophy the average age is much greater. Of Roberts' cases, all of which seem to be true cases of progressive muscular atrophy, the youngest was twenty, while the ages of the remaining four were thirty-eight, thirty-nine, forty-seven, and sixty-seven.

While in pseudo-hypertrophic paralysis there is also great weakness and wasting of muscles, though it may be obscured by the fatty deposit, there are absolutely no alterations in the spinal cord. It is also a disease of childhood, and strikingly hereditary, beginning in the lower extremities, while progressive muscular atrophy is a disease of adults, is not hereditary, and begins usually in the upper extremities. Duchenne's hereditary infantile atrophy is characterized by onset at an early age, infancy or adolescence, and by beginning in the facial muscles. It is often hereditary. The distribution of the atrophy is almost identical with that of Erb's form, except that it begins in the face. The muscles of the hands and fingers are spared ; fibrillar tremors are not present, and there is no reaction of degeneration.

Prognosis.—Many years are required to develop these symptoms in their entirety, and there may be spontaneous arrest, during which the patient may die of other causes. Sooner or later, if the patient lives, they recur, and their march is irresistible.

Treatment.—It has already been said that cure is impossible, although well-authenticated cases of arrest are reported. Mercurials and iodide of potassium should be used in cases of suspected syphilitic origin. Cooke reports a case of arrest under a course of mercury, after the disease had progressed for five years, during which many remedies were tried. In the main, the treatment must consist in measures intended to maintain the health and strength of the patient and to counteract the muscular wasting. To the former end an abundance of nutritious food, fresh air, and out-door life should be supplied, while tonics, including, especially, cod-liver oil, iron, arsenic, and strychnine, are indicated. The muscular wasting may be combated by electricity and judicious massage. Both kinds of electricity may be used, the faradic with rapid interruption to stimulate the circulation, or with slow interruption to excite individual muscles to contraction. The currents should be of moderate degree, not too frequently interrupted, and continued for a few minutes only. Duchenne recommended, particularly, treatment of important muscles like the diaphragm through the phrenic nerve, or the intercostal muscles and the deltoids before they are actually invaded by the disease. In evidence of its usefulness, he relates the case of a man who had lost many of his trunk muscles, and who was beginning to suffer with dyspnœa, on whom faradization of the phrenic nerves, repeated three or four times a week, was of great service, enabling him to walk considerable distances and to go up-stairs without fatigue. Another patient, whose arms were much wasted, was again able to support his family. The direct current—galvanism—is useful in advanced stages of the disease, where the strongest faradic currents fail to produce response. Where galvanic currents fail to excite contractions, the treatment ought to be persevered in for a long time, using very strong currents at the onset, gradually reducing them as contractility returns. Remak, who especially advocated the use of the continuous current, advised placing the positive pole in the front of one mastoid process and the negative pole on the opposite side of the neck, near the spinous process of the vertebræ, not higher than the 5th cervical, by which he produced the contractions already described as diplegic in the fingers and other paralyzed parts. Galvanization of the sympathetic has been apparently useful in the hands of some, Erb reporting a case of complete cure.

Massage is especially important and should be used in connection with electricity, but at a different time of day. Eulenburg refers to a case said to have been brought to a standstill by it.

Hypodermic injections of strychnine, $\frac{1}{100}$ to $\frac{1}{40}$ grain (0.0005 to 0.002 gm.), are alleged to have arrested the disease on the authority of Gowers.

In families in which a hereditary tendency exists, prophylactic treatment should be used. It should include hygienic measures of the kind already referred to and the avoidance of undue fatigue and expos-

ure, and in the selection of an occupation these matters should be kept in view. On the supposition that the disease is a purely local one, gymnastics, involving the exercise of the groups of muscles prone to attack, are indicated, but assume less importance from the standpoint that it is a spinal-cord disease. At the same time, the patient should have the benefit of any doubt in the pathogeny, and as gymnastics are eminently calculated to improve the general health, and thus indirectly to avert the disease, their use is indicated on these grounds.

DISEASES OF THE BRAIN.

LOCALIZATION OF CEREBRAL DISEASES.

SYNONYMS.—*Cerebral Localization; Relation of Locality to Symptoms; Topical Diagnosis of Cerebral Lesions.*

Physiology.—The brain is the organ of consciousness, and of perception of impressions and sensations,—of memory, of thought, of origination of voluntary motion, and of speech. It is also the seat of the instinctive acts. It has been learned from clinical observation in connection with studies at the autopsy table and from experiment that certain parts of the cortex have to do with certain offices, especially motion, speech, vision, and hearing, so that from the presence of certain symptoms the involvement of certain localities may be inferred. Allusion has already been made to the subject of topical diagnosis, p. 796. Such diagnosis, it is important to remember, gives no information as to the nature of the lesion, the result being the same whether it be abscess, hemorrhage, or softening. We are simply informed that such and such area is involved.

Historical.—As early as 1825 Bouillaud asserted that derangements of speech are produced only by disease of the anterior lobes of the brain. In 1836 Marc Dax, also a French physician, pointed out that aphasia was caused only by lesions in the *left* half of the brain. In 1861 Broca announced that aphasia results from a lesion of the 3d left frontal convolution, which was accordingly called the convolution of Broca. More recent observations show that it is the posterior part, or the *pars opercularis*, which is the speech centre, and, further, that lesions here are the cause of motor aphasia only. In 1870 Fritsch and Hitzig published the results of their experiments in irritating the surface of the brain in animals, such irritation being followed by muscular contractions in definite portions of the opposite side of the body. These observations were rapidly confirmed and extended in further experiments by Meynert and Flechsig among anatomists, Ferrier, Munk, Goltz, and others among physiologists, and Charcot, Nothnagel, Hughlings Jackson, and Horsley among clinicians. Our knowledge in this department is, however, still inexact, and is likely to be altered as well as increased by further studies.

I. The Motor Areas of the Cortex.

An examination of the following drawings (Figs. 75 and 76) from Ecker will furnish a sufficient knowledge of the gyri and sulci of the surface of the brain, which therefore need no further description in the text.

Functional Assignments.—The motor region is made up of the two central convolutions,—anterior central and posterior central, or ascending frontal and ascending parietal, the posterior part of the three frontal convolutions, the parietal lobule and the paracentral lobule (Fig. 76) on the median surface of the hemisphere. All diseases which destroy any considerable portion of this cortical area invariably result in paralysis of the opposite half of the body, while no matter how extensive the destructive processes elsewhere, motion remains intact if this is

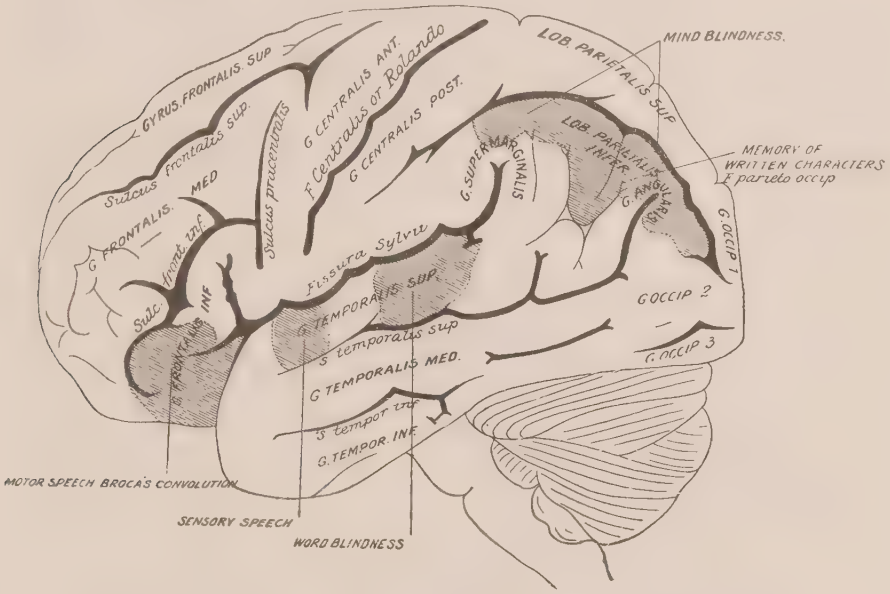


Fig. 75.—Lateral Aspect of the Brain.—(*After Ecker, modified.*)

not touched. It is more than likely, however, that an acute cortical lesion sufficient to involve all the centres and cause total hemiplegia would be fatal, while a lesser lesion, extending into the white matter, involving fibres coming from normal portions of the cortex, might produce a more extensive palsy than a purely cortical lesion.

We can even point out separate regions which act as separate centres for various groups of muscles. The centre for the movements of the facial muscles—lower division of the facial nerve—lies at the lower end of the central convolutions, and particularly of the anterior central convolution (Fig. 77). Near by and lower down is the centre for movements of the tongue and vocal cords, while the centre for the movements of the arm lies somewhat higher than that for the face, that is, about the

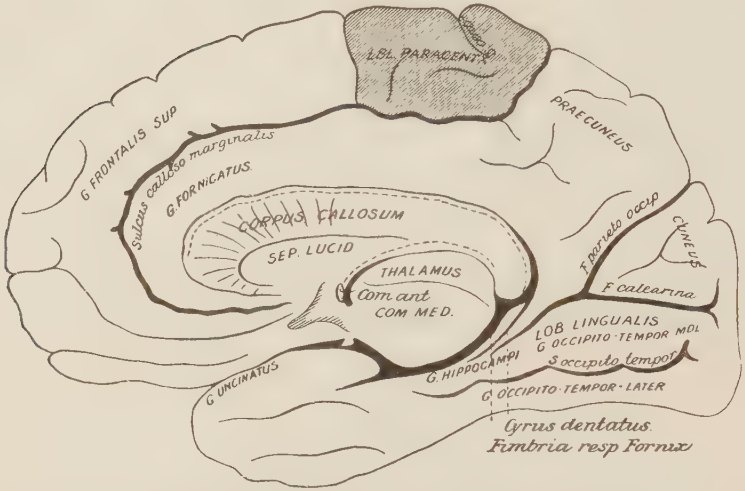


FIG. 76.—ASPECT OF THE MEDIAN SURFACE OF THE CEREBRUM, AS IT APPEARS WHEN THE TWO HEMISPHERES ARE SEPARATED.—(*After Ecker.*)
The gyri and fissures are indicated by the lettering.

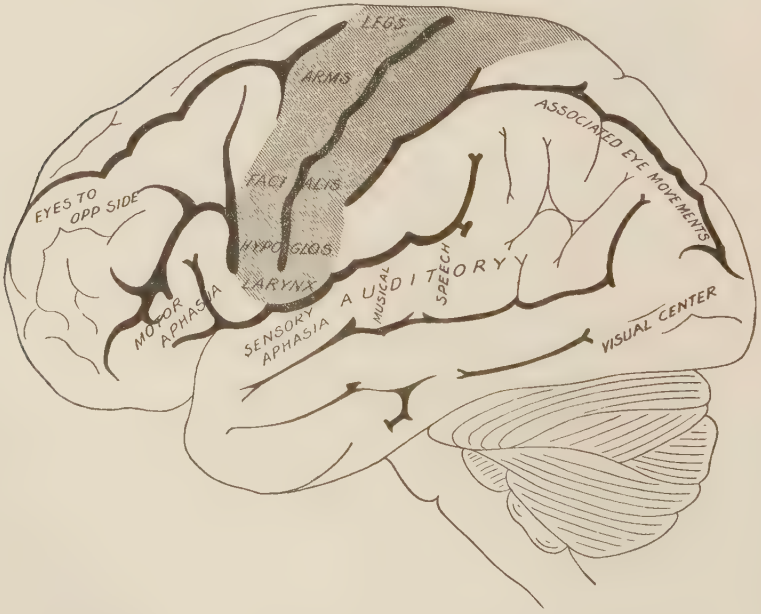


FIG. 77.—LATERAL ASPECT OF THE BRAIN.
Motor region of the cortex shaded and important centres indicated.

middle of the anterior central convolution. From above downward the various segments are represented as follows: Shoulder, elbow, wrist, fingers, the index finger, and, lowest of all, the thumb. The centre for the leg lies in the uppermost part of the central convolutions, but mostly in the paracentral lobule. Most anterior is the hip, next the knee and ankle, next the great toe, the centre for the movement of which surrounds the upper end of the fissure of Rolando; still further back are the centres for the small toes. The centres for the trunk are situated in the marginal gyrus just within the longitudinal fissure in the paracentral lobule. The different regions are not sharply defined, but merge into each other.

As to the muscular sense, the prevailing view has been that it resides also in the motor area, while there have been those who have claimed for it also a separate and different localization. Most recently M. Allen Starr and A. J. McCosh * have reported a case of injury with symptoms which go to prove the latter view and to show that the seat of the muscular sense is a "spot in the brain about at the junction of the superior and inferior parietal convolutions, clearly posterior to the posterior central convolution."

These cortical motor areas are united with spinal centres by nerve fibres which proceed from cell to cell in each without connection with intervening cells. Their route is through the white matter of the hemispheres, where they form the corona radiata, the fibres of which converge to the internal capsule which lies between the optic thalamus and the caudate nucleus on the inside, and the lenticular nucleus on the outside. The anterior portion of the capsule—the knee—is occupied by the fibres from the face-, tongue-, eye-, and speech-centres; behind these lie the fibres from the upper extremities, while those from the lower extremities occupy the middle of the posterior part. Thence the fibres of the motor path pass into the crus cerebri through its middle third, then through the pons, covered by the superficial transverse fibres of this body, into the medulla, of which they form the anterior or pyramidal tract. At the lower portion of the medulla a large portion of these fibres crosses over into the opposite half of the spinal cord, constituting the crossed pyramidal tract of the lateral column, while a small portion of fibres descends into the anterior column of the same side, forming the direct pyramidal tract, or Türck's column. Both pyramidal tracts diminish in bulk as they descend, because they give off fibres which pass into the grey matter between the anterior and posterior cornua, thence forward, dividing and subdividing, to join the network of protoplasmic processes which become continuous with the large nerve cells of anterior cornua.

These fibres form the *upper or cerebro-spinal segment* of the spinal cord. Between the motor-nerve cells in the anterior cornua and the muscles to which the motor-nerve fibres are distributed is the *lower*, or *spino-muscular segment*. In response to the law of the nutritive independence of each neuron, already announced, each of these segments has a certain nutritional independence, depending for its integrity upon

* Amer. Jour. of the Med. Sciences, November, 1894, p. 520.

the integrity of its neurons, the upper or cerebral depending upon the cortical cells and the lower upon the large cells in the anterior cornua.

Affections of the Upper Motor, or Cerebro-spinal, Segment.—If, therefore, the cortical cells of the motor area degenerate, the fibres attached to them will waste as far as the beginning of the lower segment, and if the cells in the latter degenerate or are cut off, not only do the nerve fibres below them waste, but as well the muscles to which they are distributed. Accordingly, all the cases of paralysis due to *destructive* disease in the motor cortical region have been found associated with descending degeneration of the motor tract above outlined, into the *direct*

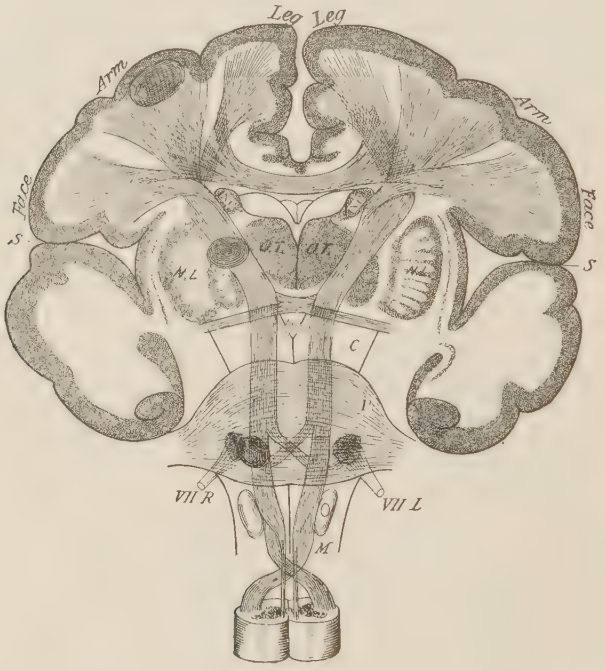


FIG. 78.—THE MOTOR TRACT.—(*After Starr.*)

S. Fissure of Sylvius. *NL.* Lenticular nucleus. *OT.* Optic thalamus. NC. Caudate nucleus. *C.* Crus. *P.* Pons. *M.* Medulla. *O.* Olivary body. The tracts for the arm, leg, and face gather in the capsule and pass together to the lower pons, where the face fibres cross to the opposite *VII* nerve nucleus, while the others pass to the lower medulla, where they partially decussate, to enter the lateral columns of the cord. The non-decussating fibres pass into the anterior median columns.

pyramidal tract in the anterior column of the cord on the same side, and the *crossed* pyramidal tract in the lateral column of the other side. At the same time the paralysis is accompanied by a spastic condition, manifested by an exaggeration of the tendon reflexes and an increase in the tension of the muscles, ascribed to a loss of the inhibitory control exerted by the cells of the cortex in the normal state. In other respects the paralysis due to cortical lesion does not differ from that due to focal

disease lower down in the upper tract, except that the latter is apt to involve more muscles because of its great compactness. Nor is there any degeneration of the muscles paralyzed by disease of the upper segment, and consequently no reaction of degeneration. Lesions of the cortex are usually limited, causing correspondingly limited paralysis and even monoplegias, never, however, affecting less than a whole limb or a segment of a limb. A lesion may involve two centres lying close to each other, producing paralysis of the face and arm or of the arm and leg, but not of the face and leg without involvement of the arm. It may happen, though very rarely, that the whole cortex is involved, producing paralysis of one side—cortical hemiplegia.

Such is the effect of destructive or negative lesion of the cortex. Quite different is that of *irritative* lesions. These produce convulsive seizures known as Jacksonian or cortical epilepsy, characterized by convulsions beginning in a single muscle or group of muscles and proceeding in a definite order to the involvement of other muscles corresponding to portions of the cortex affected. Thus the convulsions may begin in the face, and extend thence to the arm, and thence to the leg. The convulsions are also accompanied by sensory symptoms and followed by weakness of the muscles involved.

In point of fact, most lesions of the cortex are both destructive and irritative, consisting in the destruction of nerve cells in one centre and increasing the activity of cells of neighboring centres.

Lesions of the Lower or Spino-Muscular Segment.—Here, as in the upper segment, the *destructive* lesions produce motor paralysis. The added peculiarity is, however, a degeneration of the muscles as well as the nerve fibres distributed to them from the motor cells of the anterior cornua, as evidenced by the wasting of the muscles, and further characterized by the presence of the reaction of degeneration described on p. 806. In these lesions there is also a loss of reflex excitability in the areas supplied from the segments destroyed. Lesions of the lower segment may also cause paralysis of limited groups of muscles when confined to limited areas of the cord.

Irritative lesions of the lower segment do not occur unless we regard as the result of such the slow atrophy of the ganglion cells of the anterior cornua in progressive muscular atrophy, and consider the fibrillary contractions found in this affection as a result of the stimulation of these cells in their slow degeneration. They may also be caused by embolus of the axis cylinder processes in neuritis.

II. Sensory Areas of the Cortex and their Paths.

Our knowledge of the sensory areas is much less definite than that of the motor. Beginning at the periphery, we learn that sensory fibres emanating from tactile surfaces, like the skin, promptly and for the most part become associated with motor nerve filaments in the lower motor segment, the union of both constituting a mixed nerve. The two sets of fibres, however, separate again within the spinal canal, the motor filaments entering the cord by the anterior roots and the sensory by the

posterior, on which is a ganglion. The areas whence the posterior roots gather their nerves will be found in Starr's table on pp. 835–6. The precise routes of sensory impressions to the brain are not determined, but experiment and clinical pathology show conclusively that a considerable number of sensory fibres cross at once and ascend to the brain in the opposite half of the cord. The following seem to be the results of the latest histological studies :—

The sensory nerve fibres, entering the spinal cord from its spinal ganglion in the posterior root, pass to the postero-median column through the hinder part of the postero-external column and divide dichotomously, one branch passing upward, the other downward. From these longitudinal branches arise short transverse branches which penetrate the grey matter and end in the tufts or arborizations which characterize the distributary ends of nerve filaments. These tufts or arborizations in which the sensory fibre ends in the grey matter are apparently in close contact, but not in direct anatomical relation, with the ganglion cells in the anterior and posterior horns and in Clark's column, and from these ganglion cells other nerve fibres are projected, the course of which is not clear except as to those which pass into the anterior roots and those from Clark's column which pass over to form the ascending cerebellar tract. Some pass up the anterior lateral columns, a few decussate through the grey commissure with fibres from the opposite side. Finally there is a very rich decussation of sensory fibres in that part of the olivary body which enters the pons as the fillet or olivary fasciculus. These are in direct line with the fibres of the posterior columns of the cord to the brain above. Further confirmation of this course is found in the fact that if a posterior nerve root is cut the ascending Wallerian degeneration is seen only in the posterior column of the same side, and ceases in the grey matter where the the terminal arborizations occur, and no ascending degeneration is seen on the opposite side. The ascending degeneration again ceases after passing up the posterior median column in the nuclei of the funiculus gracilis and funiculus cuneatus, which are ganglionic bodies in the medulla apparently beginning another stage of the sensory path. These views allow of no separate strands of conduction for tactile, thermal, or painful impressions. The experiments of Gotch, Horsley, and Mott also go to show that mild sensory impressions pass up the same side in the posterior columns, while impressions made by pain, cold, and heat radiate into the grey matter of the cord, and through these probably again into the white conducting tracts of the lateral and, possibly, opposite posterior column. Hence pain and painful temperature sensations are only of different degrees, and excite a wider and more complex nervous mechanism than simple touch. Whence not only diseases involving extensively the grey matter, as syringo-myelia, cause alteration in the temperature sense, but also diseases of peripheral parts, as pachymeningitis and neuritis.*

* In view of the fact that histological studies of the spinal cord are attended with so much difficulty, it appears to me that their results must give way to the much more definite consequences of experiment confirmed by clinical observation, which go to show that the chief part of the sensory path crosses the middle line soon after it enters the cord and passes up to the brain on the other side.

All the sensory fibres of the opposite side of the body are collected in the posterior 3d of the posterior limb of the internal capsule, just behind the motor fibres of the upper segment, but much doubt exists as to the seat of the sensory areas in the cortex. Horsley suggested that the muscular and tactile senses are localized in the motor cortex, and that two of the three principal layers of cells in this region subserve these functions. The late experimental studies of Munk lead to the same conclusions that the so-called "sphere of sensation" lies in the same region as the motor centres of the cortex. Dana has also shown that many lesions of the motor area, especially in the hinder part, are associated with anæsthesia, while Ferrier considers the hippocampal convolution, and Schäfer the gyrus fornicatus, as the sensory centre in the cortex. Clinical evidence on this point is not uniform. In some cases of motor paralysis there is undoubted simultaneous disturbance of sensation, in others not. The muscular sense is also sometimes impaired in paralyzed limbs, in consequence of which the patient cannot tell with his eyes closed the position of the affected limb.

Among the cortical areas covering sensation must be included those for sight, hearing, smell, and taste, which will be considered in connection with affections of the peripheral nerves.

CORTICAL AREAS COVERING SPEECH.

THE VARIOUS FORMS OF APHASIA AND THEIR ANATOMICAL LESIONS.

It has already been stated that almost our first accurate knowledge of cerebral localization was the discovery by Broca, in 1861, that derangements of speech result from lesions of the 3d or inferior left frontal convolution. Such defects consist mainly in a loss of power to comprehend words correctly and to use them properly, and is covered by the general term *aphasia*. Further derangement consists in inability to articulate words, and is due to lesions of nuclei situated for the most part in the pons and medulla oblongata regulating the action of the vocal cords, the tongue, and the lips, and is known as *anarthria* or *dysarthria*.

The study of the phenomena of aphasia will be facilitated by a brief review of the conditions of acquired language. Language is acquired by the child gradually through imitation. Thus when the mother teaches it to say "cat" or "bell" or "papa" she names the word, and its sound impresses the distribution of the auditory nerve, *m*, whence it passes to the *acoustic* centre, *x*, and thence to the sensory *speech* centre, *a*, in the first or *upper temporal* convolution, where it is stored as a sound memory. From this it passes from behind forward along the association fibres to *b*, the motor speech centre in the left inferior *frontal* convolution (Broca's centre, propositionizing centre of Broadbent),* whence the muscles of

* That is, the centre where thoughts are sent in a framework of words, but through which utterance is not consummated; whence other cortical centres are necessary to motor speech, and these are found caudad of Broca's convolutions at the foot of the two central convolutions. This region Broadbent calls the *uttering centre*.

articulation are put into operation and the word is spoken. Thus the speech mechanism consists of *receptive*, *perceptive*, and *emissive* centres.

The development of *voluntary* speech in the child continues through the accumulation of associated ideas in the perceptive and emissive centres, *a* and *b*. The *word image* or picture which is the foundation of every word is made up of the sum of a number of partial conceptions or memory pictures acquired by experience and stored for further use in the different sensory areas of the cerebral cortex. Thus the memory of the *sound* of a word as spoken, the memory of the *appearance* of a word as written or printed, as well as the *muscular movements* needed to speak the word or write it, are distinct from one another and yet associated. Loss of one of these memory pictures or derangement in their association impairs the integrity of the word image and produces such defects

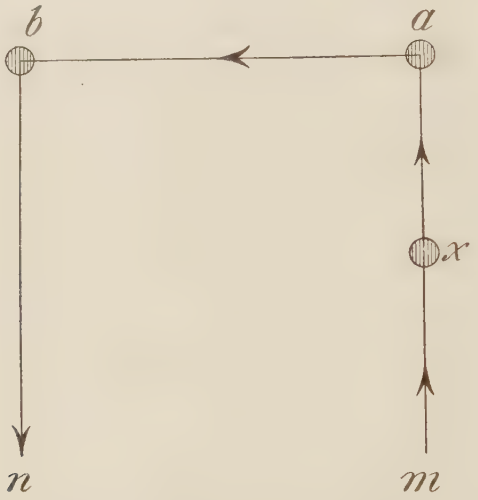


FIG. 79.—PRIMITIVE SPEECH APPARATUS OF THE CHILD IN MECHANICAL REPETITION OF WORDS, ACCORDING TO WERNICKE AND LICHTHEIM.

a. Sensory speech centre. *b*. Motor speech centre. *x*. Acoustic centre of pure sense of hearing. *mx*. Route to acoustic centre. *bn*. Motor speech tract.

in the use of the word as are covered by the different varieties of aphasia. These derangements have been arranged in two divisions, according as the defect is in the receptive and perceptive or emissive function of the brain, the former constituting the sensory aphasias, the latter the motor.

THE PHYSICAL BASIS OF THOUGHT—APRAXIA.

A *word* is a means of expression of a thought. Thus, when we say the word "bell" with a full conception of its meaning, such conception or mental picture is made up of as many distinct partial conceptions or memory pictures as there are special senses, these conceptions being seated in the most diverse parts of the brain. Especially concerned in the case of the bell is the acoustic conception, *c*, derived from its sound;

the optic, $c,'$ from its appearance; the tactile, $c,''$ from what is learned by touch, united to form one conception, as shown in Fig. 80, where the partial conceptions, c, $c,'$ $c,''$ among others, taken together, give us the idea of a bell. In the blind, of course, the sensory perceptions are smell, taste, touch, and hearing only. The schema of conscious voluntary speech may be still further simplified by combining the partial conceptions, c, $c,'$ and $c,''$ into one single point, C (Fig. 83), as the sum of intellectual concepts, m, representing any of the special senses—hearing, vision, smell, etc.

Broadbent has gone a step further, and suggested the existence of a centre to which converge sensory fibres from all the receptive centres and in which is combined all the evidence respecting the nature of the

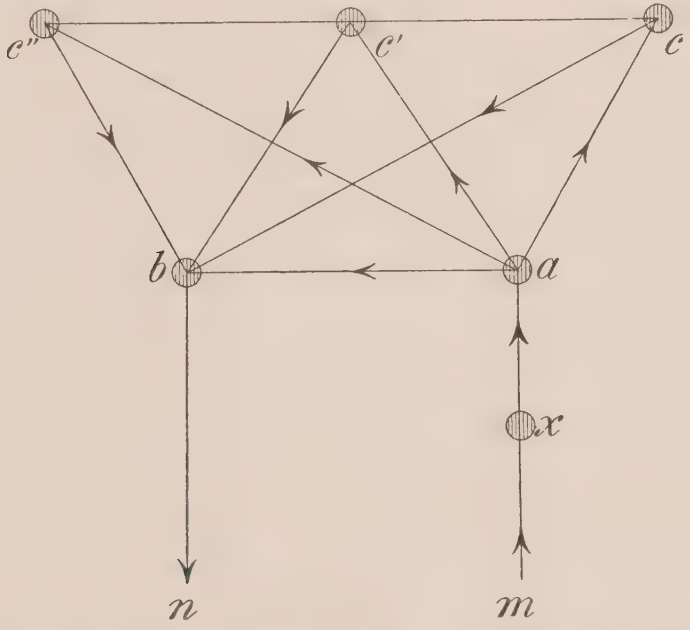


FIG. 80.—WERNICKE'S SCHEMA SHOWING THE ASSOCIATION OF THE VARIOUS PARTIAL CONCEPTIONS TO FORM THE WHOLE CONCEPTION OR WORD IMAGE OF AN OBJECT.

For the sake of simplicity, only three partial conceptions and three sensory areas are shown, instead of the many which go to make up our notions of complex objects. The letters a, b, m, and n have the same application as in the previous figure, but x may indicate the seat of any of the special senses—hearing, vision, smell, touch.

object, which he called the *naming centre*. He suggested a locality for this centre in an unnamed lobule on the under surface of the temporal lobe near its junction with the occipital lobe. Charles K. Mills * has recently reported a case with autopsy which goes to confirm Broadbent's speculation as to the exact position of this centre in the temporal lobe.

Now the loss of any one of these memory pictures is known as

* Dercum, "Diseases of the Nervous System," by American authors, 1895, p. 427.

apraxia, a condition in which there is impairment or loss of the power to discern the nature and purpose of objects, and which is not a part of aphasia. In one form of it any object, such as a watch, a knife, or a spoon, may be taken up and handled by the patient, but all knowledge of its use or purpose is gone. Such a condition was well named by Munk *mind blindness*, or psychical blindness. A person formerly familiar with the tick of a watch or sound of a bell no longer interprets such sounds aright. Or he is unable to follow melodies or appreciate music as he once did. Thus we have *mind deafness*, or auditory amnesia, or in the case of music, *amusia*. Again, the smell of the rose and violet no longer suggest these flowers, giving *mind anosmia;* or the taste of an orange, this delicious fruit, *mind ageustia;* or the soft feel of fur or velvet gives

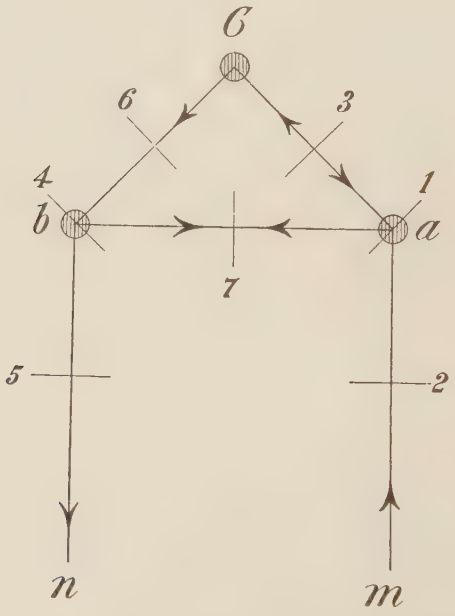


FIG. 81.—SIMPLIFICATION OF THE SCHEMA OF VOLUNTARY SPEECH BY UNITING THE IDEAS INTO THE POINT, *C*, AND OMITTING THE ACOUSTIC CENTRE, *x*.
The letters have the same meaning as in Figs. 79, 80.

no notion of these substances, *mind atactilia*. For any of these defects the term apraxia is now used, but mind blindness and mind deafness are the most important varieties. Apraxia may occur alone, but it is usually associated with sensory or motor aphasia. In simple apraxia the patient may be able to read, but the words arouse no intelligent impression in his mind. Numerous observations go to show that the lesion in mind blindness is in the supra-marginal and angular gyri, or in the tracts posterior to these in the white matter beneath them; as pointed out by M. Allen Starr, in the left hemisphere in right-handed persons and in the

right hemisphere in those left handed, *i. e.*, in the same hemisphere as that of which lesions produce aphasia. Mind-blindness is, however, at times functional and transitory, and, as such, associated with many forms of mental disturbance.

Starr also considers it probable from the association of psychical or *mind-deafness* with the form of aphasia known as *word-deafness*, to be considered presently, that it may be due to lesion in the upper temporal convolution, though autopsies are wanting.

There are as many varieties of apraxia as there are organs of sense, but the most common appears to be psychical or mind-blindness, generally associated with the form of aphasia known as word-blindness.

Aphasia, or Loss of the Faculty of Speech.

Sensory Aphasia; Word-blindness; Word-deafness; Amnesic Aphasia.—By *word-blindness* is meant loss of the memory of the appearance of a word. In this condition the patient does not recognize words which he sees on the written or printed page, and, although he may be able to pronounce them after others or write them at dictation or copy them, he does not understand what he reads or writes. On the other hand, *figures* are sometimes recalled when words are forgotten, and the patient may even be able to solve mathematical problems and to recognize playing-cards. Word-blindness may occur alone or with *motor* aphasia. The lesion in most cases of word-blindness has been in the angular and inferior parietal gyri on the left side, as located by Ferrier. *Alexia*, or inability to read, is a corollary growing out of this, as is also agraphia, or inability to write, so far as it depends on sight. It is often associated, as already stated, with *mind-blindness*, but may occur independently of it.

Word-deafness is a condition in which the patient has forgotten the significance of spoken words, though he hears them. The words of his own language are as a foreign tongue which he does not understand. Word-deafness is commonly associated with other forms of aphasia in various degrees, but cases of pure word-deafness occur in which the patient has been able to read and to speak. It is a rare variety of deafness whose lesion is placed by most students of the subject in the first temporal convolution or its posterior part, but Starr, basing his conclusion on 50 cases which he has collected with autopsies, places it, with Seppilli, in the posterior half of the *1st* and *2d temporal* convolutions of the left side in right-handed persons and of the right side in left-handed persons, as shown in the drawing.

A simple variety of sensory aphasia is *amnesic aphasia*, in which the patient simply forgets words—just as we are all, at times, at a loss for a word. Such a person sees a dog or other animal, knows perfectly well what it is, but cannot recall its name. The moment, however, the word "dog" is suggested he knows all about it. In disease usually a number of words are thus lost. Such aphasia is called *amnesic* because it is really a loss of memory of words. It may be partial, as when a

patient forgets nothing but his own name and remembers all other words, or when he is able to express himself in another tongue. If permanent, it is probably due to a break in the association tract, to be later considered, and should be so limited. Word-deafness or dumbness may be distinguished from amnesic aphasia by asking the patient to do some act, such as touching an object, when he will respond correctly if he has simple amnesic aphasia, but will not if he is the subject of word-deafness.

Allied to amnesic aphasia is sensory or *amnesic agraphia*, where a word cannot be written because it cannot be called to mind. A person thus affected may be unable to write voluntarily, but may be able to write at dictation if he is one who writes much. As already mentioned, agraphia also occurs as a part of word-blindness so far as it depends on sight. In motor agraphia writing at dictation, as well as voluntary writing, is lost.

Motor or Ataxic Aphasia or Aphemia.—Alalia.—In this condition the memory of the muscular action necessary to transfer the word image into speech is lost. There is disturbance of the emissive centre, *b*, in which this transfer takes place. The patient knows perfectly well what he wishes to say but cannot say it, though he may make the greatest effort to do so. Nor can he repeat a word after any one else. The degree varies greatly. In complete cases he may be able to read, though not aloud, and understand what is said, but cannot say a word himself. More commonly, he can say one or two words, such as "no," "yes," while in mild cases he may simply misplace or omit letters, say "widow" instead of "window," or "wrelstes" instead of "wrestles." Singularly, too, when in a passion he may be able to say the right word or to swear. This is because such words are uttered to a certain degree involuntarily. A man acquainted with the French and German languages may lose the power of expressing his thoughts in them while retaining his mother tongue, and if completely aphasic he may recover one language before the other. This is the form of aphasia long ago recognized by Broca and localized by him in the 3d left frontal convolution, and since this is in contact with the centre for the face and arm, there is not infrequently partial or complete right-sided hemiplegia. *Alexia*, or inability to read aloud, is a necessary corollary to motor aphasia so far as it depends on the power to speak.

Motor agraphia must also be distinguished from sensory. The latter is sometimes amnesic, that is, the patient cannot write the word because he cannot call it to mind; at others it is a part of word-blindness. Motor agraphia is quite independent of ability to read aloud, that is, of effort memories necessary to speech, the difficulty being connected with the movements of the hand. In the former case he may still be able to write on dictation, in the latter not. Agraphia also varies greatly in degree. The patient may write one or two letters or he may be totally unable to write voluntarily or from dictation. The seat of the lesion of motor agraphia is still unsettled. It was located by Charcot in the neighborhood of the middle of the anterior central convolution, but recent studies by Victor Horsley furnish some ground for locating it in the posterior central (ascending parietal) convolution, that is, in the

motor area. This is reasonable, because the motion of writing is like any other, and capable of being done by any part of the body, as the toes or even the nose. *Paragraphia* is a condition wherein one word is written when another is intended. It is a corollary to paraphasia, to be next considered.

Paraphasia, or *mixed* aphasia, and *monophasia* are allied to motor or ataxic aphasia. Paraphasia is a confounding of words, the wrong word being used instead of the right one, because of a confusion between the idea and the proper word. All degrees of this also occur, a single word only being sometimes erroneously used, while in others whole sentences are wrong. The patient may also use a wrong word which has a certain resemblance to the correct one, beginning, for example, with the same syllable, as "between" for "bewitch." Or the idea usurps the situation, as in the case of one of Strümpell's patients, who called a white handkerchief "snow." In these cases the association tract between the perceptive centre and the emissive centre is broken (see in Fig. 76), whence it was called by Wernicke *aphasia of conduction.* The lesion in paraphasia is usually in the island of Reil and in the convolutions which unite the frontal and temporal lobes. But any disturbance in the association processes of language, no matter where the break lies, may cause it. Thus, there may be paraphasia from a break in the posterior longitudinal bundle joining the occipital and temporal lobes, under which circumstances a patient can recognize an object when seen or a word when heard, but having seen the object cannot recall the word, and having heard the word cannot recall the object. In this case the lesion would be in the temporo-occipital white matter. In *monophasia* the patient can command but one syllable or one word or a short phrase, which he repeats over and over again.

Amimia is the loss or impairment of the power of expression by signs when caused by cerebral disease. *Paramimia*, the misuse of signs in the attempt to express thought, is comparable to paraphasia, for speech, and paralexia, for reading, and is dependent on a like cause—the destruction or impairment of commissural or association tracts between sensory and motor centres. It is not correct to suppose that the aphasic can substitute signs for words and thus express himself, for the two defects go hand in hand, even though he retain the power of moving his hands. A patient may, however, regain pantomimic power before he regains speech. Loss of pantomimic power is found often associated with destruction of the 3d left frontal convolution, but also in connection with loss of the naming or concept centres of the brain. It may accompany verbal amnesia due to disease of these areas or disturbance of the association tracts. Just as the aphasic may say "yes" when he means "no," so he may use a sign which will be affirmative when he intends it to be negative.

The following table may aid somewhat a review of the previous text, while the appended drawing from Starr's book of "Familiar Forms of Nervous Disease" shows the situation of the lesions causing aphasia:—

APRAXIA, inability to recognize the nature and purpose of an object.

		Seat of Lesion.
Mind blindness.		Supra m. and angular gyri, or the white matter beneath, in the left hemisphere in the right-handed and right hemisphere in the left-handed.
Mind deafness, including amusia, or auditory amnesia.		Upper temporal gyrus of left hemisphere in the right-handed.
Mind atactilia.		G. fornicatus, hippocampal g., præcuneus, and post-parietal (Mills).
Mind anosmia.		Uncinate gyrus (Ferrier).
Mind ageusia.		Temporal gyrus (Ferrier).

APHASIA, inability to comprehend words correctly and to use them properly.

Sensory Aphasia, inability to recognize word pictures, loss of memory of word pictures.	*Word* blindness, in which memory of the appearance of a word is lost.	Angular and inferior parietal gyri.
	Word deafness, in which memory of the sound of a word is lost.	Posterior part of 1st and 2d temporal gyri. (*Seppilli* and *Starr*).
	Amnesia, inability to recall a word.	Disturbance of association tract.
Motor Aphasia, inability to utter words, though knowing well what to say.	Including *alexia*, or inability to read aloud.	Posterior part of 3d left frontal (Broca's convolution).
Paraphasia.		
A confounding of words, in which the wrong word is used instead of the right one.		Any disturbance of the association tracts.
Amimia.		
Loss of power of expression by signs.		Third left frontal convolution, receiving or concept centres.
Paramimia.		
AGRAPHIA, inability to write.		Disturbance of association tracts.
Sensory Agraphia.		*Seat.*
Inability to write because of want of idea as to what a word is or looks like.	*a*. Amnesic agraphia.	Association tract.
	b. A part of word blindness.	Angular and inferior parietal gyri.
Motor Agraphia.		
Inability to write because of want of motor power.	A part of motor paralysis.	Not settled, but possibly middle of the ascending frontal convolution or ascending parietal.

Availing ourselves of Wernicke's condensed schema (Fig. 83), most aphasic derangements met with in practice are easily explained by it by supposing lesion and interruption of conduction in certain places. Accord-

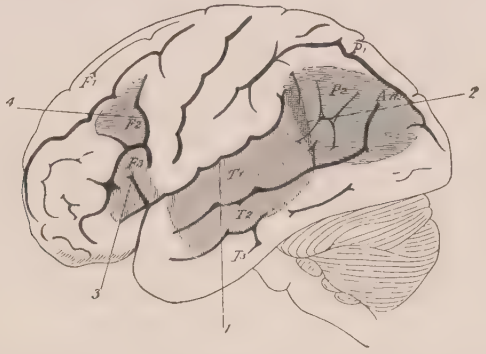


FIG. 82.—SITUATIONS OF LESIONS CAUSING APHASIA.—(*After Starr.*)
1. Lesion of word-deafness. 2. Lesion of word-blindness. 3. Lesion of motor aphasia. 4. Supposed lesion of agraphia.

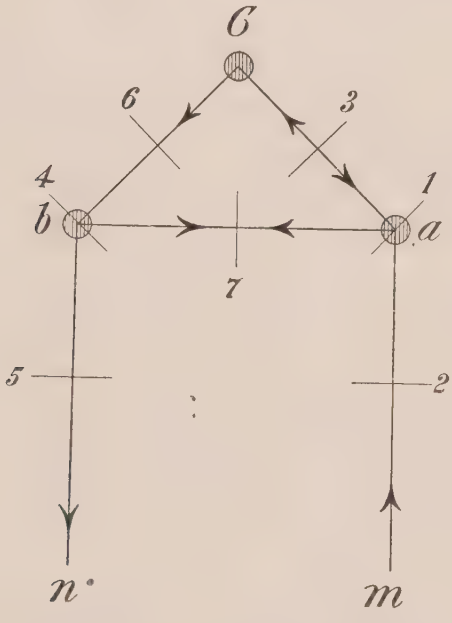


FIG. 83.—SIMPLIFICATION OF THE SCHEMA OF CONSCIOUS SPEECH BY REDUCTION OF THE IDEAS TO THE POINT, *C*, AND OMISSION OF THE ACOUSTIC CENTRE, *x*.
The letters have the same application as in previous figures. The numerals correspond to the numbering of the aphasias.

ing as the centripetal conduction, *m*, *a*, *C*, or the centrifugal conduction, *C*, *b*, *n*, is interrupted we have sensory or motor aphasia, while the interruption of the line, *a*, *b*, produces the conduction aphasia of Wernicke.

Lesions between *a* and *b* on the one hand and *C* on the other are called *transcortical* aphasias; between *a* and *b*, *cortical*, and between *a* and *b* on the one hand and the periphery on the other, *subcortical*.

Derangements of Speech of Irritative Origin.

In addition to those due to direct lesion of the speech centres there are also derangements of speech due to irritation. Such are the different kinds of stuttering, the labio-choreic and gutturo-tetanic stuttering and choreic speech. The first two probably reside in the cortical speech centres, but the choreic spasms not necessarily, since the muscles concerned in their production are un-nerved from other centres as well.

The study of derangements of speech is by no means an easy matter, but it may be facilitated by pursuing a systematic method like the following, which is that of Starr slightly modified:—

A. To determine whether apraxia is present.	(1) Test the power of recognizing the nature, uses, and relations of objects.
B. To test integrity of the auditory speech area and association tracts between other sensory areas and the temporal convolutions.	(1) The power to recall the spoken name of objects seen, heard, handled. (2) The power to understand speech and musical tunes. (3) The power to call to mind objects named.
C. To test the condition of the visual word memories in the angular gyrus and of the connections between this area and the surrounding sensory and motor areas.	(1) The power to understand printed or written words. (2) The power to read aloud and understand what is read. (3) The power to recall objects whose names are seen. (4) The power to write spontaneously and to write the names of objects seen, heard, etc. (5) The power to copy and write at dictation. (6) The power to read understandingly what has been written.
D. To test the integrity of Broca's centre and its association tracts.	(1) The power to speak voluntarily; and if it is lost, the character of its defects. (2) The power of repeating words one after another.

Where aphasia is associated, as is so often the case, with paralysis of the right arm the writing test may be made with the left hand, when the patient will often produce the so-called aphasic mirror writing, which can only be read by the use of a mirror. Or if he cannot write with the left hand, as also happens, he may be asked to form words by letters cut out of printed pages.

Prognosis and Treatment.—Aphasia is a symptom of a disease and not the disease itself. Yet it is a symptom which in its various phases informs us so precisely of the seat of the lesion that it sometimes suggests a point of operative interference comparatively easy of access. Where the symptoms of diffuse cerebral disease are wanting, and where the continuation of the symptoms and the addition of others suggest the presence of a possible circumscribed cause, such as abscess or tumor, operation is justified, and by it not only cortical but also subcortical lesions and abscesses have been relieved. In cases of *sensory* aphasia the

trephine should be applied over the *temporo-parietal region*, in *word-deafness* over the *posterior temporal*, in *word-blindness* over the *angular gyrus*, in word-deafness and word-blindness combined over the *inferior parietal*, especially if verbal amnesia be present. In purely *motor aphasia*, in which the understanding of language is preserved but the power of talking lost, the trephine should be applied over the posterior part of the *third frontal convolution*, or Broca's centre. The lesion of simple agraphia is not sufficiently determined to warrant surgical interference.

Where urgent symptoms do not exist, attempt should be made to reëducate the patient, and much may be accomplished in this way by perseverance, especially in the young. With adults, the prognosis is more unfavorable, especially in cases of complete motor aphasia associated with right hemiplegia. In them the patient may be taught to write with his left hand. Sensory aphasia, if it exist alone, is commonly transient.

Cortical Areas Whose Function is Unknown or Uncertain.

After subtracting the motor, visual, and speech areas of the two hemispheres, there remain extensive cortical areas, to which, as yet, no definite function can be assigned, and which are unexcitable. They include :—

(1) *The frontal region, including all the frontal convolutions except the posterior half of the 3d frontal on the left side.*—Of this area the most that can be said is that, if injured, mental symptoms are quite likely to be prominent—symptoms ascribable to a loss of self-control. It is to the greater development of the region of the frontal lobes in man as compared with the lower animals that his higher mental qualities are ascribed. Various forms and degrees of dementia have been observed after such lesions, and when such mental symptoms are present it may with reason be inferred that there is lesion of the frontal lobes, especially when lesion elsewhere can be excluded.

(2) *The region of cortex lying between the ascending parietal convolution and the occipital convolutions, including all the parietal convolutions except the left inferior parietal lobule.*—In these it was at one time thought might lie the centres of tactile sense, but the tendency at present is to make the sensory and motor areas of the cortex coincide, since lesions of the posterior central convolution are found associated with anæsthesia in the paralyzed part, and because the centres for the parts in which sensation is most acute,—namely, thumb, fingers, and hand, toes and feet,—lie in this region.

(3) *The region covering the entire temporo-sphenoidal lobe on the right side and the temporo-sphenoidal on the left side not concerned in speech.*—To the latter region the function of hearing is assigned, but the remainder, so far as the cortex is concerned, appears unexcitable. Abscesses are common here after otitis media, and are sometimes reached with the trephine, the diagnosis being based on the presence of otitis with symptoms of brain disease.

(4) The *apex of the temporo-sphenoidal lobe*, including the uncinate

convolution. To this the olfactory sense has been ascribed with some show of reason.

(5) *Of the entire median surface of the hemispheres*, except the paracentral lobule, which is motor, and the cuneus, which is visual, and including the gyrus fornicatus and the hippocampal cortex, the function is unknown.

Tracts Within the Brain—Centrum Ovale, Internal Capsule, Central Ganglia, Corpora Quadrigemina.

Centrum Ovale.—In the centrum ovale, constituting the mass of white fibrous substance beneath the cortex and above the level of the basal ganglia, the fibres of the motor paths are more or less closely associated with other systems of fibres. They include three sets—*projection*, *commissural*, and *association* systems; the first connecting the cortex with nervous structures lying below it, the second joining the two hemispheres, while the third, or association fibres, join different parts of the same hemisphere. By these fibres adjacent convolutions, alternate convolutions, and more distant regions are connected, and through these as a physical basis the activities of the various cortical areas are harmonized and the different memories united.

The diagnosis of lesions involving this mass is exceedingly difficult. We can only surmise in cases of disturbance of association, such as occur with aphasia and kindred disorders, that the association fibres have been destroyed. So, also, if simultaneously bilateral symmetrical motions are suspended, there must have been a break in the commissural fibres. A break in the continuity of the fibres of the corona radiata must produce the same symptoms as if the corresponding centre were destroyed. In like manner disease of the white substance of the occipital lobe may cause hemianopia, of the temporal lobe auditory disturbance such as word-deafness; and if the coronal fibres which proceed from the 3d left frontal convolution are diseased, motor or ataxic aphasia occurs. Yet quite extensive disease of the white substance of the frontal lobe has been found post-mortem without any symptoms having been present during life.

Internal Capsule.—Since in the comparatively narrow space in the anterior half of the posterior limb of the internal capsule is centred the pyramidal tract on its way from the cerebral convolutions to the crura cerebri, a very limited focal disease in this locality will lead to hemiplegia on the opposite side, while clinical experience shows that most cases of persistent hemiplegia are occasioned by disease in this spot. If the lesion involves the hinder 3d of the posterior limb, in which runs the sensory tract, including also fibres for the organs of special sense, there is also hemi-anæsthesia, often with involvement of the special senses.

A purely motor hemiplegia, therefore, unattended by impairment of sensation, implies a lesion that does not involve the most posterior portion of the internal capsule, while such involvement is probable when there is sensory disturbance as well as motor paralysis.

Central Ganglia, *i. e., Caudate Nucleus, or Corpus Striatum Proper, Lenticular Nucleus, and Optic Thalamus.*—In the writer's student days the

corpus striatum was regarded as the great motor ganglion and the optic thalamus as sensory, while most hemiplegias were ascribed to lesions of the former, and hemianæsthesia was ascribed to lesion of the optic thalamus. But while the optic thalamus has nothing to do with sensation or voluntary motion, it is conceded that it may have to do with the movements of mimetic or emotional expression, such as laughing and crying, which is lost in lesion of the thalamus constituting one form of *amimia* but which remains when the thalamus is intact, even though the half of the face is paralyzed and cannot be moved voluntarily. It is also likely that some of the fibres of the optic nerve terminate in its posterior portion, known as the posterior tubercle or pulvinar, while the remaining fibres go to the corpus geniculatum externum. Hence destruction of the hinder part of the thalamus produces complete hemianopia of the opposite side. Focal disease of the thalamus has also occasioned post-hemiplegic chorea and other post-hemiplegic symptoms of irritation. Recent experiments also go to show that the lenticular nucleus contains centres for regulating heat.

Beyond this, little definite is known of the effect of lesions strictly limited to the central ganglia, while disorganization of them has been found unattended by any symptoms during life.

Corpora Quadrigemina and Crura Cerebri.—Lesions of the *corpora quadrigemina* are rare. Not much, therefore, is known of their diagnosis. The anterior tubercles are connected with fibres of the optic nerve, and, if both are destroyed, total blindness results, while disorganization of one produces hemianopia. But as lesions elsewhere cause them, these symptoms cannot be regarded as pathognomonic of quadrigeminal disease. Unilateral or even bilateral paralysis of the oculo-motor nerve has been observed in connection with lesions of the quadrigeminal bodies, as have also nystagmus and immobility of the pupil. But this is because the nuclei of the motor nerves of the eyeball lie very close to the tubercles and may, therefore, be involved in such a lesion. According to Nothnagel a staggering gait with oculo-motor paralysis, associated with general symptoms of a tumor, points to the corpora quadrigemina as its site. The paralysis is apt to be of irregular distribution, involving upward and downward movements of the eye, and should be an early symptom.

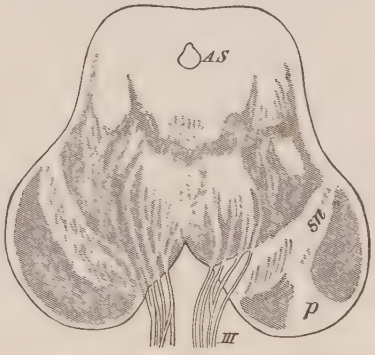


FIG. 84.—TRANSVERSE SECTION THROUGH THE CRURA CEREBRI IN SECONDARY DEGENERATION OF THE RIGHT PYRAMIDAL TRACT. *Sn. Substantia nigra. p.* The degenerated and therefore translucent pyramidal tract. III. Oculo-motor nerves. AS. Aqueduct of Sylvius.—(*After Charcot.*)

If the *crura cerebri* are diseased, there often result characteristic symptoms, viz., paralysis of one side of the body (arm, leg, and face), and on the side opposite the hemiplegia a paralysis of the motor oculi or 3d nerve—crossed paralysis. An examination of Fig. 84, from Charcot, will explain this. A lesion on the right side at *p*, in the right pyramidal

tract, might involve the oculo-motor nerve, III, on that side, but would produce a hemiplegia on the left side. Since the crura also contain sensory fibres from the opposite side, a lesion in one crus may also produce hemianæsthesia of that side. Tegmental lesions should also produce sensory paralysis.

Cerebellum.—The cerebellar hemispheres may suffer extensive lesions without symptoms, but if the central portion or vermiform process is injured two symptoms result which are quite characteristic of cerebellar disease, namely, *uncertainty of gait*, known as cerebellar ataxia and *vertigo*. The trunk and lower extremities alone are affected in cerebellar ataxia, standing, as well as locomotion, being interfered with. The patient can lie abed and move his legs almost as well as ever, but as soon as he rises he begins to sway back and forth with his whole body, even while standing. This tendency is increased if he bring his heels together, but is diminished while the legs are widely separated. In this respect cerebellar ataxia does not differ from that of the ataxia of posterior sclerosis. On the other hand, closing the eyes does not aggravate it because the cutaneous and muscular sensibility of the lower limbs remains normal. So, too, when he tries to walk he totters, but there is none of the stamping and pitching gait of tabes dorsalis. It is more the true drunkard's reel, at one time forward, and rolling now to one side and now to the other. Unfortunately, it is not so peculiar to cerebellar disease as to be pathognomonic of it, and we can only suggest that the middle peduncles of the cerebellum are involved. The upper extremities are almost never implicated, but Hughlings Jackson has called attention to a paresis of the trunk muscles as the result of which the movements of bending, erection, and lateral flexion of the trunk cannot be performed.

The *vertigo* of cerebellar disease, if severe, is one of the most distressing symptoms with which one can be afflicted. It varies greatly, and is not constant in cerebellar disease, while it may substitute the ataxia altogether. It occurs, however, under the same circumstances, that is, when the patient stands or moves about, disappearing when he lies down. The vertigo and ataxia are not associated, and one may be present and the other absent.

Headache is a frequent symptom in cerebellar disease, having been present in 83 out of 100 cases collected by W. C. Kraus. Most frequently it is occipital; more rarely there may be pain in the side of the head or forehead. *Vomiting* is also a result of chronic disorders of the cerebellum, being present in 69 of Kraus's cases. So is *visual disturbance* due to optic neuritis, which was found in 66 cases. None of these symptoms are pathognomonic, and each one may be a symptom of disease elsewhere in the brain. The most valuable is, perhaps, persistent occipital headache, especially if associated with the cerebellar gait. It might be expected that retained reflexes would be a distinctive sign as contrasted with their absence in tabes dorsalis. But, in fact, they are sometimes absent, this being the case no less than 12 times in Kraus's 100 cases.

Other symptoms which suggest cerebellar disease, but are not distinctive, are *neuralgic pains* in the region of the neck and occiput;

blocking of the venæ Galeni and dilatation of the lateral ventricles, producing in children hydrocephalus; pressure on the medulla oblongata, causing paralysis of the cranial nerves, glycosuria, or even sudden death if the vital spot is impinged upon; finally, bilateral rigidity from pressure on the motor paths. On the other hand, there may be cerebellar disease without any symptoms whatever, especially as long as the middle lobe is not involved.

Form of Lesion.—By far the most frequent cerebellar lesion is *tumor*—in fact, some sort of tumor was found in all except 12 of Kraus's cases, of which ten were *abscess*, and one each of *softening* and *hemorrhage*. The remainder were sarcoma and tubercle each 22, glioma 18, tumor of nature unspecified 13, cyst seven, and one case each of softening endothelioma, cyst and sarcoma, cancer, gumma, and fibroma. Thirty-two times the tumor occupied one or the other hemisphere, the middle lobe 17 times.

Disease of the middle cerebellar peduncles is accompanied by the so-called *forced positions* and *forced movements*. As a result of the former, the subject always lies in bed upon a particular side, whether conscious or unconscious, and if put on the other side he re-assumes his former position involuntarily. Sometimes this is accompanied by a corresponding forced position of the head and eyeballs, the extremities being seldom affected. The forced movements are less frequent. They consist either in oft-repeated rotations of the body on its longitudinal axis or, if the patient can walk, involuntary circular movements. There is no guide by which to determine which of the two peduncles is affected under these circumstances, while in a few cases of brain disease the same symptoms have been observed without involvement of the cerebellum.

The following very convenient summary from Strümpell's "Text-Book" contains the most important facts bearing on the localization of cerebral disease, and will be found useful for reference:—

"(1) The most frequent cause of ordinary hemiplegia is a lesion of the *pyramidal* tract in the posterior limb of the internal capsule. If the hemiplegia be persistent, then this tract is actually destroyed; if temporary, the tract has been functionally deranged for a time by focal disease in the neighboring parts of the brain.

"(2) Monoplegic cerebral paralysis is usually due to affections of the *cortex* of the brain, that is, the central convolutions and the paracentral lobule. Monoplegia of the face and tongue is the result of lesions in the lower extremity of the anterior central convolution. Monoplegia of the arm is referable principally to some lesion of the middle 3d of the anterior central convolution. Monoplegia of the lower extremity implies some affection of the paracentral lobule.

"(3) Hemiplegia or monoplegia, if associated with epileptiform convulsions affecting either one-half or one particular portion of the body, are almost always caused by cortical lesions. These same symptoms of motor irritation with accompanying paralysis are likewise ascribable to some irritation of the above-mentioned regions of the cortex.

"(4) Hemiplegia with crossed paralysis of the oculomotor nerve indicates a lesion of the crura cerebri.

"(5) Hemiplegia with crossed facial paralysis implies, with an approach to certainty, that the lesion is situated in the pons.

"(6) Post-hemiplegic chorea (*vide infra*) seems to occur especially where there is focal disease in the neighborhood of the optic thalamus or of the posterior part of the internal capsule.

"(7) Hemianæsthesia (of the skin and of the organs of special sense) seems to result principally from lesions of the most posterior portion of the internal capsule.

"(8) Hemiopia may be due to a lesion of the occipital lobe. Probably, also, a lesion of the posterior extremity of the internal capsule may cause it, in which case it is usually associated with hemianæsthesia. Finally, it may be produced by affections of the posterior tubercle of the optic thalamus, or of one of the anterior corpora quadrigemina, or of one of the optic tracts.

"(9) Genuine motor aphasia indicates disease of the 3d left frontal convolution.

"(10) Word-deafness seems to be due to disease of the 1st left temporal convolution.

"(11) Difficulty in articulation implies disease of the medulla, as does also dysphagia.

"(12) Staggering gait and vertigo are the most constant symptoms of cerebellar disease. Forced positions and forced movements are seen chiefly in connection with lesion of the crura cerebelli ad pontem.

"(13) Staggering gait and ocular paralysis are indicative of lesions of the corpora quadrigemina."

DISEASE OF THE CRANIAL NERVES.

OLFACTORY NERVE.

The olfactory nerve may be affected in various parts of its course and distribution, at its central origin in the uncinate gyrus, in its trunk, in the olfactory bulb itself, and in its distribution to the olfactory region of the nose.

The effect of lesions in any of these localities may produce subjective sensations of smell, or *parosmia*, of which various foul odors are illustrations; hypersensitiveness of the normal sense, or *hyperosmia*, in certain extremes of which the patient, generally a highly sensitive woman, can distinguish one person from another; or loss of the sense of smell, *anosmia*.

The lesions may be tumors of the brain, instances of which have been found in the hippocampal lobules, or disease in the hemispheres. There may be congenital defect of the olfactory centre or atrophy of the nerve, which may explain the occasional anosmia in locomotor ataxia. There may be inappreciable changes, caused by injuries to the head or by concurrent disease such as epilepsy, the aura of which is some-

times manifested by parosmia. The area of distribution of the olfactory nerve in the nose may be destroyed by chronic nasal catarrh or polypus. Hysterical neuroses of the olfactory are not infrequent.

Diagnosis.—The nasal region should be carefully explored by the rhinoscope and the sense of smell should be tested. For this purpose the essential oils, such as aniseed, cloves, or peppermint, in various degrees of dilution, are employed. Cologne water, musk, or asafœtida may be used for the same purpose. Pungent substances should be avoided, as they stimulate the 5th nerve in the nasal mucous membrane and thus the subject perceives what he does not smell. By such agents the 5th nerve is tested. No conclusion can be drawn as to anatomical differences on the two sides without a rhinoscopic examination.

Treatment is useless, except the condition be due to curable or removable polypus.

OPTIC NERVE AND TRACT.

There may be derangement of the retina, of the optic nerve, of the chiasm and the optic tract.

(1) Affections of the Retina.

These may be *organic* or *functional.*

(*a*) *Organic Disease.*

The organic affections include inflammation and hemorrhages, or both.

Hemorrhage into the retina occurs in Bright's disease, most commonly chronic interstitial nephritis, in gout profoundly affecting the system, in leucocythæmia, anæmia, syphilis, purpura, ulcerative endocarditis, and other forms of septicæmia. The hemorrhages are in the layer of the nerve fibres. At first bright red, and then becoming darker and eventually lighter in color, they ultimately assume a diffuse cloudiness due to serous infiltration. The hemorrhages vary in extent, and often follow the course of the vessels. In septicæmia they are due to capillary septic embolism and often have white spots in the centre due to the massing of leucocytes. Other white spots are due to fibrinous exudate, fatty degeneration of the retinal elements, or localized sclerosis of the same. Similar hemorrhages sometimes occur in the pia mater in the same cases.

Retinitis occurs under the same circumstances as hemorrhage, especially in chronic nephritis, syphilis, anæmia, leucæmia, and also malaria; diabetes mellitus, purpura, and chronic lead poisoning. Albuminuric retinitis may occur in all forms of chronic nephritis, more frequently in the interstitial variety, of which disease it may be the earliest symptom recognized. It is characterized in general by the presence of white spots of various extent and distribution, as seen by the ophthalmoscope. They

are caused by degenerative processes and hemorrhages. Gowers recognizes three forms :—

(1) A degenerative form, which is the most common, in which there are retinal changes, but scarcely any alteration of the optic disc.

(2) A hemorrhagic form, in which there are many hemorrhages, but slight evidence of inflammation.

(3) An inflammatory form, in which there is much swelling of the retina and obscuration of the optic disc.

In some instances of the latter the inflammatory changes in the optic nerve predominate over those of the retina, producing an *opto-neuritic* form, in which the appearances are more allied to those of papillitis or choked disc, such as is caused by intra-cranial disease.

Syphilitic retinitis is a rare affection in acquired and congenital disease. In the latter it is called *retinitis pigmentosa*. Syphilitic choroiditis is less rare. Retinitis is not uncommon in chronic anæmia, especially in the pernicious form. After excessive loss of blood the patient often becomes blind, either suddenly or in the course of one or two days. In such cases a neuro-retinitis has been found quite sufficient to explain the blindness, which, in rare instances, may be permanent and complete. A rare variety of anæmic retinitis is malarial retinitis, first described by Stephen MacKenzie. It may be associated with hemorrhage. In leucæmic retinitis the retinal veins are large and hemorrhage may also occur, with white and yellow areas.

(*b*) *Functional disturbances of the retina, or amauroses.*

This may be toxic. Of this the most striking and best known variety is *uræmic*. Its suddenness is its most striking feature, and it is very frequently the forerunner of uræmic convulsions, although it may occur without them. It, too, may be the first symptom noted in Bright's disease. The retina is free from changes visible by the ophthalmoscope and the condition is probably due to the action of the poison on the nerve centres. It generally disappears, not quite as suddenly as it comes on, but comparatively quickly, while the impaired vision of retinitis albuminurica is more or less permanent. Similar is the amaurosis from *lead poisoning* and massive doses of *quinine*. *Hysterical* amaurosis is more frequently a dimness of vision—amblyopia—but true blindness may occur in one or both eyes. *Tobacco* amblyopia is usually gradual in its appearance, and affects more especially the centre of the field of vision. There may be congestion of the optic disc, and if the use of tobacco is persisted in there may be a permanent organic change with atrophy of the discs. A scotoma for red and green is invariably present.

In *nyctalopia*, or night-blindness, objects are clearly seen by the day or strong artificial light, but are invisible in the shade or twilight. In *hemeralopia* the reverse state of affairs exists, objects being seen with discomfort in bright daylight or strong artificial light, but are easily seen in deep shade or twilight. Retinal hyperæsthesia is sometimes met with in hysterical women, but is not frequent in true retinitis.

(2) Affections of the Optic Nerve.

Those which are of medical significance are *optic neuritis*, or choked discs, and *optic atrophy*. The intra-cranial trunk of the nerve is rarely affected by reason of its shortness. It may, however, be compressed by a tumor in adjacent parts, as of the pituitary body or of the bone; by aneurism of the ophthalmic artery within the orbit or internal carotid within the skull. The trunk may also be the seat of inflammation which may extend from carious bone or meningitis, or it may be rheumatic.

(*a*) *Optic Neuritis, Papillitis, or Choked Disc.*

Definition.—Inflammation of the intra-ocular end of the optic nerve.

Anatomical.—It will be remembered that the optic nerve pierces the sclerotic and choroid coats at about $\frac{1}{10}$ inch (2.5 mm.) to the nasal side of the centre of the retina, which is occupied by the yellow spot of Sœmmering. In this spot the sense of vision is most perfect, while the optic papilla or disc with its central "cup" is the only part of the retina from which the power of vision is absent. The central cup is due to the separation of the nerve fibres, which leaves a central hollow, pale because of the absence of blood-vessels, while the periphery of the disc has a rosy tint from the presence of the minute blood-vessels that lie among the nerve fibres. The "cup" varies in size and may be absent, the vascular portion of the disc at times extending over it. The tint of the vascular portion of the disc also varies, and differences are only of significance when noted at successive examination of the same case.

Morbid Anatomy.—It is by swelling and diminished transparency rather than by recognizable signs of congestion that the first stage of optic neuritis is characterized. Then there follows lessening of the sharpness of the edge of the disc, and finally its total obscuration, as seen in the right half of Fig. 85 as contrasted with the left half. It is to be remembered that the normal contrast is sometimes diminished within the limits of health with this difference, that the pathological indistinctness is better seen with the direct method of examination, while the indistinctness sometimes normally present is more evident to the indirect. The abnormal change is earlier recognized on the nasal side, because there are more nerve fibres there than at the temporal edge. In the second stage the swelling rapidly increases and the whole circumference of the disc disappears, though the cup is still represented by a slight depression. The swelling extends even beyond the normal disc, becoming two and three times as wide. The swollen disc assumes a red or greyish-red color to the indirect examination, but by the direct a fine striated appearance is noted, the striæ radiating from the centre of the disc and corresponding to the course of the fibres. White spots may appear on its surface, due to degeneration of the nerve fibres, and may be seen in the figure. As the swelling increases the retinal vessels, at first unaffected, suffer from the compression, the veins becoming wider and tortuous, the arteries narrowed or remaining normal while hemorrhages may occur. The retina may also be invaded, producing a neuro-retinitis.

In very slight degrees of inflammation the swelling subsides and recovery takes place. In high degrees it remains for a long time, owing to the presence of inflammatory products, which gradually, however, undergo the usual contraction of cicatricial tissue, and a condition of "consecutive atrophy" results, in which the disc is white and atrophied.

Etiology.—Most commonly optic neuritis is caused by intra-cranial disease, especially tumor, in nine-tenths of all cases in which it is said to be present. It gives no information as to the seat of the tumor. It is now generally ascribed to a descending neuritis and not as formerly to intra-cranial pressure. In over 90 per cent. of cases the neuritis is bilateral, though often unequal in the two eyes. Unilateral neuritis is generally due to disease within the orbit or at the optic foramen. Meningitis, either tubercular or simple, is the next most frequent cause. It is said to be rather more common in meningitis of the base than of the convexity. Such neuritis is less severe than that caused by tumor. Cerebral abscess may cause it; so may diffuse cerebritis. When unilateral it is generally on the side opposite the lesion. In thrombotic softening and hemorrhage optic neuritis is rare, but in embolic softening it is more common.

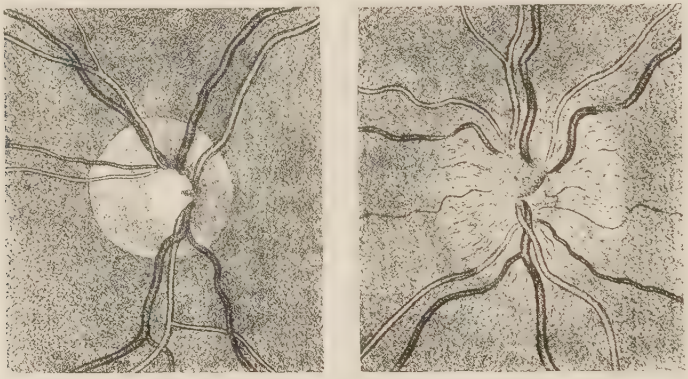


FIG. 85.—COMMENCING OPTIC NEURITIS FROM A CASE OF CARIES OF THE SPHENOID BONE WITH SECONDARY MENINGITIS.—(*After Gowers.*)

The left figure shows the normal right optic disc with clear outline and deep central cup. The right figure of the left papilla shows well-marked neuritis; the edge of the disc is concealed by a swelling which extends beyond the normal limits of the disc. The central cup is encroached on, but not yet quite obliterated. Some of the vessels are partly concealed at their emergence, and the veins lose their central reflection.

Optic neuritis may result from chlorosis, Bright's disease, anæmia, lead poisoning, after acute fevers, especially scarlet and typhoid. In the latter it may be associated with brain symptoms, especially headache. About six per cent. of all cases of multiple sclerosis are accompanied with optic neuritis, due to inflammatory or sclerotic patches in the nerve, usually slight and of short duration, often one sided in consequence of unilateral involvement of the nerve by a sclerotic patch.

Symptoms.—Mild degrees of optic neuritis may be without symptoms except such as are revealed to the ophthalmoscope. With higher degrees, acuity of vision, color vision, and the visual field all suffer and may be lost. Its severest effect on vision may not appear until contraction sets in, because it is at this period that the nerve elements suffer most in integrity. The defective sight is not, however, necessarily due to changes in the disc or retina; it may be due to intense inflammation in the nerve behind the eye or to intra-cranial disease.

Prognosis.—Even in severe cases there may be some improvement of vision with subsidence of the inflammation. On the other hand, vision may be permanently lost.

(*b*) *Optic Atrophy.*

There are three varieties of atrophy of the optic nerve:—

(1) Primary.
(2) Secondary.
(3) Consecutive.

Primary or *simple* atrophy is that form which is not preceded by any recognizable inflammatory change in the papilla or surrounding structures. It occurs in degenerative diseases of the brain and spinal cord, more frequent in multiple or disseminated sclerosis and locomotor ataxia. It is present in about 40 per cent. of cases of multiple sclerosis, and in various degrees in at least 15 per cent. of tabes. In dementia paralytica it is present in about five per cent. Primary atrophy is sometimes hereditary, occurring only in the males of a family after puberty. Other causes to which the condition has been ascribed are cold, alcohol, lead, sexual excesses, diabetes, and the specific fevers.

Secondary atrophy is the result of damage to the optic nerve behind the eye or at the chiasm. It is characteristic of it that demonstrative signs of atrophy follow instead of accompany the deranged vision, of which, too, hemianopia may be a form.

Consecutive atrophy is that form of atrophy which succeeds neuritis or papillary neuritis. It has the same causes and the same significance. Secondary and consecutive atrophy are alone the result of uncomplicated intra-cranial disease, for although primary atrophy accompanies disseminated sclerosis, locomotor ataxia, and general paralysis of the insane, it is not caused by the associated brain disease, but is the result of the same widespread tendency to degeneration.

The ophthalmoscopic appearances in primary atrophy differ somewhat from those of consecutive and secondary, the disc being grey-tinted,—whence the name *grey atrophy*,—with its edges well defined, while the arteries appear almost normal. In secondary and consecutive, the disc has an opaque, white appearance, with irregular outline, and the arteries are small.

The **symptoms** of optic atrophy are the defects of vision already detailed when treating optic neuritis.

As to **prognosis**, in primary atrophy the ultimate result is usually blindness, but in secondary and consecutive atrophy some vision remains, even in severe cases, while in mild cases recovery is not impossible.

(3) Lesions of the Chiasm and Tract.

Anatomical.—The decussation of the optic tracts at the chiasm is peculiar. Each tract as it reaches the chiasm divides and sends a portion —the smaller—of its fibres to the temporal half of the corresponding retina, and the remaining portion to the nasal half of the opposite retina. Thus the right tract supplies the right or temporal half of the right retina and the right or nasal half of the left retina; the left tract supplies the left or temporal half of the left retina and the left or nasal half of the right retina. The decussating fibres occupy the middle of the chiasm, and the direct fibres the corresponding side. (See p. 931.)

Effect of Lesion of the Chiasm: Hemianopia.—(*a*) If the central portion of the chiasm, composed of decussating fibres only, is involved (lesions *b* and *c*), the result will be anæsthesia of the inner half of each retina and blindness of the outer half of each field of vision, it being remembered, of course, that the half field which is blind is the reverse of the half of the retina which is anæsthetic, since the picture formed on each half of the retina is projected to the opposite half of the field of vision. Such half blindness is known as *hemianopia*, and the form just described, in which the outer or temporal half of each field is blind, is known as *temporal* hemianopia.

(*b*) If the whole chiasm is involved, as is not infrequently the case as the result of pressure by tumors, there will, of course, be total blindness.

(*c*) If the lesion is intermediate, involving the direct fibres on one side of the chiasm as well as the central fibres, there will then be blindness in one eye and temporal hemianopia in the other.

(*d*) The rarest of all forms of hemianopia is *nasal* hemianopia, due to a symmetrical lesion involving only the direct fibres passing to the temporal halves of the retina, whence results blindness in the nasal field only. It is found sometimes in tabes dorsalis and in tumors involving the outer part of each tract.

Effect of Unilateral Lesion of the Tract.—If there be a lesion involving the left tract at *d*, the left or temporal half of the left retina and the nasal half of the right retina become anæsthetic and useless, the right half of the field of vision is blotted out, and there results a right *lateral hemianopia*, which is called homonymous hemianopia. The reverse is the case if the lesion is in the right tract. The number of cases involving the right side is about equal to the number involving the left. When the left half of one field and the right half of another are blind, or the reverse, the condition is known as *heteronymous* hemianopia.

In the usual forms of temporal hemianopia the obscure fields are by no means always exact demi-fields, which would be the case if the dividing line passes exactly through the fixing point, or macula lutea. It may diverge to the temporal side so as to leave a small area around this within the seeing half. These differences are due to peculiarities in the decussation rather than to lesion. The half fields which remain, though commonly natural, are sometimes contracted. This is usually due to an inflammatory affection of the peripheral fibres of the optic nerves in front of the chiasm.

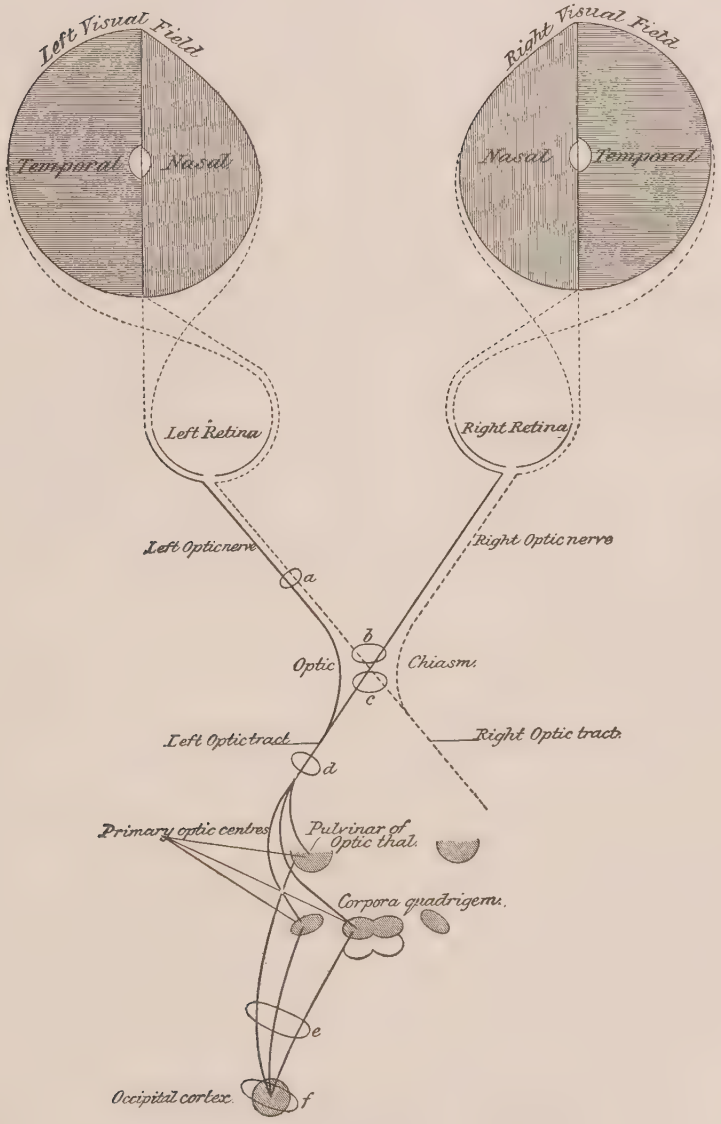


FIG. 86.—DIAGRAM OF COURSE OF OPTIC NERVE FIBRES, FROM THE CORTEX TO THE RETINA.—(*After Sahli, Modified and Extended.*)

There are other differences in the dividing line, such as obliquity, want of sharpness, etc., due to the same cause, but minute description of these belongs to special works on nervous diseases. Since the inclusion of the central region in the seeing half of the field occurs, equally in right- and left-sided hemianopia, it follows there must be a passage of fibres from the macular region to the optic tract of each hemisphere, else this region would be blinded by disease of one or other tract. There is usually the same loss of vision for color in the half field, but half vision for color may be lost in central disease without any change in the field for white. This is known as hemiachromatopia.

(4) Lesion of the Tract and Centres.

The optic tract on each side crosses the crus cerebri backward to the posterior part of the optic thalamus, where it divides into two unequal portions, an outer and inner. The *outer* and larger division sends fibres to the pulvinar of the optic thalamus, the external geniculate body, and to the anterior quadrigeminal body. Thence it passes backward through the posterior part of the internal capsule, forming the fibres of the optic radiation in the white substance of the occipital lobe, into the visual area of the cortex, of which the cuneus is the chief cortical centre, though other parts of the occipital cortex also receive and store up visual impressions. The *inner* portion, containing probably only intertractal fibres, passes to the internal geniculate and the posterior quadrigeminal body.

Whence it is plain that vision may be influenced by lesions in any of the following situations :—

(1) In the tract itself.

(2) In the optic thalamus and tubercula quadrigemina, into which the larger part of each tract enters.

(3) In the fibres passing from the tubercula quadrigemina to the occipital lobe, as at *e*, in the hinder part of the optic radiation.

(4) In the cuneus, *f*; and, finally,

(5) In the angular gyrus, as shown by clinical observation confirmed by autopsy.

The effect of lesion in any one of these situations is to produce anæsthesia of half of the retina corresponding to the affected side and a homonymous hemianopia of the opposite half of the visual field.

Morbid States Affecting the Optic Nerve, Chiasm, Tract, and Centres.—Outside of the affections of the retina, which concern the ophthalmologist chiefly, and outside of optic neuritis or papillitis as a result of intra-cranial disease already considered, the affections of the *optic nerve* which concern the physician are tumors springing from the pituitary body or the bone, aneurism of the ophthalmic artery within the orbit or of the carotid within the skull, interstitial inflammation from adjacent focus, or rarely from rheumatism and injury.

The *optic chiasm* is encroached upon by tumors in the neighborhood, especially on the pituitary body, by tubercular or syphilitic growths in its substance, or by inflammation invading it from the adjacent dura mater or carious bone ; by internal hydrocephalus, the distended infundi-

bulum of the 3d ventricle pressing on the middle of the chiasm; interstitial inflammation of a possible gouty origin or associated with spinal tabes; and, finally, interstitial hemorrhage.

The *optic tract* may be invaded or compressed by tumors springing from the inner part of the temporo-sphenoidal lobe, by softening in the lenticular nucleus, or softening after thrombosis of the internal carotid, or by disseminated sclerosis. Primary softening in the tract is rare, as is also hemorrhage.

The *cortical visual centres* may be invaded by hemorrhage, softening tumors, pressure by depressed bone in fracture, and other traumatic causes.

Symptoms of Lesions of the Optic Nerve, Chiasm, Tract, and Cortex.—

1. *Visual Effects.*—

(*a*) Lesions of the optic nerve cause defects of vision on the same side, with lessening of the reflex action of the pupil proportionate to interference with vision. The impairment of vision includes extent of field of vision as well as degree. There may be concentric limitation of the visual field because the peripheral layer of nerve fibres near the optic foramen is damaged by processes external to it. In other cases there is irregular defect, and in others still the loss of sight is total and lasting.

To the ophthalmoscope there may be at first no change, but if the lesion is considerable the atrophic condition soon makes its appearance "secondary" to changes in the nerve, as distinguished from "consecutive" atrophy which succeeds papillitis. There may be slowly supervening atrophy without recognizable papillitis. Central loss of vision, due to axial neuritis, is less common, but occurs sometimes in tobacco amblyopia.

(*b*) In lesions of the chiasm the characteristic symptom is temporal hemianopia, or loss of the outer half of each field of vision; this is because they mainly affect the chiasm at its central portion where the fibres, after decussating, pass to the nasal half of each retina. Usually, however, the process, be it tumor or inflammation, which causes temporal hemianopia extends laterally, involving the non-decussating fibres of one side of the chiasm, causing total blindness of the corresponding eye, or if extending to both sides blindness of both eyes. These different stages may often be traced in a single case as the disease progresses. The term "oscillating bi-temporal hemianopia" is applied to a rapid and frequent variation of the dark fields, and is regarded as more or less diagnostic of basal syphilis, such as gumma or syphilitic meningitis. More rarely we have the nasal hemianopia already described. Slight variations in the extent of the dark fields have been referred to as the result of peculiarities in decussation rather than of lesion or seat of lesion.

(*c*) In lesions of the optic tract between the chiasm and geniculate bodies there is lateral hemianopia.

(*d*) Lateral hemianopia is also a result of lesion of the central fibres of the nerve between the geniculate bodies and cerebral cortex.

(*e*) Lesions of the cuneus cause lateral hemianopia. A lesion in

each hemisphere, affecting the visual path back of the chiasm, will cause a double hemianopia, with total loss of vision in both eyes. Such a result has followed successive lesions in the two occipital lobes.

Transient hemianopia often occurs at the onset of cerebral hemorrhage in consequence of general interference with the functions of the cerebrum.

(*f*) Lesion of the angular gyrus may be associated with hemianopia, crossed amblyopia, and mind blindness.

(*g*) Hemianopia may be due to functional disease. Transient hemianopia is sometimes a symptom of migraine, either as an isolated symptom apart from headache and gastric disturbances or associated with them. It may affect now one half the field and now another.

Hemianopia is also rarely a symptom of hysteria.

2. *Other Symptoms Associated with Hemianopia.*—In about one-half of the cases of hemianopia there is transient or permanent hemiplegia, the result of the same lesion, the hemiplegia being on the side of the loss of vision, so that the patient cannot see the paralyzed side. Hemianæsthesia may also be associated, and defects in speech are sometimes found in right-sided cases.

Hemiachromatopia has been alluded to (p. 932). In this condition there is no change in the field for ordinary objects, but all colors appear grey as soon as the vertical line is passed. The symptom, according to Gowers, probably depends on disease of one part of the occipital lobe, and is proof of a separate centre for color not yet precisely located, perhaps in some part of the occipital cortex in front of the apical region.

The limitation of the remaining functionally active half field and the isolated loss for colors are the only known differences in the features of hemianopia due to differences in the seat of the lesion in the optic path behind the chiasm, the former indicating a lesion in the optic radiation near the thalamus, the latter in the occipital lobe.

3. *Amblyopia.*—Amblyopia is another form of sight defect due to brain disease. The term is used to indicate a *partial loss or blurring of vision.* The amblyopia of brain disease is crossed,—*i. e.*, there is dimness in the sight of the eye opposite the cerebral lesion, which is generally in the lower and posterior part of the parietal lobe,—the angular and supramarginal gyri. There is concentric limitation of the visual field, different in different cases, and along with it the color fields are also reduced. While not noticeable to the patient, careful examination will also discover a slight reduction in the field of the normal eye, not involving the color fields.

Similar eye defects, associated with hemianæsthesia, occur sometimes in hysteria, with which it may be confounded. From the amblyopia of optic-nerve disease that of hysteria is, however, distinguished by the absence of anæsthesia. As a simple functional loss of vision may rarely result as a reflex from irritation of the 5th nerve or from hysteria, so a functional amblyopia, affecting both eyes, may also result from such causes—indeed, is more common than the organic form. A carious tooth may act in this way. Amblyopia from errors of refraction must not be confounded with the amblyopia due to brain disease.

Diagnosis.—How shall we interpret these phenomena of vision concerned with the optic nerve and tract? Some conclusions are easy, others are difficult by reason of the limitations in our knowledge. Accurate investigation of fields of vision, with a view to the study of hemianopia and other defects in the visual fields variously caused, is made by means of the perimeter, for directions concerning the use of which the student is referred to works on ophthalmology. Hermann Sahli suggests an easy, rough method quite sufficient for recognizing marked difference in the field of vision, performed as follows: The physician seats himself opposite the patient, whose right eye—supposing this to be the one to be tested—is opposite the physician's left, the other eye of each being closed. The two open eyes being thus fixedly opposed, the physician passes his finger to and fro across the field of vision exactly midway between the two eyes. In this way he can compare his own field of vision with that of the patient, noting at what moment the finger is seen approaching from the periphery by each. Care must be taken that the finger is kept exactly midway between the physician and patient, and in order to do this the examiner must from time to time open his closed eye. Defective sight in one eye with diminished reflex action of pupil proportionate to the defect, the function of the remaining eye being intact, means disease of one optic nerve. In those rare cases of functional disease in which the sight of one eye only is involved, the perfect responsiveness of the pupil distinguishes it from organic disease of the nerve. Total loss of sight in both eyes may mean chronic atrophy, damage to the chiasm, or disease of both tracts or in both hemispheres. In these cases the symptoms are at first partial, and in this way the diagnosis is aided.

Central scotoma means damage to nerve fibres in the centre of the trunk of the optic nerve, either inflammatory or the result of hemorrhage. Peripheral limitation of vision means damage to fibres running in the periphery of the nerve. Sectorial blindness in one eye means disease of the nerve, decided in degree but limited in extent.

Temporal hemianopia means disease of the chiasm, while the combination of complete blindness of one eye with temporal hemianopia in the other means disease of the chiasm which has extended to the outer fibres and even to the optic tract on the side on which blindness is complete.

Lateral hemianopia is due to disease back of the chiasm, and the determination of which one of the numerous points in the tract between the chiasm and the extensive occipital area which is the cortical centre of vision stimulates diagnostic acumen. Most that can be attempted is the settling of the question as to whether the disease is in the tract between the chiasm and the geniculate bodies or in the fibres beyond bounded by the visual centres. To this end the *hemianopia pupil reaction* of Wernicke is sought. A perfect pupil reflex requires the integrity of the retina, the fibres of the optic nerve and tract, and the centre in the geniculate body which receives the impression and sends it along the 3d nerve to the iris. When the light is thrown on the blind half of the retina the pupil contracts as much as if it is thrown on the seeing half if the disease is

in the hemispheres, but if the disease is in the tract it does not contract because the path to the corpora quadrigemina is interrupted. The test requires much care and experience in its employment. Seguin directs that the patient, being in a darkened room with a light behind his head in the usual position, be directed to look to the other side of the room so as to eliminate accommodation movements. Then a faint light is thrown from a plane or large concave mirror, held well out of focus, upon the eye, and the size of the pupil noted. With the other hand a beam of light, focussed by an ophthalmic mirror, is then thrown directly into the optical centre of the eye, then laterally in various positions and from above and below the equator of the eye, noting the reaction of all angles of incidence. According or not as a response is obtained in the pupil the inference is drawn.

Amblyopia with concentric reduction of the field, decided in one eye and slight in the other, may be due to atrophy of the nerve or disease of the higher visual centre in one hemisphere or to hysteria. If atrophy, the ophthalmoscope recognizes the lesion and the responsiveness of the pupil is diminished. If disease of one hemisphere, the nerve is normal to the ophthalmoscope, the pupil contracts perfectly under the action of light, and the onset is sudden or accompanied with other signs of organic brain disease. Crossed amblyopia, or dimness of vision in the eye of the opposite side with contraction of the field in the better eye, has been already referred to as a more or less characteristic result of lesion of the angular gyrus. Mind-blindness, described on p. 912, may also be a result of lesion in this locality. In hysteria the symptoms are the same as in disease of the higher visual centre, and the diagnosis depends on the presence or absence of signs of organic or functional disease. In hysterical amblyopia the loss of sight is rarely complete. In hysterical and neurotic defects of vision there may be a derangement of the natural relation in color fields. Thus while normally the blue field is most conspicuous in the last-named conditions, it is often overshadowed by other colors.

LESIONS OF THE MOTOR NERVES OF THE EYEBALL.

Anatomical.—The 3d cranial nerve (oculo-motor) supplies the levator palpebræ superioris, the rectus superior and inferior, the rectus internus, obliquus inferior, the sphincter of the iris, and the ciliary muscles. The 4th cranial nerve (the trochlear) supplies the superior oblique; the 6th cranial nerve (the abducens) the rectus externus. The functions of the muscles to which these nerves are distributed are sufficiently indicated by their names.

Third Nerve.—Lesions may involve the nerve at its nuclear origin or in its course. Lesion of the 3d nerve at its origin involves also usually the origin of the other motor nerves of the eye, producing general ophthalmoplegia, as the result of which the eyeball is motionless, and an object moved about in front of it can be followed only by moving the entire head. The nerve may be affected in its course by traumatic causes, meningitis, gummy tumors, aneurism, or by neuritis, frequently

rheumatic, seen in diphtheria, locomotor ataxia, and diabetes mellitus. The effect may be spasm or paralysis.

The results of *spasm* of the muscles supplied by the 3d nerve are manifested in *nystagmus*. This consists in an involuntary, rhythmical, oscillatory movement of the eyeball usually horizontal, but sometimes rotary, more rarely vertical. It is a result of paresis of the muscles of the eye, but occurs also in the absence of demonstrable paresis. It is met in congenital or acquired brain lesions, and is often a striking feature in albinism. In meningitis and hysteria there is also sometimes spasm of the muscles supplied by the 3d nerve, especially the external rectus, and levator palpebræ which is the antagonist of the orbicularis.*

Paralysis of the muscles supplied by the 3d nerve, which includes all the eye muscles except the external rectus and superior oblique, results in outward squint, ptosis, or drooping of the upper eyelid ; the absence of contracting power in the pupil, which remains of medium size ; loss of accommodation, and double vision, or diplopia. Such paralysis, involving all the branches of the nerve, may be recurrent, especially in women, often at the menstrual period, or at longer intervals.

It is sometimes associated with pain in the head and sometimes with migraine. The individual attack lasts sometimes a few days, sometimes as many weeks. Partial involvement of the 3d nerve may include the levator palpebræ, superior rectus, the ciliary muscle and iris, while the external muscles, that is, the internal and inferior recti and inferior oblique, may escape.

Ptosis only, due to paralysis of the levator palpebræ, complete or partial, may occur under various conditions. It may be congenital and incurable, or due to cerebral lesion ; or it may be hysterical, when it is apt to affect both eyes and is associated with other symptoms of hysteria. It may be caused by disease of the sympathetic nerve (pseudo-ptosis) and associated with symptoms of vaso-motor palsy, viz., elevation of temperature on the affected side, redness or œdema of the skin, and contraction of the pupil on the same side. It occurs sometimes in the bilateral form in idiopathic muscular atrophy with involvement of the face muscles. Finally, it is seen in weak, delicate women as a transient event, especially in the morning. When the result of a definite lesion of the 3d nerve, at its nucleus or in its course, it may also be associated as with a paralysis of the superior rectus alone, or of the internal and inferior in addition.

Conditions of the Pupil.—The conditions of the pupil should be studied with light of moderate intensity, and in doubtful states the pupil under examination should be compared with that of the eye of a healthy individual about the same age.

Myosis, or contraction of the pupil, is found physiologically during sleep, especially in elderly persons ; pathologically as an early symptom in tabes dorsalis, in progressive paralysis, and as an effect of eserin, pilocarpin, morphine, and in complete chloroform narcosis.

* Blepharospasm is a spasm of the orbicularis muscle which is supplied by the facial nerve. It amounts usually only to twitching of the eyelids, but may be so severe as to close them completely, so that it is not in the power of the patient to open them.

Mydriasis, or dilatation of the pupil, occurs in deep unconsciousness, during extreme pain, in dyspnœa, peripheral blindness, especially from optic atrophy, in oculo-motor paralysis, rarely in tabes dorsalis and progressive paralysis. It is also an effect of atropin, duboisin, cocain, and of the early stage of chloroform narcosis.

The pupil may be unduly large from palsy of the sphincter (3d pair) fibres or spasm of the radiating (sympathetic) fibres; or the pupil may be over small from the opposite conditions.

Other limited paralyses due to 3d nerve disease are cycloplegia and iridoplegia. *Cycloplegia* is paralysis of the ciliary muscle, producing loss of power of accommodation. In this state of affairs distant vision is good, but near objects cannot be seen distinctly. It may occur in one or both eyes, being in the latter event more usually due to disease of the nuclear origin of the 3d nerve. It is one of the earliest manifestations of diphtheritic paralysis and is a symptom also of tabes. It may be corrected by the use of glasses.

Iridoplegia is paralysis of the iris, and its three forms are thus classified by Gowers, one associated and two reflex :—

(1) *Accommodative iridoplegia* is the form in which the pupil does not diminish in size during accommodation. It is tested by having the patient look at a distant object and then at a near one in the same line of vision, so as to avoid any change in the amount of light entering the eye. It is usually associated with paralysis of accommodation, but the ciliary muscle may be efficient and yet the associated action of the iris be lost, and the reverse. This loss is less common than that of reflex action. It is the result of the same cause as cycloplegia.

(2) *Reflex Iridoplegia, or Argyll-Robertson Pupil.*—The path for the optic reflex is along the optic nerve and tract to the geniculate bodies, thence to the ciliary ganglion, and through the ciliary nerves to the eye. In testing for this condition each eye should be tried separately, the other eye being covered, but not closed. The patient is asked to look toward a dark part of the room, when a bright light is thrown suddenly in front of the eye at the distance of three or four feet, so as to avoid the effect of accommodation. If the patient looks at a nearer light, he will accommodate for it, and the pupil may contract when there is no action to light. Such absence of light reflex without loss of the accommodation contraction was first pointed out by Argyll-Robertson.

(3) *Skin Iridoplegia.*—Loss of skin reflex. If the skin of the neck is pinched or pricked, or stimulated by an electric shock, the pupil dilates reflexly. Since active dilatation of the pupil is through the sympathetic nerve, the motor path for this action must be along the cervical sympathetic, and the fibres connecting this with the cord, at the lowest part of the cervical region. The centre on which it depends is said to be beneath the corpora quadrigemina, to the outer side of that for reflex light, in which event it is likely that the sensory path must pass in the cervical region of the spinal cord and thence through the sympathetic also to the centre.

These reactions are lost when the path is interrupted or the centre is damaged. Thus, the light reflex is lost or impaired in disease of the

optic nerve including the retina, or disease of the 3d nerve. Disease of one optic tract does not lessen the action unless the light falls on the blind half of the retina, because, as already stated, the fibres from the central and most sensitive region of each retina pass through both optic tracts, whence disease of one does not abolish the reflex. The skin reflex is lost in disease of the cervical sympathetic and sometimes of the cervical spinal cord, especially when there is associated loss of sensibility. Thus tumor of the cord sometimes produces this symptom.

When the eye reflexes are lost without disease of the sympathetic or cervical cord, it is generally due to degenerate disease of the centres. Locomotor ataxia, in which it is a common and early symptom, is a conspicuous instance. Less frequent is general paralysis of the insane, and other degenerative processes less definite. It may occur also without other nerve symptoms. In most of the cases in which it has come under Gowers' observation, thus isolated, the patient had constitutional syphilis for years. The two palsies of skin and light reflex are commonly associated, but not always. The pupils are often small, reduced to two millimeters or even one millimeter in diameter.

Inequality of pupils, or *anisocoria*, is also a symptom of progressive paresis and tabes, but occurs also in healthy persons.

Fourth Nerve.—The 4th cranial nerve (trochlear) is liable to be compressed by tumors as it passes around the outer surface of the crus into the orbit by aneurism or the exudation of basilar meningitis. Its nucleus in the upper part of the 4th ventricle may be involved in tumors, or undergo degeneration with other ocular nuclei. As the superior oblique muscle supplied by it acts in such a way as to direct the eyeball downward and rotate it slightly, paralysis causes retardation of downward and inward movement, often so slight as not to be noticeable. The head is inclined somewhat forward and toward the sound side, and there is double vision when the patient looks down, as in descending stairs. Paralysis of this nerve is seldom met with alone, except in nuclear disease.

Sixth Nerve.—The 6th nerve (abducens), emerging at the junction of the pons and medulla, passing forward and entering the orbit, is liable to be affected by meningitis at the base, or by tumors, especially gummata, or by cold. The external rectus being alone supplied by it, the effect of its paralysis is to produce internal squint, and the eye cannot be turned outward. It is a frequent ocular palsy, because the nerve has so long and exposed a course.

If the nucleus of the 6th nerve is affected, a very interesting condition results, which was first studied by Beevor. In consequence of the paralysis of the external rectus, the eye of that side is turned inward, while at the same time the internal rectus of the eye of the opposite side has lost the power to turn its eye inward. Consequently, both eyes are turned to the side opposite and away from that of injury. Thus if the nucleus of the right 6th nerve is involved, both the right and left eyes are turned toward the left. Such opposite deviation

away from the side of lesion is known as "conjugate deviation." It is due to the fact that the nucleus of the 3d nerve, which supplies the internal rectus, receives from the nucleus of the opposite 6th nerve, through the pons, fibres of its own. Whence in lesion of the nucleus of the 6th nerve there is paralysis of the internal rectus supplied by the 3d nerve, though the nucleus of the 3d nerve is not involved.

In consequence of the proximity of the nucleus of the 6th nerve to that of the 7th, or facial, disease of the former is apt to involve the latter. Whence, say if there is lesion of the left nerve, there follows conjugate deviation of both eyes to the right, with a complete paralysis of the left half of the face.

Phenomena in General of Paralysis of Motor Nerves of the Eye.—These include, first, limitation of movement and strabismus referred to. In addition to these occur certain derangements of vision known as *secondary deviation, erroneous projection, double vision.*

Secondary Deviations are thus demonstrated: After covering the sound eye, let the paretic eye fix itself upon a point which it cannot reach at all, or can reach only after extreme exertion. Then remove the covering hand from the sound eye, and it will be found that the latter has been moved much too far in the same direction, the abnormal attempt at innervation of the affected eye passing over to the associated muscle of the healthy eye and causing in it too great a contraction.

Erroneous Projection furnishes the idea that an object at which we are looking is further on one side than it really is, or that the movement of the eye in following it, when moving, is greater than it is. Under these circumstances, in an attempt to touch the object with the fingers the latter may go beyond it. This grows out of the fact that when the eyes are at rest in the mid position, an object at which we are looking appears directly opposite the face. Turning the eye to one side, the object appears to the side of its former position, and if the object moves we estimate the extent of its motion by the amount of movement of the eyeball following it. Now, when one muscle is weak, the increased innervation required to contract it gives the impression of a greater degree of movement than actually takes place. This is erroneous projection. Now, as the equilibrium of the body is largely maintained by a knowledge of the relation of external objects to it, obtained by the action of the eye muscles, the erroneous projection due to paralysis disturbs the harmony of visual impressions and may produce dizziness, known as ocular vertigo.

Double Vision results from the fact that if one eye is paralyzed the axes of the two eyes do not coincide, nor do the images in the two retinæ. The image produced in the sound eye is called the true image, that in the affected eye the false image. In simple or homonymous diplopia the false image is on the same side as the paralyzed eye, in crossed diplopia it is on the other side. It is one of the most annoying symptoms of paralysis of the eye muscles.

Ophthalmoplegia.—Ophthalmoplegia, or nuclear palsy, is a term applied to a chronic progressive paralysis of the ocular muscles, due to disease of the ocular nuclei. It is called *internal* when the internal muscles

only are involved, *i. e.*, the iris and ciliary muscles ; *external*, when the external muscles are affected more or less completely. When both internal and external muscles are involved it is known as *total* ophthalmoplegia.

Historical.—The term was first used by Brunner in 1850. The nature of the cases was pointed out by v. Graefe in 1856, and in 1868 compared by him with bulbar palsy. Forster localized the lesion in 1878 for external palsy, including all the muscles except the iris and ciliary muscle. Internal ophthalmoplegia was described by Hutchinson in the same year, and the external form, with post-mortem proof of its nature, in 1879.

Symptoms.—These vary according to the position and character of the lesion, which may be degenerative, hemorrhagic, or the result of pressure by tumors or the product of basilar meningitis. They are bilateral, except in the instance of the 6th nerve, with resulting conjugate paralysis. Gowers describes three modes of onset, chronic, acute, and sudden.

The *chronic form* is the most common, due to nuclear degeneration, or more rarely to tumor and embolic obstruction, and still more rarely to hemorrhage. In this form there is a great variety of combination and degree. Thus there may be internal ophthalmoplegia only or external or both. In the internal form there may be loss of the iris-reflex only or of the ciliary muscle-action only. In the external variety the levator and superior recti are commonly first involved, the other muscles gradually. There may be loss of the upward and downward movement of the eye, ptosis, and conjugate lateral palsy. There may be double vision, generally of short duration. In the total form the eye is fixed and immovable. Each variety may be associated with locomotor ataxia, general paralysis, progressive muscular atrophy, and bulbar palsy, often with syphilis. It is more common in males and occurs occasionally in the young ; and there is a form in children known as infantile oculo-facial palsy which may be congenital or begin later ; rarely, as a sequel of diphtheria, late and permanent.

The disease may be very slow in developing and may require years. Sometimes one eye is more affected than the other. If the internal muscles are unaffected the disease is pretty sure to be nuclear disease, because these muscles can scarcely escape bilateral disease of the nerve trunks. Indeed, Graefe thought this absence of involvement of the internal muscles characteristic. Palsy of the external ocular muscles is apt to be accompanied by facial palsy.

In *sudden nuclear palsy*, the second in frequency, the onset may take but a few minutes or an hour or two. The causes in such cases are commonly obstruction to the basilar arterial branches, rarely embolic obstruction, and still more rarely hemorrhage. The obstruction is usually bilateral. The lesions are irregular and the symptoms are correspondingly irregular and unsymmetrical, and the tendency is to recover. In these respects it differs from the chronic form also in that hemiplegia is a frequent accompaniment, generally on the side opposite the greater eye palsy. When hemorrhage is a cause the resulting ocular palsy lasts usually but a few hours, while the other phenomena of pressure by effused blood, which is apt to spread, make their appearance.

The *acute nuclear palsy* is rare. It develops in a few days or weeks and is possibly of inflammatory or toxic origin, whence called by Wernicke *poliomyelitis superior*, but toxic cases may occur without inflammation. This form, according to Gowers, may be due to peripheral neuritis and not to nuclear disease. Alcohol may be a toxic cause. The eye muscles are invaded irregularly and it is common for the internal muscles to escape. In fatal cases the causal influence extends to the centres of other nerves and to the cortex. In cases that survive there is improvement, various in degree.

Treatment of Ocular Palsies.—The cause should be sought and, if found, treated. Although syphilis is thought to be one cause of this disease and of locomotor ataxia, with which it is so frequently associated, disappointment follows the syphilitic treatment in the majority of cases. Yet mercury perhaps accomplishes more than any single remedy. Arsenic, strychnine, and iron are sometimes used, strychnine hypodermically.

In acute cases where there is pain, hot fomentations, leeches, and counter-irritation may be used. In chronic forms, electricity has been extensively used, galvanism being preferred. Benedekt recommended placing the anode, or positive pole, on the forehead, and the cathode, or negative pole, on the margin of the orbit near the affected muscle. If the faradic current is used, the orbital pole is held still; if the voltaic, it is kept moving over the skin, or the current is broken by the commutator. To overcome the ptosis, electrical stimulus is applied to the 3d nerve, as the muscle is not accessible.

The diplopia is removed by a prism not strong enough to completely fuse the two images, but of sufficient force to approximate them, so that the fusion may be completed by muscular action. Such action may be practised for an hour each day. The dizziness due to erroneous projection can only be removed by throwing the eye out of use by an opaque glass. Operative treatment is not recommended.

TRIFACIAL OR FIFTH NERVE (TRIGEMINUS).

Anatomical.—This important mixed nerve of the face supplies by its motor trunk the muscles of mastication, by its sensory portion the skin of the face, the mucous membrane of the mouth and nasal cavity, the conjunctiva and cornea, also the anterior part of the tongue with gustatory fibres, and the nose with olfactory fibres. The gustatory fibres reach the lingual fibres of the 5th by the chorda tympani nerve.

Lesions.—(1) There may be lesions of the pons, especially hemorrhage, or spots of sclerosis invading the trigeminus nucleus.

(2) Injury or disease at the base of the skull, especially acute and chronic meningitis and caries of the bone, tumors, syphilis, new formations compressing the trunk or Gasserian ganglion. Fracture of the base rarely affects this nerve.

(3) Tumors or aneurisms pressing on the 1st division (ophthalmic) of the nerve through the cavernous sinus, on the 2d division (superior

maxillary) and on the 3d division (inferior maxillary) by invasion of the spheno-maxillary fossa.

(4) There may be inflammation of the nerve, which is rare.

The sensory division may also be affected in hysteria and lesions of the posterior part of the internal capsule. The gustatory fibres of the trigeminus may be influenced by peripheral lesions of the facial, whence the chorda tympani is derived.

Symptoms.—*Paralysis of the sensory portion:* The distribution of the anæsthesia varies according as the whole trigeminus or only a half is involved. In total anæsthesia there is loss of sensation in half the corresponding side of the head, including the conjunctiva and cornea, mucosa of the lips, tongue, hard and soft palate, and nose of the same side. Hence on the tongue or mucous membranes there are often ulcers which come from unconscious laceration of the teeth. There is loss of the sense of taste and smell. The muscles of the face are also insensible, whence their movements are slower. The so-called trophic phenomena are also observed, and among them the much-discussed *neuro-paralytic ophthalmia*, an ulcerative keratitis, beginning, also, always in the lower segment of the cornea, and passing over into purulent inflammation of the whole eyeball. It seems, on the whole, more likely that the inflammation is primarily due to the action of irritants which in health are excluded by the proper closure of the eyelids, though the inflammatory process itself may be trophically influenced. The salivary, lachrymal, and buccal secretions may be diminished and the teeth become loose. Herpes is a trophic result which may develop in the course of the nerve, is painful, and may last a long time. So, too, the anæsthesia may be preceded by tingling. The skin of the face is sometimes swollen.

Paralysis of the motor portion, which supplies especially the muscles of mastication, the masseters, temporals, and pterygoids, is not common. It is most frequent in diseases of the base of the skull, compressing this branch. Difficulty in chewing is the result. If on one side, the patient can only chew on the other; if on both sides, he cannot chew at all. The lower jaw hangs down and cannot be moved from side to side because of the paralysis of the pterygoids. If on one side, the external pterygoid cannot push the jaw toward the sound side, and when depressed the jaw is pushed by the muscle of the sound side toward the paralyzed side. Cases have occurred associated with cortical lesion; from one such Hirt inferred that the motor centre for the trigeminus is in the neighborhood of the lower 3d of the ascending frontal convolution.

Spasm of muscles of mastication is met with in connection with muscular cramp, the muscular contraction of tetanus (trismus), sometimes in tetany and meningitis, and reflexly through painful affections of the jaw or teeth, or from irritation near the motor nucleus. It is also sometimes hysterical. Clonic spasm occurs in muscles supplied by the 5th nerve, constituting "chattering teeth." It occurs generally in connection with general conditions, such as chorea, but it may happen as a local symptom in women late in life.

Diagnosis.—This is not difficult. Sensibility is tested in the ordinary way. The preliminary pain must not be mistaken for neuralgia.

Gustatory sense is tested in the anterior end of the tongue by applying weak acid or salt solutions and comparing the effect on the two halves. The motor power is tested by biting on a piece of wood or cork or by moving the jaws against resistance.

Treatment.—This must depend upon the cause, which should be carefully sought. Syphilitic new formations are the lesions most commonly amenable to treatment. In the absence of such causes the treatment must be symptomatic. Stimulating liniments and faradization through the electric brush are often useful. Galvanism may also be used, brushing the part with the cathode. The anæsthetic part should be carefully protected against irritants.

In the absence of tangible cause, systemic treatment is not indicated, except to build up the general health of the patient.

LESIONS OF THE FACIAL NERVE, OR SEVENTH PAIR.

The seventh pair (portio dura of the 7th, old classification) is the motor nerve of the face, and is subject to paralysis of motion and to spasm.

Paralysis of the Facial.

Synonyms.—*Mimetic Facial Paralysis; Bell's Palsy.*

Paralysis may be caused by lesions in the cortical centre of the nerve, in the nucleus itself, and in the nerve trunk.

Supra-nuclear Paralysis.—The cortical centre resides in the foot of the central convolution, from which pass out fibres along with the pyramidal fibres through the internal capsule to the facial nucleus in the upper medulla on opposite side. Accordingly, it is very constantly involved in hemiplegias—in fact, facial paralysis forms a part of most hemiplegias. Such a paralysis, due to lesion above the facial nucleus, is known as supra-nuclear. In such a palsy the voluntary muscles of the lower half of the face and the corresponding half of the palate are paralyzed, while the secretory and gustatory functions of the facial are not affected. The orbicularis and forehead muscles are intact, being innervated by the upper branch of the facial. This feature, together with the normal electrical excitability of both nerve and muscle, the involvement of voluntary movements rather than the emotional, the intact reflexes and taste-sense, all point to a *central* facial paralysis as distinguished from a peripheral. This involvement of the lower half of the face is due to the fact that only the inferior or face branches of the facial receive exclusively crossed innervation, while the upper branch, like the ocular muscles and the motor trigeminus, is innervated from both hemispheres, so that a defect in one may be equalized by the other. This will be understood by an examination of the schematic drawing (Fig. 87), from which it is plain that a one-sided brain lesion at *a* paralyzes only the inferior and not the upper facial. That the upper and lower branches of the facial nerve have separate nuclei has not been as yet anatomically proven. That they are, however, *functionally* distinct is further shown by the fact that in bulbar paralysis, a

disease of the nerve nuclei of the medulla, only the inferior facial is involved. That the upper face muscles are totally uninvolved in central facial paralysis is not quite true, for careful examination will show that the function is not quite as perfect as in health; the patient cannot close the eye of the paralyzed side by itself, as in the normal state; whence it follows that the upper half of the face is innervated from both hemispheres, as is also shown in Fig. 87. The crossed influence is, however, the larger.

Cortical facial paralysis, *monoplegia facialis*, has been found associated with lesions in the centre for face muscles in the lower Rolandic

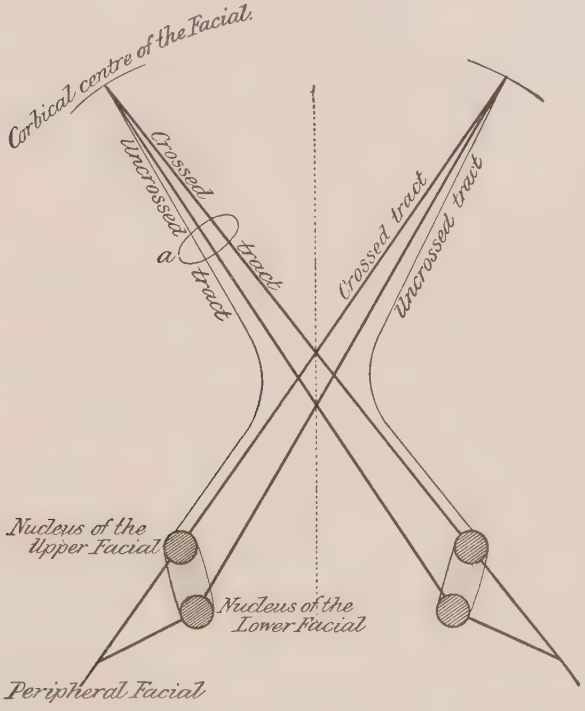


FIG. 87.—SCHEMA FOR CENTRAL INNERVATION OF THE FACIAL NERVE.
The upper nucleus is innervated from both hemispheres, though mostly from the opposite side, while the lower nucleus is innervated almost totally from the other side.—(*After Sahli.*)

region, but isolated facial paralysis due to involvement of the nerve fibres in their path to the nucleus, is rare. Cortical facial paralysis, as already explained, is on the same side as that of the arm and leg.

Nuclear Paralysis.—Paralysis may also be caused by lesions of the nucleus, but is not common. There may be tumors, chronic softening, hemorrhage, the diphtheritic poison, while rarely anterior poliomyelitis may involve the facial nucleus. The symptoms are essentially the same as those of paralysis of the trunk of the nerve, or peripheral facial palsy.

Infranuclear Paralysis.—Peripheral facial paralysis includes all forms due to involvement of the nerve trunk. The distinctive features of this as compared with cortical paralysis, have already been stated. It still remains, however, to determine the precise segment of the nerve involved, to be again referred to when treating of diagnosis.

Etiology.—Cortical paralyses are usually due to compression or destruction of the cortical centre, as by traumatism, hemorrhage, tumor, meningitis, or embolism

The most frequent cause of peripheral paralysis is exposure to cold, as to a cold wind or a draught at an open window. Such cases include the so-called rheumatic paralysis, and may be due to neuritis. Disease of the middle ear and caries of the petrous portion of the temporal bone are relatively frequent causes, which have evident explanation in the course of the facial through the fallopian canal adjacent to the tympanic cavity, whence it may be invaded. At the base of the brain, tumors, syphilitic new formations, and inflammatory processes also involve the facial. Rarely swelling of the parotid gland is a cause of pressure. Finally, the facial is frequently implicated in disease of the brain and medulla. The causes of nuclear paralysis are named above.

Symptoms.—The total complex of symptoms varies with the exact seat at which the nerve is invaded. Paralysis of the facial muscles of expression produces the most striking physiognomy. The homely description, understood by everyone, is that the face is drawn to one side. And so it is—to the sound side. Examination discovers that on the opposite and paralyzed side there is a remarkable smoothness of face, the wrinkles have disappeared from the forehead, the labio-nasal fold is gone, and the half of the face is quite expressionless. The corner of the mouth is lowered, while saliva frequently flows from it, the eye is wider open than natural and can be only partially closed even during sleep—*lagophthalmus*—and the eye waters. These symptoms are rendered still more striking on effort at smiling, talking, and whistling, at turning up the nose, wrinkling the forehead, inflating the cheeks, or closing the eyes. On attempting the latter, the upper lid drops as though heavy, the eye is turned upward, the pupil covered, but quite a space remains "uncovered." The so-called "corneal" and "optical" reflexes, by which through closure of the lid the eye protects itself from the entrance of foreign bodies seen to approach, is lost, and a tendency to conjunctivitis results.

Winking, in complete facial paralysis, is impossible. Whistling is impossible, and speech may be interfered with, owing to the difficulty in forming labial sounds. The proper muscles of mastication are not paralyzed, but owing to paralysis of the buccinator muscle food collects between the teeth and cheeks on the paralyzed side, and an attempt to sniff reveals paralysis of the nasal muscles. The upper teeth cannot be uncovered and an attempt to drink is only partially successful because the lips cannot be kept close to the glass. The tongue is usually described as protruded toward the paralyzed side, but this appears to have been an error. The organ is really central when examined in its relation to the incisors, and the erroneous impressions arise from the fact that the lips are drawn to the sound side. German authorities all speak of a paralysis

of the soft palate on the affected side, since facial fibres pass through the superficial petrosal nerve to the spheno-palatine ganglion. It is described as drooping, while effort at phonation raises the soft palate obliquely to the affected side. Both Gowers and Hughlings Jackson, however, deny this symptom in most cases, and are sustained by the discovery of Horsley and Beevor that the soft palate is innervated by the spinal accessory.

Derangement of taste also occurs in the anterior two-thirds of the tongue on the paralyzed side in cases where the facial is involved in that part of its course in which it contains the chorda tympani nerve, that is, in the Fallopian canal between the genu and the origin of the chorda tympani. When the nerve is affected outside of the skull the sense of taste is intact. Tactile sense in the tongue is also sometimes lessened; salivary secretion diminished, producing dryness of the mouth; hearing more acute, especially for low notes. This is due to paralysis of the stapedius muscle, which is antagonized by the tensor tympani innervated by the trigeminus. Hence results a greater sensitiveness of the membrana tympani. Other disturbances of hearing are also present, but they are generally due to associated aural trouble. Herpes is also an occasional symptom ascribed to the presence of trigeminal filaments among those of the facial.

Facial paralysis usually begins suddenly, rarely gradually. Sometimes there are prodromata, consisting in abnormal sensations of taste, pain in the ear and face, and ringing in the former, all from inflammation of the nerve. With this exception, pain is not common.

Diagnosis.—The recognition of the presence of paralysis of the trifacial is for the most part easy. More difficult and proportionally important is it to ascertain in what part of its course the function of the nerve is cut off. This is rendered easy by the appended schematic drawing of the distribution of the facial nerve after Erb.

The phenomena vary in accordance with the following:—

(*a*) Lesion between 1 and 2, trunk of the facial below the Fallopian canal. Paralysis of the facial muscles; taste, secretion of saliva, hearing, and palate normal.

(*b*) Lesion between 2 and 3 within the Fallopian canal. Paralysis of facial muscles, derangement of taste, eventually diminished secretion of saliva, but hearing and soft palate normal.

(*c*) Lesion between 3 and 4. Paralysis of the facial muscles, derangement of taste, diminished secretion of saliva, abnormal acuteness of hearing, soft palate normal.

(*d*) Lesion between the 4 and 5 in geniculate ganglion. Paralysis of the facial muscles, derangement of taste, diminished secretion of saliva, abnormal acuteness of hearing, and paresis of soft palate.

(5) Lesion between 5 and 6 above geniculate ganglion. Paralysis of the facial muscles, diminished secretion of saliva, abnormal acuteness of hearing, paresis of soft palate, but no disturbance of taste.

The student is referred to what has been said above as to the modification rendered necessary by the observations of Gowers and others on the non-involvement of the soft palate in facial palsy.

We are aided also in recognizing precise forms by the causes, if

known, as the presence of ear disease, exposure to cold or traumatism. Co-existing symptoms of brain or bulbar disease must also be considered. Reaction of degeneration cannot occur in true cerebral facial palsy, only in peripheral palsy or such bulbar paralysis as affects the facial below the nucleus itself. In cortical facial paralysis the frontal distribution of the facial nerve is not paralyzed; in the peripheral it is; nor are the eye-muscles affected in the former.

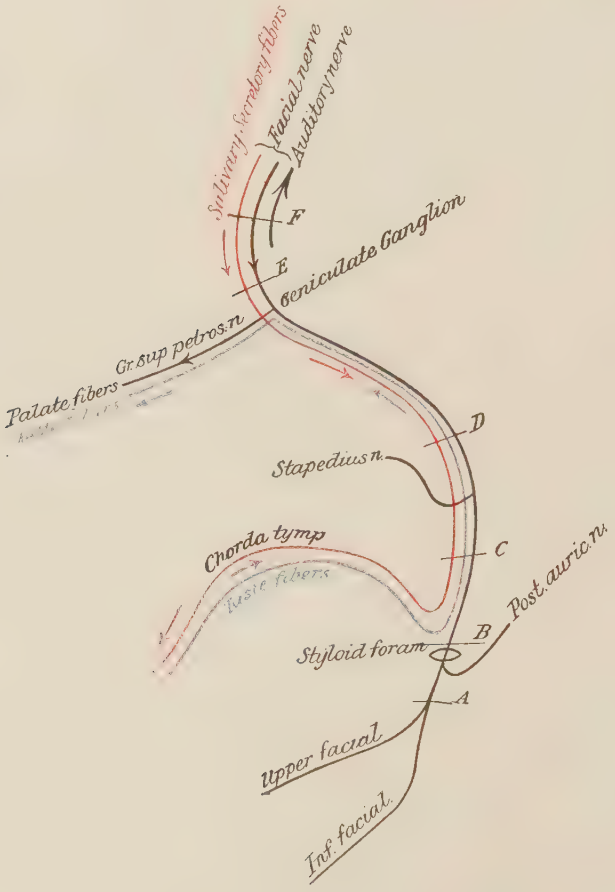


FIG. 88.—SIMPLIFIED DRAWING OF THE PERIPHERAL DISTRIBUTION OF THE FACIAL NERVE.—(*After Sahli.*)

Prognosis and Course.—The prognosis varies with the etiology and degree of severity. Some cases get well rapidly; others partially recover, many are permanent. The following division of forms with their probabilities according to Erb will be helpful.

(1) *The Mild Form of Facial Paralysis.*—To this many rheumatic cases belong. The affection is usually one of facial muscles only. Electrical excitability in the paralyzed muscle remains normal and there are no severe and deep-seated changes in nerves or muscles. Recovery is rapid, usually in two or three weeks.

(2) *Middle Form.*—There is partial reaction of degeneration, the excitability of nerve being diminished but not lost; in the muscles, however, in two or three weeks, there is decided increase of galvanic excitability to direct excitement; the anodal closure contraction being greater than while contractions are slow. Recovery may still be quite rapid, usually in from four to six weeks.

(3) *Severe Form.*—Complete reaction of degeneration in nerve and muscles, *i. e.*, loss of faradic and galvanic excitability of nerve, loss of faradic excitability of muscle, and quantitative and qualitative changes in galvanic excitability of muscle. In this form there is always degeneration of nerve and muscle, so that, if recovery takes place at all, it is only after two or six months, or longer.

In these cases there often intervene symptoms of motor irritation, consisting :—

(1) In a marked tonic contraction of the paralyzed muscles, sometimes very striking.

(2) Single spasmodic contractions of muscles.

(3) Of special associated movements. Thus, if the patient closes his eyes or winks, there always follows a marked distortion of the corner of the mouth, which cannot be restrained.

(4) An increased reflex irritability, as the result of which on pricking or blowing on the skin vigorous muscular contractions follow.

These symptoms last for a very long time, for years in incurable or imperfectly cured cases.

Further points bearing on prognosis have reference to the nature of the primary disease. Paralysis caused by tumors of the base of the brain and caries of the petrous bone is almost always incurable. If the paralysis is due to middle-ear disease the prognosis depends on the curability of it. The electrical examination affords helpful data. If at the end of one or two weeks electrical excitability still remains normal, a rapidly favorable termination may be predicted. If, on the other hand, the reaction of degeneration is present, a much longer course and delayed recovery, if any, may be predicted. Relapses may occur.

Treatment.—The treatment is, of course, that of the lesion which lies at the bottom of the paralysis. If it is a syphilitic, inflammatory product, the iodides should be administered in the usual ascending doses. Middle-ear disease should receive the promptest and closest attention, as some of the most unfortunate cases are thus caused. Any possible cause of pressure should be sought and removed.

Where cold is the cause, and the case comes early under observation, warmth, either dry or moist, should be applied to the distribution of the nerve in the face, while mild counter-irritation at the pes anserinus is useful. Decided blistering is of questionable utility, but it is harmless and may do good.

For the paralysis remaining after the removal of the cause electricity is indicated, and more especially the constant current. A weak current should be used for three to five minutes at a time, interrupting four to six times a minute, placing first the anode and then the cathode in the auriculo-mastoid fossa, the other pole in front of the ear. Galvanism and

faradization may be applied to the muscles themselves, including the orbicularis, the direct effect of the electricity on which is shown by an increased power to close the eye immediately after the application of the current. Massage of the muscles may be used.

Sulphate of strychnine is a drug which has some reputation in facial paralysis, although it is difficult to trace the results of its use. It is recommended to be used by subcutaneous injection, daily or on alternate days.

Facial Spasm.

Synonyms.—*Mimetic Facial Spasm; Convulsive Tic.*

Facial spasm is manifested in a variety of ways. Mimetic facial spasm, or convulsive tic, consists in a clonic contraction of the muscles supplied by the facial nerve, a few or all, usually unilateral, sometimes bilateral. A similar condition, especially frequent in children, as the result of imitation, or a habit of grimacing, is known as *habit-spasm.*

Etiology.—No cause can be found for most cases. Possible causes are exposure to cold, lesions at the base of the skull, or irritation of the facial centre in the cerebral cortex. Other cases may be explained by reflex causes, such as irritation by carious teeth, intestinal worms, or disease of the sexual organs. Others have been ascribed to violent mental excitement. Disposition to the disease is heightened by a hereditary neuropathic habit.

Symptoms.—These consist in short contractions in the muscles affected. Sometimes the contractions are of longer duration. The face is the subject of constantly changing grimaces during waking hours. Sometimes, however, there are intervals of complete rest. The contractions are commonly without exciting cause. Sometimes they invade adjacent muscles, as those of mastication, or the tongue, or the muscles of the neck. Voluntary motion is unimpaired, and there is no pain or anæsthesia.

Blepharospasm.—A variety of the partial form is *blepharospasm*, a tonic or clonic spasm of the orbicularis muscle. In the clonic form, it is apt to be associated with spasm of the lateral facial muscles, and there is constant twitching of the side of the face with partial closure of the eye. In another clonic variety there is constant contraction of the eyelids and consequent winking.

The tonic form is usually reflex in origin, bilateral, and may last for days or weeks, with occasional interruptions. The reflex cause is commonly some affection of the eye, producing photophobia, or it may reside in some other point in the distribution of the trigeminus. The clonic form may also sometimes be traced to a reflex cause.

Very interesting in connection with blepharospasm is the discovery by v. Graefe of certain so-called "pressure points." These are points at which pressure causes the spasm to cease, so that the eyelids "fly up as if by a spring." These are commonly found at points of exit of the trigeminus, but have also been found on the vertebral column and elsewhere.

Convulsive Tic.—The convulsive tic of the French, or "*Maladies des tics convulsifs*," is an affection allied to hysteria, which commonly begins

in childhood, especially in children with a neurotic hereditary tendency. It is characterized by twitching of the facial and sometimes of the muscles of the body, associated with explosive utterances. These may be utterances of some word which the child has heard—echolalia—or some profane or filthy word—coprolalia. Actions may also be imitated, and mental symptoms especially. Certain forms of imperative conception may be present.

Prognosis.—This in all forms is, as a rule, unfavorable. There are intervals of suspension, sometimes of considerable length, but the spasm recurs, and the disease generally remains incurable.

Treatment.—The treatment is correspondingly unsatisfactory, but a number of things may be done. Causes of reflex irritation should be sought and removed, such as carious teeth and ophthalmias. Paquelin's cautery may be applied to the trunk of the nerve, or to pressure-points if they exist. Nerve section of the supra-orbital nerve has been practised in blepharospasm. Nerve stretching has been followed by relief, at least as long as the paralysis continues, which is commonly a welcome substitute for the twitching. The constant current may be used, seeking also for pressure points, to which the anode is to be applied. If there are none, this pole should be applied to the trunk of the nerve and the different branches of the pes anserinus. In cases of reflex origin, Berger reports satisfactory results obtained by applying the anode to the occiput just under the protuberance, while the cathode is held in the hand—an attempt at galvanization of the medulla. The single sitting should last from five to ten minutes. S. Weir Mitchell recommends the freezing of the cheek every day or every other day with the rhigolene spray. At least transient relief follows.

As to medicines, those usual in nervous affections should be tried—bromide of potassium, strychnine by hypodermic injection, arsenic, iron, oxide of zinc, atropine, curare.

The treatment of convulsive tic is that of hysteria.

LESIONS OF THE AUDITORY OR EIGHTH NERVE.

The eighth pair (portio mollis of the 7th in the older classification of Willis) may be affected anywhere in its course from its cortical centre in the upper part of the first temporo-sphenoidal convolution, thence in the internal capsule across to its nucleus in the pons ; or at the base of the brain after it passes out of the pons between the olivary and restiform bodies ; around the latter forward across the posterior border of the crus cerebelli into the internal auditory meatus to its distribution in the cochlea and vestibule. The proximity of this nerve to the facial at the base of the brain and in the internal auditory meatus is to be remembered. As indicated by its name, it is softer and more vulnerable than the facial, so that equally acting causes may affect it and leave the facial intact.

The symptoms directly due to disease of the auditory nerve are limited to some derangement of hearing, and it is their association with

others which widens their significance in the study of nervous diseases. The derangements of hearing resulting from such lesion are six :—

(1) Loss of hearing, or deafness.

(2) Increased sensitiveness, auditory hyperæsthesia, or hyperacusis.

(3) Symptoms of irritation, causing subjective aural sensations—tinnitus aurium, and allied symptoms.

(4) Disturbances of equilibrium or sensation of such, due to irritation of the fibres in the semicircular canals.

(5) Certain rare instances of involuntary movements, due to disease of the nerve within the ear, as oscillatory motions of the head.

(6) Purely functional derangements of hearing, occurring especially in connection with hysteria, and anæmia following large hemorrhage.

(1) Loss of Function ; Nervous Deafness.

Etiology.—Deafness may be congenital when it is due to labyrinthine defect. According to Gowers, 80 per cent. of deaf mutes are congenitally deaf. The remaining 20 per cent. become so from disease in early life. Of congenital cases it is said that the intermarriage of relations having similar defects is responsible for some, while such intermarriage, even where there is no such defect, is held responsible for a smaller number. Partial as well as total deafness may be congenital.

Of the cases of acquired nervous deafness, disease of the labyrinth, either primary or secondary to that of the middle ear, causes most. The labyrinth is subject to inflammation, acute or chronic, to syphilitic disease, degeneration, and hemorrhage. It may be invaded by meningitis, cerebro-spinal or tubercular. Its membrane may undergo degeneration, due to gout or simple old age. The product of all these may be fibrous or calcareous new formation. The deafness caused by certain drugs, as quinine, has been ascribed to congestion of the internal ear, and that of loud noise, as the explosion of artillery, to hemorrhage.

Lesions of the nerve trunks are less common causes. They may be of the same character as those of the labyrinth, except primary inflammation, although even this is alleged. Primary degeneration may occur in locomotor ataxia. The nerve may be compressed by thickening of the cranial bones, calcareous nodules, tumors, extravasated blood.

The nuclei within the pons may be damaged by hemorrhagic extravasations and tumors. Above the nuclei there may be lesion encroaching on the superficial layer of the tegmentum, a lesion in the internal capsule or in the cortical centre.

Symptoms.—Since, as already stated, derangement of hearing constitutes the only essential symptom of nervous deafness, any enlargement of the subject can only be made by considering the modifications and conditions of this symptom, and such methods as exist of determining the precise seat of the lesion.

Ability to hear through the bone while the air conduction is impaired, implies that the function of the labyrinth is intact, and that deafness is due to obstruction of the meatus or disease of the middle ear and not to nerve deafness. This is further confirmed if the bone conduction is intensi-

fied by closing the meatus, since in this way the vibrations which ordinarily pass out by the meatus are retained. On the other hand, if there is diminished bone conduction it does not necessarily follow that the labyrinth is diseased, because there may be anchylosis of the stapes, which will diminish bone conduction, although no amount of disease of the middle ear will extinguish it if the labyrinth is intact. Further, in health air conduction is heard after bone conduction ceases. This is the basis of Rinne's test, in which the vibrating tuning-fork is first placed upon the mastoid process and allowed to remain until the sound dies away to the patient, when the fork is suddenly transferred to the external auditory meatus of the same ear. If the air-conducting apparatus is normal the fork should again be heard. Again, there may be a moderate impairment of hearing and the relative delicacy of the air conduction be maintained. Absence of bone conduction is, however, the characteristic symptom of nerve deafness. So, to a less degree, is deafness to short and high-pitched sounds, whence the high-pitched, short sound of a watch is a delicate test of the ability to hear through the bone. Simple senile labyrinthine degeneration may be responsible for inability to hear the watch through bone in persons sixty years old or more.

Can we distinguish between labyrinthine disease and disease of the nerve before its terminal distribution? Given the absence of bone conduction, if the facial nerve is paralyzed, and there is no disease of the middle ear or bone, we may conclude that the nerves (facial and auditory) are affected at the base of the brain, or in the internal meatus. If there is disease of the middle ear along with deafness and paralysis of the facial, it is probable that the facial nerve and labyrinth are affected by extension of the disease from the tympanum, but not certain. An involvement of the trunk of the nerve at the base is also probable if some other nerve near it, as the 6th, is involved. It has already been mentioned that the auditory nerve is more sensitive to pressure than the facial, whence an agency, such as an inflammatory product, pressing on both nerves may affect the auditory and leave the facial intact.

No distinctive symptoms have been found associated with lesion of the auditory nuclei in the pons. Such lesion is very rare but has been found associated with deafness on the other side, while it has also been found when the hearing has been unaffected. Sudden deafness, associated with other symptoms of a lesion of the pons or medulla, should excite suspicion of nuclear lesion, especially if paresis of limbs on the opposite side be one of those symptoms.

The auditory fibres between the cortex and the auditory nucleus in the pons in the superficial layer of the tegmentum, may also be affected and produce deafness. Such a lesion has been a tumor of the corpora quadrigemina. Disease of the internal capsule, causing deafness, is associated with hemianæsthesia and symptoms involving the other special senses.

Lesions of the cortical centre are very rare, though they have been sufficiently frequent to confirm the results of experiment on the monkey, which go to show that the 1st temporo-sphenoidal gyrus represents the centre for hearing, since the destruction of this gyrus on the left side in

man has been attended by word deafness. Hemorrhages, softening, pressure by fractures or tumors, may be lesions in this situation.

Treatment.—This is for the most part unsatisfactory, at least from the physician's standpoint. Careful otoscopic examination should be made with a view to discovering disease of the external and middle ear, and the aural surgeon should invariably be consulted in derangements of hearing of more than brief duration, with a view to certainty of diagnosis between nerve deafness and disease of the middle or external ear. Suspected syphilitic tumors should be treated by iodides. Blister in front of or behind the ear may be useful, especially in acute cases; but deep blistering should be avoided in front lest it cause facial neuritis. Electricity has been employed with partially satisfactory results.

(2) Auditory Hyperæsthesia.

True hyperæsthesia, or hyperacusis, is a condition in which ordinary sounds are heard with more than ordinary acuteness, and sounds ordinarily inaudible become audible. In dysæsthesia, or dysacusis, ordinary sounds, although not intensified, produce discomfort. There is generally present some preëxisting symptom, as a headache, during which sounds ordinarily without effect, intensify the headache. Both of these conditions occur in functional as well as organic brain disease. Of the former, hysteria is an instance; of the latter, meningitis.

Treatment.—The treatment, outside the removal of the cause, is by nervous sedatives, as the bromides and preparations of valerian, and asafœtida.

(3) Irritation of the Auditory Nerve—Tinnitus Aurium.

The term tinnitus includes almost every conceivable form of auditory subjective sensation, of which the most common is ringing, roaring, or hissing. It may include humming, or ticking, the sound of rushing steam, roaring of machinery and the like, the sound of a bell, and even articulate speech, music, or voices. It may be persistent or intermittent, with rhythmical intermissions—these commonly correspond with the beating of the pulse. They may be so slight as to be forgotten when the attention is directed to something else, or they may be heard through everything, causing the sorest distress and misery. In fact, their victims have been even impelled to self-destruction. The clicking symptom, sometimes audible to those standing near, is often very annoying, and may be due to clonic spasm of the muscles connected with the Eustachian tube or levator palati. The so-called premonitory "aura" of epileptic seizures is a variety of tinnitus.

Etiology.—Beyond what is conveyed by the word "irritation," it is exceedingly difficult to discover the cause of tinnitus. Changes in the labyrinth appear to be the most common, and Gowers tells us that "Evidence of nervous deafness, mostly due to changes in the internal ear, is distinct in four-fifths of the cases which come under the physician's notice." Disease of the middle and external ear, including inflammation and wax accumulation, is also a fruitful cause, while in a few cases the process

may be wholly in the auditory centres, the nucleus of the nerve or cortical area. Blood movement not usually audible may become so. Internal aneurism is a possible cause. Tinnitus is a very frequent symptom in gouty cases, in my experience, especially when associated with the nervous temperament. So it is in anæmia and neurasthenia. An epileptic aura is often a tinnitus. A systolic brain murmur is sometimes heard over the ear in children, and even in adults.

Treatment.—This is generally most unsatisfactory. The ear should be explored and its surgical diseases treated.

The gouty diathesis must be treated by the salicylates, colchicum, purgatives, and diet; anæmia and neurasthenia by iron, arsenic, nutritious food, and rest. Large doses of salicylic acid, it is known, produce ringing in the ears—a fact to be remembered always.

The bromides are sometimes beneficial, and a few drops of tincture of belladonna are sometimes added. Nitro-glycerine has been highly commended. Beginning with doses of $\frac{1}{1000}$ grain (0.00066 gm.) they should be rapidly increased until the physiological effect is produced. My experience with nitro-glycerine is that the physiological effect is often not attained in adults by doses of $\frac{1}{1000}$ grain (0.00066 gm.).

Counter-irritation is undoubtedly useful at times. It should be applied behind the ear, and actual vesication is the most efficient form. Temporary effect is sometimes often striking, while permanent effect may be produced by repeated blistering.

(4) Disturbance of Equilibrium Associated with Defect of Hearing. Labyrinthine Vertigo. Menière's Disease.

Definition.—The term Menière's disease is applied to a vertigo usually sudden, associated with deafness and noises in the ear.

Pathology and Etiology.—In 1861 Menière described some cases in which vertigo was produced by a sudden lesion of the labyrinth. Since then, the term Menière's disease has come to be applied to all cases of sudden vertigo associated with labyrinthine disease. Gowers says that, "In nine cases out of ten in which there is definite giddiness, not epileptic in nature or obviously due to organic brain disease, it is due to a morbid state of the labyrinth or auditory nerve endings." Thus, the vertigo becomes the result of the irritation of the nerve.

In addition to clinical sources for confirmation of this view, is the fact that experimental lesions in the semicircular canals of animals result in vertiginous movements. In point of fact, aural vertigo results from almost any of the morbid processes possible to the labyrinth and the nerve endings it contains, but not from disease of the middle ear. The precise nature of the morbid change can only be conjectured. It is twice as frequent in men than in women, and four-fifths of all cases occur between the ages of thirty and sixty. Cold, gout, and syphilis have been followed by it, probably through inflammation, and possibly resulting hemorrhage. The slower forms may be due to degenerative processes, like those of tabes or such as are due to age. Vaso-motor neuroses of the vessels of the labyrinth have been held responsible.

Symptoms.—The vertigo is usually sudden and paroxysmal, though there may be slight continuous dizziness between paroxysms, which occur at intervals of from a few days to as many weeks. Occasionally they are daily. They may be spontaneous or an exciting cause of trifling character may bring them on, such as turning, coughing, or sneezing. Gastric disturbances may excite them—a fact to be remembered in diagnosis from gastric vertigo. There may be brief unconsciousness. The attacks generally pass off in a few minutes, leaving the patient pale, faint, and nauseated, often in a cold, clammy sweat. Vertigo may or may not be accompanied by a tendency to fall forward or backward, or to one side, and the victim may have to grasp something to save himself from falling. External objects may appear to circle about him. The seeming movements of person and external objects are usually in the same direction.

The auditory symptoms—deafness and tinnitus—may be in one or both ears, and more in one side than in the other. In the latter case the sense of movement may be toward or from the ear most affected, but when the subjective and objective movements coincide in direction they are more often toward the affected side.

The deafness is nervous and always partial. The tinnitus is usually roaring or throbbing. There may be ocular symptoms. They are secondary, and include nystagmus and diplopia. Pressure on the drum or the meatus may bring on the nystagmus and sometimes an apparent jerky movement of objects. Diplopia, nystagmus, and jerky movements may occur together.

Diagnosis.—The essential symptoms of Menière's disease are dizziness, tinnitus, and deafness. Gastric disturbance is not peculiar to it. The deafness must be proven to be nervous and not the result of defective air conduction. True gastric vertigo is not associated with deafness, while other symptoms of dyspepsia are present with it.

While the aura of epilepsy is sometimes accompanied by giddiness, there is no impairment of hearing. Moreover, in Menière's disease, slight vertigo is more or less constant, the tinnitus persistent, and loss of consciousness, if present, is very brief. It is the *petit mal*, with its brief unconsciousness, with which the confusion may occur.

The vertigo of cardiac valvular disease, especially aortic insufficiency, and of arterio-capillary fibrosis, and chronic interstitial nephritis, is unaccompanied by any of the other distinctive signs of Menière's disease. Gelier's vertigo, characterized by attacks of paretic weakness of the extremities, ptosis, and profound depression, but without loss of consciousness, occurring especially among laborers in the canton of Geneva, should be mentioned as a possible source of error.

Prognosis.—This depends upon the degree of permanence of the lesion causing the malady. In cases due to remediable causes—such as gout and even syphilis—recovery is possible, while palliation is not infrequently attained. Other cases are obstinate and incurable. Relief, however, comes to the dizziness when the deafness becomes total.

Treatment.—When traceable to gout and syphilis the remedies appropriate to these diseases should be prescribed. The salicylates and

iodides are most frequently useful, but the lithium salts and colchicum are to be remembered. The salicylates should be given in moderate doses rather than the large ones, which produce the ringing in the ears. In the absence of a knowledge of definite cause the bromides are the remedies to be most relied upon. Twenty to 30 grains (1.3 to two gm.) should be given at a dose, and Gowers recommends the addition of a few minims of the tincture of belladonna. Nitro-glycerine has been recommended. The general health should be looked after.

Counter-irritation by blistering behind the ear is sometimes followed by prompt results.

LESIONS OF THE NINTH OR GLOSSO-PHARYNGEAL NERVE.

Anatomical.—This triply-mixed nerve supplies sensibility to the soft palate, the tonsils, the upper part of the pharynx, the Eustachian tube, and tympanic cavity; motor impulses to the stylo-pharyngeus and the middle constrictor of the pharynx, and the sense of taste to the posterior 3d of the tongue and the palate.

The study of the precise pathology of this nerve is rendered difficult by its numerous communications with other nerves, notably with the 5th, the facial, and the pneumogastric; and the fact that it is rarely involved alone. Experimental inquiry with it is also difficult. There is reason to believe that it receives its taste fibres from the 5th, since lesion of its roots has never been attended with loss of taste.

The nerve may be impinged upon by meningitis, tumors, degenerations, and the like.

Symptoms.—Symptoms of such lesion would be difficult deglutition and perversion of the sense of taste—parageusia—or complete gustatory anæsthesia.

Modifications of the sense of taste are tested by means of sapid substances in solution, applied to the anterior and posterior parts of the tongue by a glass rod or brush; suitable substances being used for each taste. Thus, for bitter, a solution of quinine may be used; for sweet, a solution of sugar; dilute acetic acid or vinegar for acid, and common salt for the saline taste.

Ageusia may result not only from lesions of the glosso-pharyngeal nerve, but also of the gustatory or lingual branch of the 5th, and of the 5th itself within the cranial cavity; from affections of the chorda tympani in disease of the middle ear; of the facial between the entrance of the chorda tympani and the geniculate body, and in lesions of the peripheral, terminal, and organs of the nerves of taste. Disturbances of taste have been noted in affections of the posterior portion of the internal capsule.

Perversion of the sense of taste is known as "parageusia." It is a rare phenomenon, found in patients with facial palsy, in the hysterical, and the insane, in whom, also, subjective sensations of taste may occur. The latter also occurs as an aura in epilepsy. Hyperæsthesia of taste is even more rare, and is a purely hysterical symptom.

LESIONS OF THE PNEUMOGASTRIC OR VAGUS NERVE, THE TENTH PAIR.

Anatomical.—This nerve has by far the widest distribution of any of the cranial set, supplying the pharynx, larynx, lungs, heart, œsophagus, and stomach, and in part, also, the intestines and spleen. The symptoms of its involvement are, therefore, numerous and varied.

It is a mixed nerve of motion and sensation, some of its most important motor functions being derived from the spinal accessory. It is the chief sensory nerve for the respiratory centre in the medulla oblongata, but contains, also, accelerating and inhibitory fibres for this centre. The former office preponderates, so that section of the nerve renders respirations less frequent, though deeper, while stimulation of the divided central end accelerates them, and acceleration may proceed to tetanic arrest. The inhibitory fibres are contained chiefly in the superior laryngeal nerve, stimulation of which arrests breathing with the muscles in a state of relaxation.

It is also the inhibitory nerve of the heart, slight stimulation increasing the length of diastole, while stronger stimulation arrests the action. On dividing the nerve cardiac contractions become more frequent. It is also inhibitory for the vaso-motor centre, and its stimulation produces relaxation of the arteries throughout the body. It is the motor and sensory nerve for the œsophagus, sensory nerve for the stomach, and partly the motor nerve for the stomach and intestines.

Lesions Involving the Nucleus and Trunk of the Pneumogastric and Branches.

The *nucleus* in the medulla may be involved in softening, hemorrhage, or slow degeneration, but adjacent nuclei are also affected at the same time, whence resulting effects are associated, and are especially seen in bulbar palsy.

The *trunk* of the nerve near its origin may be compressed by thickened meninges, tumors, or aneurism of the vertebral artery. In its course it has been implicated in incised wounds, and tied in ligature of the carotid. Neuritis and neuromata are possible. The results of such lesion are commonly paralytic, rarely irritative. The former, if total, are diminished breathing rate, "suffocation," frequent pulse rate, death. The results of partial paralysis are better considered in connection with lesions of the separate branches of the pneumogastric, some of which are also invaded separately.

Lesions of the Pharyngeal Branches.—These branches of the pneumogastric, together with branches of the glosso-pharyngeal, form the pharyngeal plexus, from which the muscles and mucous membrane of the pharynx are innervated.

Etiology.—Nuclear disease is a most common cause of paralysis of the pharynx. It shares with disease involving adjacent nuclei, constituting

bulbar palsy, already considered ; but it may also be caused by meningitis, or bone disease at the base of the skull, or it may form part of the lesion in diphtheritic paralysis.

Symptoms.—The results are mainly paralytic ; occasionally irritative, producing spasm. The symptoms of *paralysis* are difficulty in swallowing, food lodging instead of descending into the œsophagus. A most frequent consequence is the entrance of food into the larynx, causing spasm and even choking. Pulpy food is better swallowed than liquids, the latter passing easily into the posterior nares when there is paralysis of the soft palate ; and even when the paralysis is limited to the superior constrictor of the pharynx owing to contraction of the middle constrictor. When the nerves on one side only are involved, the difficulty is much less. Should there be a doubt in diagnosis between paralysis of the pharynx and obstruction by morbid growth the passage of a bougie will clear it up.

Spasm of the pharynx is always functional in origin, chiefly hysterical. The so-called "globus hystericus," or sensation of a ball in the throat which has to be swallowed down but immediately rises again, is one of its manifestations ; so is eminently the spasm in hydrophobia. Extreme degrees are those in which persons cannot swallow their food in the presence of others.

Lesions of the Laryngeal Branches.—These are two, the superior, and inferior or recurrent laryngeal. The former supplies the mucous membrane above the vocal cords, the crico-thyroid, and the depressors of the epiglottis. The inferior or recurrent laryngeal on the left side winds around the arch of the aorta ; on the right around the subclavian. The nerves then pass up to the larynx between the trachea and the œsophagus, supply all the laryngeal muscles except the crico-thyroid and epiglottic, and the mucous membrane below the cords ; also that of the trachea. All of the motor fibres in these nerves come from the spinal accessory ; the sensory pass to the medulla in the roots of the pneumogastric.

In order to appreciate the phenomena of paralysis of the larynx it should be remembered that the glottis is opened or closed only by the movement of the posterior extremity of the cords, the anterior remaining always fixed, and that this movement is effected chiefly by the arytenoid cartilages attached to the cricoid by an articulation which permits free movement. Each arytenoid is shaped like an irregular pyramid prolonged at the base into two processes,—an anterior or vocal, from which the cord passes to the thyroid cartilage, and an external or muscular, to which the muscles are attached. When the latter, which is at right angles to the vocal process, is moved back this process moves outward from its fellow, the cord is abducted, and the glottis opened. If the muscular process is moved forward the vocal is moved inward toward its fellow, the cord adducted, and the glottis closed. These movements are further aided by movements of the arytenoids away from or toward each other.

Symptoms.—These are phonic, respiratory, and altered position of the cords, as recognized by the laryngeal mirror. The voice may be

changed or lost, the entrance of air in breathing impeded, while the closure of the glottis, necessary to coughing, is usually imperfect. The voice and respiratory functions of the larynx are regulated by the same muscles and nerves, but by centres that differ in anatomical connection, if not in position.

In breathing, the cords are abducted or separated during inspiration, the extent being proportioned to the force of inspiration. During expiration they are a little nearer than in inspiration. In phonation they are made tense and brought together, the degree of adduction and tension varying with the note produced. After death the vocal cords assume a position of slight abduction from the middle line, a little nearer than during ordinary breathing, known as the cadaveric position. The position is one of relaxation, approximated, but never fully attained, during life.

The symptoms of deranged function of the laryngeal nerves admit of classification into those of *paralysis* and *spasm*.

(*a*) *Total Paralysis.*—In what is known as complete paralysis of the laryngeal muscles—which does not, however, usually include the crico-thyroid—the vocal cords assume the cadaveric position (page 407), from which they cannot be moved. Hence, vocal sounds cannot be produced. In deep inspiration the current of air may bring them a little closer, and there may be slight stridor, and instead of the natural explosive cough there is only a sudden rush of air through the glottis. If one cord is paralyzed, it alone is motionless in the cadaveric position. Some phonation may now be possible, because the unaffected cord may be over-adducted beyond the middle line, but the voice is low-pitched and often hoarse. During inspiration the abduction of the healthy cord prevents stridor, while an explosive cough is impossible because of the glottis not being closed with sufficient firmness to produce it, unless the paralysis is very slight.

The *causes* of complete paralysis are central disease, disease of the trunk of the vagus, or of the recurrent pharyngeal.

(*b*) *Bilateral Abductor Paralysis.*—In abductor paralysis involving the posterior crico-arytenoids the cords are near together in the position of phonation, and cannot be abducted even as far as the cadaveric position. They can, however, be brought together in phonation and in coughing, at the cessation of which they recede a little, but the normal wide abduction of inspiration does not take place. This slight recession is due to the elasticity of the attachment of the cords. The adductors, unopposed, undergo secondary contracture, so that if the paralysis is of long duration the chink of the glottis becomes permanently narrower. The tensors are still active, as well as the adductors, hence the voice is little affected. The chief difficulty is with breathing, since the normal recession of the cords essential in inspiration does not take place, while they are even brought closer together by the pressure of the entering air. Hence, inspiration is accomplished with stridor, and the obstruction to the entrance of air brings into play the extraordinary muscles of respiration, the effect of which is to prolong the inspiratory act. Expiration is unimpeded, the current of outward air tending to open the cords. The ab-

sence of voice involvement and of cough may cause the obstruction to be referred to the trachea, but the absence of expiratory stridor excludes this, while the movement of the larynx up and down during breathing is greater than in tracheal stenosis. The added urgent dyspnœa, the loud inspiratory stridor, livid features, and cold extremities, finish an unmistakable picture; so that a laryngoscopic examination is not necessary to complete the diagnosis. In bilateral palsy there is even great danger, as a little catarrhal swelling may close the larynx, and tracheotomy may be necessary to save life.

The *causes* of abductor paralysis are central disease and local influence, such as laryngeal catarrh and degeneration of the posterior cricothyroids, possibly of toxic origin. Disease of the recurrent laryngeal has produced such paralysis, although this nerve supplies fibres to the adductors as well as abductors. On the other hand, the abductors have been found degenerated when the other muscles were found normal. Paralysis of both cords is generally due to the disease of both nerves, and may be produced by pressure on both vagi and both recurrent laryngeal nerves. Central causes are tabes and bulbar palsy. Abductor paralysis is also a rare symptom in hysteria, when it is bilateral, with characteristic symptoms, and has caused death.

(*c*) *Unilateral Abductor Paralysis.*—In this the affected cord is near the middle line, and it does not move in inspiration. There is hoarseness and roughness of voice and sometimes dyspnœa, but the mobility of the other cord permits the function of the larynx to be carried on with tolerable comfort. If the adductors become involved, as is sometimes the case, phonation is still more impaired.

The most frequent cause is aneurism involving the left cord,—though other tumors may cause it,—and on the right side the nerve may be involved in a thickened pleura.

(*d*) *Adductor Paralysis.*—In adductor paralysis due to involvement of the lateral crico-arytenoid and the arytenoid muscles the cords are apart and cannot be approximated. In true adductor paralysis, there is still the power of separating the cords on deep inspiration, but no power to bring the cords nearer than in the cadaveric position.

The *causes* of adductor paralysis are rarely organic disease of the nerves or centres. It is the condition causing the oft-quoted *hysterical aphonia*, and may be brought on by over-use of the voice and catarrhal laryngitis. The patient with hysterical aphonia can sometimes sing, though she can talk only in a whisper. It is most common as a partial paralysis. While the cords cannot be approximated for the phonation, they can be in coughing. Hence it was called by Turck phonic paralysis. Another partial adductor paralysis is due to the loss of power in the arytenoid muscle, resulting in defective closure of the posterior part of the glottis and hoarseness or loss of voice.

(*e*) *Tensor Paralysis.*—Little is known of this, except that palsy of the internal fibres of the thyro-arytænoidæus causes the edge of the cord to be concave.

Diagnosis.—The laryngoscope is necessary to a proper diagnosis of laryngeal palsies, but symptoms are also useful. The inability to produce

explosive cough is of great value in pointing to palsy of organic origin, if there is no local lesion to prevent it.

(1) Entire loss of voice and absence of cough point to bilateral palsy of organic origin.

(2) No cough, voice low pitched and hoarse, paralysis of one cord.

(3) Loud inspiratory stridor without loss of voice, total abductor paralysis.

(4) Little change of voice or cough, unilateral abductor paralysis.

(5) No voice, perfect cough, no stridor, unimportant adductor palsy.

The following table from Gowers contains the symptoms, laryngoscopic picture, and lesions in separate columns :—

Symptoms.	Signs.	Lesion.
(1) No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
(2) Voice low pitched and hoarse; no cough; stridor absent or slight on breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line in phonation.	Total unilateral palsy.
(3) Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
(4) Symptoms inconclusive; little affection of the voice or cough.	One cord near the middle line, not moving during inspiration; the other normal.	Unilateral abductor palsy.
(5) No voice; perfect cough; no stridor or dyspnœa.	Cord normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

Spasm of the Larynx.—In spasm of the larynx the adductors are alone concerned. The closers of the glottis are stronger than the openers, while reflex mechanism is connected chiefly with those muscles because of the importance in guarding against the entrance of foreign bodies into the larynx. Spasm is quite common in children, especially the rickety, and not rare in adults under the name of laryngismus stridulus. It is generally reflex, although the reflex cause is not always discoverable. The patient commonly wakes up at night in an attack of intense dyspnœa; but it may occur at any time. The symptoms are like those of ordinary croup.

The paroxysm differs from that of abductor paralysis in that the stridor accompanies expiration as well as inspiration. The attacks occur in locomotor ataxia, in the so-called laryngeal crises, in tetany, in the paroxysms of hydrophobia, sometimes in alternation with attacks of migraine, and in hysteria.

Spasm is also sometimes excited by attempts to speak, when aphonia results. The condition is the reverse of phonic paralysis, in which the

cords cannot be brought together in speaking, while in spastic aphonia they come together too forcibly.

Disturbances of the sensory innervation of the larynx is mostly confined to the irritation which causes cough and spasm.

LESIONS OF CARDIAC BRANCHES.—The cardiac plexus is made up of fibres derived in part from the pneumogastric, and in part from the sympathetic. The vagus fibres are motor, sensory, and probably trophic.

The *motor fibres* include those which inhibit, control, and regulate the cardiac action. Their irritation inhibits the heart's action and causes slowness of the pulse, or bradycardia. In complete paralysis of the vagi the inhibitory action is abolished and the accelerator influence is unhampered, producing rapid pulse, or tachycardia. Yet it sometimes happens that complete paralysis of the vagus is followed by no cardiac symptoms.

The causes of these effects are, unfortunately, not always discoverable. Pressure of a tumor, accidental ligature of one vagus, irritation of its nuclei, anginal attacks, in one instance associated with a small tumor of the vagus, have all been followed by bradycardia. Toxic blood-states are also held responsible for it. Some persons are able to control the action of their own hearts, notably a Col. Townsend, who could control the action of his heart at will. The heart may sometimes be slowed by pressure against the pneumogastric in the neck.

The opposite condition, tachycardia, has been produced by diphtheritic neuritis, tumors of the vagus, or its accidental removal.

Sensory phenomena in connection with parts supplied by the cardiac branches of the pneumogastric are unusual, but any uncomfortable sensations arising from palpitation or irregularity are conveyed by branches of the pneumogastric.

Trophic influence in the pneumogastric is inferred from the fact that the heart has been found in a state of fatty degeneration after injury to the nerve.

LESIONS OF GASTRIC AND ŒSOPHAGEAL BRANCHES.—Among phenomena ascribed to effect on these branches are spasm of the œsophagus and difficulty in swallowing. The vagus is also the sensory nerve of the stomach, and pain in this organ is felt through this nerve. The severe gastric crises which occur in locomotor ataxia are due to central irritation of the vagus-nuclei. The senses of hunger and thirst are also believed to be conveyed through it, and have been lost in disease involving the root, but appetite is not always lost after section of the nerve, while in some cases of disease of the nerve there has been excessive appetite. On the other hand, loss of appetite is due to so many causes that it cannot be ascribed to pneumogastric lesion without careful investigation.

The pneumogastric is also the motor nerve of the stomach, though motion of the organ is not entirely arrested after its section. Vomiting is probably produced through its agency, and is excited by central and reflex irritation. Meningitis, which so frequently excites vomiting, does so through it ; the pressure of a tumor on the nerve has had a like effect, also direct pressure on an exposed nerve.

Lesions of Pulmonary Branches.—While the vagus sends branches to the lungs little is known of their office. They are supposed to go to the bronchial muscles, and it is held that asthma is a neurosis of these fibres. Irritation of the afferent pulmonary fibres certainly produces spasm. Stimulation of the respiratory centre also causes energetic respiratory movements, while rapid congestion and even hemorrhage have been noted after section, though these effects may possibly be of reflex origin excited through the sympathetic, since the vaso-motor fibres in the vessels of the lungs are derived from the sympathetic. After section of the vagus, animals die of chronic pneumonia, not, however, as the result of trophic influence, but because of the entrance of foreign particles into the bronchi, in consequence of paralysis of the larynx and œsophagus.

The phenomena of hiccough may be the result of disease of this nerve, as they are of the respiratory centre.

Prognosis in Pneumogastric Lesions.—This varies greatly. In central and nuclear disease it is unfavorable; unfavorable also where the result of pressure from intrathoracic tumors, especially aneurism. In hysterical and purely local affections the prognosis is more favorable.

Treatment.—This is of course that of the causal lesion, if it can be discovered. Syphilis is the more curable of the central causes. Other causes of central disease are not removable.

Of diseases of the trunk, neuritis of the vagus is as amenable to treatment as the polyneuritis of which it is a part. The laryngeal symptoms due to involvement of the recurrent laryngeal are as remediable as the causes which produce them. If they are caused by aneurism of the aorta, or cancer, treatment is useless; if caused by syphilitic and scrofulous growths the prognosis is more hopeful.

In the paralysis of more purely local origin, especially the hysterical, phonic, and diphtheritic form, electricity offers the most promising results. The method of its employment will be found detailed under diseases of the larynx. Either form of electricity may be used. Strychnine is a useful remedy, especially in the local forms mentioned. The method preferred is by hypodermic injection, the nitrate being employed in doses of $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.0011 to 0.0022 gm.) daily.

In addition to strychnine, other tonics should be used to restore the general health of the patient; laryngeal gymnastics have been recommended and used with some success. They consist in pressing firmly with the thumb and forefinger on each side of the upper and hinder part of the thyroid cartilage, the patient being requested to make a simple sound during the compression.

The treatment of laryngeal spasm demands also the removal of the cause if possible, in addition to which sedatives, local and general, especially the bromides and cocaine, may be used. Chloral, chloroform, and nitrite of amyl by inhalation, may be necessary to break up the spasm.

LESIONS OF THE ELEVENTH PAIR OR SPINAL ACCESSORY NERVE.

Anatomical.—This nerve, purely motor in its function, has two portions—an internal, which passes to the pneumogastric and innervates the laryngeal muscles, and an external or spinal portion. The former has been considered. The latter is essentially a set of motor fibres from the cervical spinal cord, which ascends into the cranial cavity and passes out again with one of the cranial nerves to be distributed to the sterno-cleido-mastoid and trapezius muscles, whose innervation they share with the dorsal nerves. The purpose of the trapezius is chiefly to raise the shoulder, that of the sterno-cleido-mastoid to turn the head to the opposite side, the chin being at the same time raised. This is accomplished by drawing the occiput toward the side of the muscle acting.

Lesions.—The nuclear origin of the nerve may be involved and contribute to the phenomena of bulbar palsy, or it may share in progressive central degeneration, causing wasting in the muscles supplied, which may be a part of a more general muscular atrophy. The trunks of the nerve, or both nerves, may be compressed in the foramen magnum by meningitis. Outside the skull there may be wounds, tumors, caries of the vertebræ, and resulting abscesses, and sometimes abscesses springing from the cervical glands. Rarely the spinal accessory may be invaded by rheumatic neuritis.

The resulting conditions are paralysis and spasm. Those of the internal or accessory portion have been described under lesions of the pneumogastric. It remains to consider only those of the external branch.

Symptoms of Paralysis of the External Branch of the Spinal Accessory.—The seats of the paralysis are the sterno-mastoid and trapezius muscles. Where one sterno-mastoid is involved the head is moved with difficulty to the opposite side, but there is no wry neck or torticollis, though in some cases the head is held obliquely. The trapezius is not so much involved because it is well supplied with cervical and dorsal nerves, but the portion which passes from the acromion to the occipital bone is motionless. The middle portion of the muscle is also weakened, the shoulder droops, and the angle of the scapula is rotated inward by the action of the rhomboids and the levator anguli scapulæ. Elevation of the arm is also partial, because the trapezius does not fix the scapula at a point whence the deltoid can work. The paralysis is well seen when the patient takes a deep breath or tries to shrug the shoulders. Wasting almost always accompanies the loss of power.

In bilateral paralysis the power of holding the head in the upright position is impaired. If both sterno-cleido-mastoids are affected the head tends to fall backward; if both trapezii, it falls forward so that the chin rests on the sternum. The latter is the characteristic position of the head in progressive muscular atrophy, and in children who have chronic meningitis about the foramen magnum, pressing on both nerve trunks, and in cervical meningitis the result of caries. A peculiar drooping of the head

is sometimes seen during the first year of life of children, which Gowers says may be due to injury to the spinal accessory nerves in difficult labor. In recent cases the nerves may give characteristic reaction of degeneration. In central disease the reaction varies, as it does in progressive muscular atrophy.

Treatment.—This must have for its object, first, the removal of the cause, or the morbid process which produces it. After this, the weak muscles are to be treated by massage and electricity. Faradization is, perhaps, most efficient for this purpose, and either form of current will answer.

Symptoms of Accessory Spasm (*Torticollis: Wry Neck*).—Though the muscles supplied by the spinal accessory are not the sole ones responsible for these conditions, they are the ones chiefly concerned. The terms are applied to unnatural positions of the head resulting from contraction of these muscles. There are two principal varieties:—

(1) Fixed wry neck, or congenital torticollis.

(2) Spasmodic wry neck.

These two may be regarded as *true* torticollis, and are to be distinguished from two somewhat similar states which may be called *false* torticollis. The first of these is the ordinary "stiff neck," which is really a rheumatic condition due to exposure to cold, and characterized by pain and tenderness, for the relief of which the position is assumed, and should not be called wry neck. The second is a twist-neck, not due to muscles, but to some other cause, most frequently disease of the cervical vertebræ. This deviation puts the sterno-cleido-mastoid on the stretch, and thus may give rise to the impression that it is responsible.

(1) Congenital Torticollis, or Fixed Wry Neck.—This depends on the shortening of some muscle, commonly the sterno-cleido-mastoid, which is also often atrophied, hard, and firm. It is met with most frequently in children, and is thought to be due, in some cases at least, to injury of the muscle produced by traction during birth. In others it is ascribed to developmental shortening of the muscle, due to the inclined position of the child's head in the pelvis. It is not always noticed immediately after birth because of the natural shortness of the child's head. A similar condition may result from injury to the muscle during life, producing inflammation and cicatricial contraction. It affects the right side almost exclusively. It is more or less constantly associated with facial asymmetry, first noted by George Wilks, and further studied by Golding Bird, who suggested that the two conditions are parts of one affection, which has a central origin. In fixed wry neck the head is turned toward the side opposite to that of the contracted muscle, which stands out conspicuously and cannot be turned toward the latter. While the sterno-cleido-mastoid is the muscle almost invariably responsible in these cases, the trapezius is occasionally the seat of similar atrophy.

Treatment.—The treatment is by section of the contracted muscle. Some appliance may be necessary for a time to keep the head in proper position, especially where secondary changes in the articulation have taken place. I have used in simple rheumatic wry neck an appliance

consisting of webbing or "saddle-girth" about three inches wide, stretched from side to side of the bed and raised a few inches above the mattress—the distance to be regulated by circumstances—on which the patient lay at night, instead of a pillow on the side to which the head is drawn. This expedient may be used after operation. The facial asymmetry is apt to remain after the wry neck is cured, and may even become more conspicuous.

(2) Spasmodic Wry Neck.—This is a condition analogous to the facial spasm occurring as a symptom of disease of the facial nerve. There are two forms, the tonic and clonic, which may alternate in the same case or, as is most usual, occur separately and remain so.

Etiology.—It is for the most part an affection of adults, and, according to Gowers, is more common in females, that is, in 22 out of 32 cases. While this must be true of England, the opposite seems to be the case in this country, since of eight or ten cases observed by Osler in Philadelphia and Montreal all were men. It is more common in middle life, two-thirds of all cases occurring between the ages of thirty and fifty. In women under thirty it is apt to be of a hysterical origin; rarely it is ascribable to the same cause in boys. It is prone to occur in neurotic families. Very rarely it occurs in the first year of infantile life, ceasing after a few months. Cold has been assigned as a cause; also traumatism.

In the *tonic* form when the sterno-cleido-mastoid is responsible the head is continually turned to the opposite side, the chin raised, and the occiput drawn down toward the affected side—the *caput obstipum spasticum.* When the trapezius is involved the head is still more depressed toward the same side. In combined and bilateral spasm of these muscles the head is drawn backward, producing the *retrocollic spasm.* In prolonged cases the muscles involved are prominent and rigid, and there may be spinal curvature with the convexity toward the sound side.

In the *clonic* form there are paroxysmal twitchings of the head, which may be very severe and correspondingly distressing. When there is predominating unilateral spasm of the sterno-cleido-mastoid the head is turned to the opposite side and the chin raised with every contraction of the muscle. In unilateral spasm of the trapezius the head is drawn more backward with each contraction and toward the shoulder of the affected side. In bilateral and combined spasm there is clonic retrocollic spasm, with shaking and nodding movements—the so-called "salaam convulsions" sometimes seen in children. They may be produced also by contractions of the other muscles of the neck. Tonic and clonic spasm of the splenius may occur either alone or in combination with that of the trapezius and sterno-cleido-mastoid. In splenius spasm the head is also drawn backward and toward the affected side, and there will be noted muscular swelling to the outside of the cervical portion of the trapezius. The splenius is, according to Gowers, associated with the sterno-mastoid about half as often as the trapezius. The retrocollic spasm is commonly associated with a wrinkling of the forehead in both the tonic and clonic form.

In the clonic form the contractions may come on suddenly, or be preceded by stiffness and irregular pain. The movements occur every

few minutes, and the head cannot be kept still, although the movements cease during sleep. They are increased by emotion, excitement, or fatigue. There is sometimes pain, but at others merely a sense of fatigue. The muscles in time may become hypertrophied, but never waste.

Pathology.—This is very obscure. Reasoning, rather than demonstration, leads to the conclusion that the muscular contractions probably depend on the over-action of nerve cells, and not on irritation of nerve fibres; while sometimes those of the higher or cortical centres, and sometimes the lower spinal centres, are concerned.

Diagnosis.—The distinction is between true and false torticollis, in which there is deviation of the head from some other cause than muscular contraction, and it is only the form of true torticollis due to shortening of one sterno-cleido-mastoid which is likely to be confounded with the false. In the spurious form the sterno-mastoid is tense on the side toward which the face is turned, and in the true form the tension is on the side opposite. In retrocollic spasm the invariable association of contraction of the frontalis muscles, producing the peculiar wrinkling of the forehead, distinguishes it from simple tremor. The hysterical form occurs in women under thirty, and this fact is presumptive evidence of its presence, while hysterical spasm is also apt to spread from the neck to the trunk; in the true torticollis form it is limited to the neck.

Prognosis.—The prognosis is always grave, and the more severe and extensive the spasm the more unfavorable. Relief is more possible in the first half of life than the second. Cases do, however, occasionally get well, and temporary relief is more frequent.

Treatment.—If cause can be found which is responsible it ought to be removed. If discovered in an acute stage, rest and fomentations or dry heat are indicated. Electricity has, perhaps, more reputation than any other remedy. The faradic brush may be applied over the skin of the affected muscles, and to the swelling. Gradually increasing faradic currents may be used. If the galvanic current is used, a weak one is preferred, and the anode, or positive pole, is placed below the occiput or highest accessible part of the nerve, and the negative on each contracting muscle, for ten minutes at a time.

Sedatives and narcotics have also some reputation. Among these the bromides and Cannabis Indica are included in large doses. Five-minim (0.3 c. c.) doses of the fluid extract of Cannabis may be given, rapidly increased. The drug is proverbially unreliable. The hypodermic use of morphine is of undoubted value in relaxing the spasm, but the dangers of its protracted use almost preclude it. It would be unfair to the drug, however, to omit the statement of Gowers that "Continued for several months in doses increased gradually to half a grain or a grain a day it has entirely removed the spasm." Naturally such persons are weaned from the drug with difficulty.

Mechanical supports for fixing the head are recommended, but are not well borne. Surgical measures have been employed,—such as section and exsection, and stretching of the nerve and section of the muscle,—with, at best, but temporary results. Mention should be made, however, of the deep-seated operation of W. W. Keen and Noble Smith, which

consists in dividing the nerve, and the posterior branches of two or three cervical nerves which also supply the splenius and complexus. This reduces the spasms that reside in these muscles to a slight degree, while the otherwise paralyzing effect of the division of branches of the spinal nerves is comparatively unimportant.

LESIONS OF THE TWELFTH PAIR OR HYPOGLOSSAL NERVE.

Anatomical.—This is the motor nerve of the tongue, and supplies also the depressors of the hyoid bone and the hyoglossus and geniohyoid of the elevators. It arises from the medulla oblongata beside the olivary body. Its cortical centre is probably the lower part of the ascending frontal gyrus or the root of the 3d frontal convolution. It is subject to paralysis and spasm.

Etiology.—(1) *Cortical disease* is frequently responsible for paralysis of the tongue on the opposite side, as is seen in the numerous cases of hemiplegia associated with this condition. The same accident occurs when the fibres between the cortex and the nucleus in the medulla are invaded. Apoplexies and other causes of compression, softening, thrombosis, and embolism, are agencies operating to this end.

(2) *Nuclear disease* is another cause. It is usually degeneration, rarely sudden softening; the former as a part of bulbar palsy and locomotor ataxia, and the latter from vascular obstruction. The effect is almost always bilateral, the nuclei being so close together that it is scarcely possible to involve one only, although such isolated result has occurred in sudden cases and, rarely, in slow ones, as in tabes and general paralysis.

(3) *Infra-nuclear disease* may operate at various sites—

(*a*) Within the medulla the root fibres may be invaded by a tumor or by softening.

(*b*) Outside the pons the fibres may be damaged by the products of meningitis, simple or syphilitic, and by new formations. The nerve may be compressed in its foramen by outgrowth of bone. Outside the skull the nerve is compressed by tumors, by inflammatory products, or injured by disease communicated from caries of the upper cervical vertebræ, and by penetrating wounds. Hence, the spinal accessory often suffers coincidently and there is paralysis of the palate, occasionally of the vocal cords with or without wasting of the trapezius and sterno-mastoid. The hypoglossal may be the seat of neuritis.

Symptoms.—(*a*) *Of Hypoglossal Paralysis.*—These are motor only. When there is supra-nuclear disease in addition to the palsy of the tongue there is hemiplegia, but no wasting of the tongue, which is protruded toward the affected side, nor change in electrical reaction. In nuclear disease the lesion is apt to be bilateral palsy. The tongue lies motionless in the floor of the mouth, and speech and deglutition are seriously impaired. Mastication is interfered with mainly because the tongue cannot regulate the position of the food, the proper muscles of mastication being

intact. There are atrophy and reaction of degeneration. The mucous membrane is thrown into folds. The condition is likely to be a part of a bulbar palsy. In infra-nuclear disease only one nerve is affected, there is wasting with reaction of degeneration and fibrillary twitching. Speech is not much impaired, nor swallowing.

(*b*) *Of Spasm.*—Spasm of the tongue as an isolated event is very rare. It may be unilateral or bilateral. It commonly occurs as a part of some other convulsive affection, as epilepsy or chorea, or spasm of the facial muscles. It may also occur in hysteria. In the biting of the tongue in epilepsy the organ is thrust between the teeth by spasmodic contraction of the genio-glossus and caught by the jaws through a spasm of the masseters. Spasm of the tongue occurs in some forms of stuttering, the spasm often preceding the explosive utterance of words. In other cases there are various protrusions and deviations of the tongue, produced in some instances by irritation of the 5th nerve, variously induced, it may be, by a carious tooth. The spasm may be clonic, the tongue being thrust in and out many times in a minute, at others more slowly. It may be associated with facial spasm. It may occur during sleep.

Diagnosis.—This is generally easy. If there is hemiplegia and palsy, but no wasting of the muscles of the tongue, no reaction of degeneration, the lesion is supra-nuclear. If there is paralysis of the tongue on the one side, and of the limbs on the opposite, there is probably a unilateral lesion in the medulla below the nucleus. When the disease is on the surface of the medulla the paralysis is commonly unilateral, and is associated with paralysis of the corresponding half of the palate and vocal cord, because of the involvement of the spinal accessory.

Prognosis.—The prognosis is usually unfavorable because the lesion is incurable.

Treatment.—The treatment embraces that of the disease producing it. The symptom of lingual paralysis may be treated with electricity—with an electrode in the shape of a tongue depressor.

The treatment of spasm has been by sedatives, including bromides, by iodides, and by electricity.

DISEASES OF THE SPINAL NERVES.

CERVICAL PLEXUS.

Affections of the Phrenic Nerve.—Paralysis of this nerve may be the result of a lesion in the anterior horn of the grey matter of the cord, at the level of the 3d and 4th cervical nerves; of a lesion to these nerve roots in disease of the membranes of the cord or of the vertebræ; or by compression by aneurisms or other tumors. Exposure to cold, producing neuritis, may cause it; and it may be a part of a diphtheritic palsy.

Symptoms.—The result is *paralysis of the diaphragm*, which is complete if both nerves are involved, as is the case in disease of the cord or its membranes; partial when a tumor or other cause affects one nerve. Respiration is still carried on by the intercostals, and when the victim is quiet there is little or no embarrassment, but examination shows the abdomen retracted in inspiration and protruded in expiration. In other cases, in consequence of increased movement of the thorax, the upper abdominal walls are drawn outward with inspiration—a movement not to be mistaken for movement of the diaphragm. On exertion, however, there is *dyspnœa*, which is also observed if the paralysis is sudden. The effect of paralysis of a single phrenic involving one-half of the diaphragm is scarcely noticeable.

A further effect is to aggravate any lung affection, as bronchitis or pneumonia. There is difficulty in coughing effectually, and, therefore, of emptying the lungs of mucus, accumulation of which may result in impairment of resonance at the base of the lungs in bronchitis, and in the physical signs of œdema.

Diagnosis.—Nervous breathing resembles the breathing of paralysis of the diaphragm in that this muscle is used very little, while the upper thorax is freely used. If, however, the attention of persons thus breathing is distracted, or they are watched when not conscious of observation, the diaphragmatic breathing will become at once apparent.

The diaphragm does not move when it is inflamed, or in diaphragmatic pleurisy, but it is because of the extreme pain which its motion, under these circumstances, causes.

The diaphragmatic palsy from diphtheritic neuritis is only a part of the symptoms present, and when it is due to spinal disease there is usually atrophy of other muscles, together with other symptoms of that disease.

Prognosis.—This depends upon that of the disease of which it is a part, except in diphtheritic neuritis, where it is the direct result of the disease, and where the prognosis is unfavorable.

Treatment.—The treatment is that of the disease of which it is the result. If there is neuritis, effort should be made to galvanize the nerve by pressing one pole outside the clavicular portion of the sterno-mastoid, and the other pole over the epigastrium or the corresponding half of the diaphragm. Counter-irritation may also be applied in the triangle of the neck, outside the clavicular portion of the sterno-mastoid.

LESIONS OF THE BRACHIAL PLEXUS.

Of the Combined Plexus.—This may be affected above the clavicle by causes producing pressure on the nerve trunks—the five lower cervical and 1st dorsal—after they leave the spine and before they unite to form the plexus. Such causes are tumors and other morbid processes in the neck. More frequently, causes operate below the clavicle, of which the most frequent is prolonged luxation of the humerus, especially under the coracoid process. One or more branches may be thus involved, producing a corresponding amount of paralysis, to which is added wasting of muscles, with reaction of degeneration, and trophic changes in the skin. Fracture of the humerus is another cause. Blows or falls on the shoulder and injuries in the neck may produce the same results, as may also compression during birth. The muscles involved may be the deltoid, supraspinatus, infraspinatus, biceps, and brachialis anticus.

Neuritis of the brachial plexus also occurs rarely as a primary inflammation. More frequently it is an ascending neuritis from a lesion of a peripheral branch, involving, first, the radial and ulnar, and spreading thence upward. The result ultimately may be complete loss of power in the arm. A still rarer disease is neuroma.

Lesions of Individual Nerves.—*Of the Long Thoracic or Posterior Thoracic—Serratus Palsy.*—This nerve is especially subject to pressure through its long course and position, especially in the posterior triangle of the neck. Such pressure may be direct, as by heavy burdens on the shoulder, or as the result of severe muscular effort in carrying or wielding a hammer, or long exertion with the arm raised, as in white-washing a ceiling. The result may be a neuritis. Neuritis may also be caused by cold. The same nerve may be involved in progressive muscular atrophy or polio-myelitis anterior. From natural causes it is more common in men.

The result is a dislocation of the scapula of the corresponding side, which presents a winged appearance in consequence of projection of its angle and posterior border, rendered especially distinct when the arm is moved forward, since the scapula is no longer held to the thorax by the serratus. In severe cases faradic irritability is lost, though voltaic excitability may remain. Severe neuralgic pain may precede the paralysis.

The course of serratus palsy is slow, and sometimes the paralysis is permanent.

Treatment.—The treatment consists in maintaining the nutrition of the muscles by electrical stimulation. Counter-irritation may be applied over the scalenus muscle, because it is in it that the nerve is most frequently injured. The arm should be kept at rest, and to this end should be carried in a sling, embracing the elbow in such a way as to raise the shoulder.

Nerves of the Arm.—(*a*) *Of the Circumflex Nerve.*—This rises from the posterior cord of the plexus and supplies the deltoid and teres

minor, and the skin over the deltoid. It may be injured by dislocations, blows, bruises, pressure by a crutch, or position long maintained as during illness. Neuritis may result from these causes and from cold, or by extension of inflammation from the joint.

There is loss of power in the deltoid and the arm cannot be raised, also a loss of sensation in the skin over the lower part of the muscle. The muscle wastes and the shoulder becomes flattened. The joint may relax and a space arise between the head of the humerus and the acromion. On the other hand, adhesions may form, partly trophic, since the articulation is supplied by the same nerve. Movement may be further impaired by thickening of the ligaments.

Paralysis of the deltoid is to be distinguished from anchylosis, in which the scapula moves with the arm, which it does not in palsy.

(*b*) *Suprascapular Nerve.*—This nerve rises from the trunk formed by the union of the 6th, 5th, and a branch of the 4th cervical, but its own fibres are derived from the 5th and partly from the 4th cervical. It is occasionally injured alone or with the circumflex in dislocation of the humerus, and by falls on the shoulder, or by carrying heavy weights. The result is palsy of the supra- and infraspinatus muscles. The first is of little significance, but the latter causes a defect of rotation outward of the humerus, interfering with many movements, of which one is carrying the hand along in writing. The scapula is rotated so that the lower angle is rotated upward and inward.

(*c*) *Musculo-spiral Paralysis—Radial Paralysis.*—This nerve arises from the posterior cord of the brachial plexus, and apparently derives its motor fibres from the nerve roots forming the plexus except the 1st dorsal. It supplies the triceps, all the muscles of the back of the forearm, the extensors of the wrist and fingers, both the supinators, as well as the skin on the radial side of the back of the hand, back of the thumb, index finger, and half of the middle finger.

It is more frequently paralyzed than any single nerve because of its position—winding around the head of the humerus after it leaves the plexus. It is often bruised by the crutch, producing the so-called "crutch palsy," by blows and fractures, and especially by pressure when sleeping with the arm over the back of a chair or with the arm under the body. Even a sudden and violent contraction of the triceps, as in pulling on a tight boot, may bruise it. More rarely it is the subject of a neuritis from cold.

In a lesion of the nerve high up, all the muscles above named are involved; when near the middle of the humerus the triceps generally escapes. The supinator longus generally suffers, but escapes if the lesion is below the origin of the branch supplying it, and sometimes in partial injury of the nerve higher up. A characteristic symptom of extensor palsy is the "wrist-drop," while the inability to supinate is also striking. Sensation is rarely lost, though there may be tingling without loss of sensibility.

Paralysis of the musculo-spiral is to be distinguished from the wrist-drop of lead palsy, which is, however, bilateral, while the supinators are unaffected and the onset is gradual. Bilateral wrist-drop is common in

other forms of neuritis, especially the alcoholic, but the gradual mode of onset and the involvement of the legs are their characteristics.

The prognosis is usually favorable, the pressure palsy disappearing in a few days, while recovery is the rule even when delayed.

Erb's rules as to prognosis apply as follows: If both faradic and galvanic irritability are maintained, recovery may be expected in fourteen to twenty days; if these are lessened for the nerve and increased for the muscle, while An C > C*a* C, with contraction sluggish, recovery may take place in four to six weeks, sometimes eight to ten weeks. When there is evidence of degeneration of the nerve, the prognosis is more unfavorable, so that recovery may be delayed from two to fifteen months.

(*d*) *Ulnar Nerve.*—This comes through the inner cord of the plexus from the last cervical and 1st dorsal. It is the first of all the brachial nerves to be affected by disease ascending from the dorsal to the cervical part of the cord. It supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, muscles of the little finger, the interossei, lumbricales, the adductor, and inner head of the short flexor of the thumb. Its sensory portion supplies the ulnar side of the hand, back and front—more on the back—two fingers and a half, with one finger and a half on the front.

The course of the nerve, superficial behind the elbow and at the wrist, makes it vulnerable. It may be injured in wounds of the forearm and about the elbow, in dislocations and fractures about the shoulder and elbow, and continued flexion of the elbow. Neuritis is a possible cause.

The hand moves toward the radial side because of paralysis of the ulnar flexor, and adduction of the thumb is impossible, the first phalanges cannot be extended, and in long-standing cases the "claw hand" may be produced, consisting in over-extension of the 1st phalanges and flexion of the others. There may be wasting of the muscles supplied by the nerve. There is loss of sensation in the sensory distribution.

A similar condition of the ulnar nerve may be produced by inflammation of the cervical portion of the cord, which has but to be remembered.

(*e*) *Median Nerve.*—Its motor fibres arise from all the cervical roots that enter the brachial plexus. They supply the pronators, the radial flexor of the wrist, flexors of the fingers—except the ulnar half of the deep flexor—the muscles that abduct and flex the thumb, and two radial lumbricales. The sensory fibres supply the radial side of the palm and the front of the thumb, the first two fingers, half the 3d finger, and the dorsal surface of the same fingers.

Isolated palsy of this nerve is not frequent, but it may be caused by wounds or fractures of the forearm, rarely from injuries of the upper arm. There may be neuritis from compression.

The wrist can only be flexed toward the ulnar side, the thumb is in a state of persistent extension and cannot be opposed to the tips of the fingers. Pronation is impossible beyond the mid-position to which the supinator can bring the forearm; an attempt is made to supplement this by rotating the humerus inward and separating the elbow from

the side. The 2d phalanges cannot be flexed on the 1st, nor the distal phalanges of the 1st and 2d fingers, while in the 3d and 4th fingers this action can be performed by the ulnar half of the deep flexor. There is conspicuous wasting of the thumb muscles, which gives a characteristic appearance.

There may be complete or partial loss of sensibility. If there is anæsthesia it is more marked on the palmar surface.

Treatment of Lesions of Nerves of the Arm.—The first principle of treatment is the removal of the cause, whatever it may be, as determined from the etiology. If neuritis is present it must be treated. Rest by supports or splints may be necessary, on the one hand; electrical stimulation and massage, on the other.

LUMBAR AND SACRAL PLEXUSES.

The Lumbar Plexus.—This is sometimes damaged by growths in the abdomen, especially of the lymph glands, by inflammatory process, by psoas abscess, and diseases of the bones and vertebræ affecting the nerve roots. The obturator nerve may be injured during parturition; the anterior crural nerve by the same cause, by wounds of the groin or thigh, by dislocation of the hip, and sometimes by growths about the spine.

Symptoms.—In paralysis of the *obturator* adduction of the thigh and crossing of the legs are impossible, while outward rotation is also deranged.

In paralysis of the *anterior crural* extension of the knee is impossible; there is wasting of muscles, with anæsthesia of the antero-lateral part of the thigh, and of the inner side of the leg and big toe. There may be pain in the area of distribution.

Paralysis of the *superior gluteal nerve*, which is rare in isolated form, causes loss of the power of abduction and circumduction of the thigh from paralysis of the gluteus medius and minimus.

The Sacral Plexus.—This suffers from compression by growths in the pelvis, pelvic inflammations, and compression during labor. In addition to spontaneous neuritis, there may also be a neuritis ascending to it from the sciatic nerve. The sciatic may be affected by wounds, dislocation of the hip, disease of the bone, and morbid growths. It is also not very rarely the seat of neuroma.

The result of lesion to the *sciatic* varies with its seat. If near the sciatic notch there is paralysis of the flexors of the leg and all the muscles below the knee; while injury below the middle of the thigh involves only the latter muscles, the flexors of the legs escaping. There is anæsthesia of the outer half of the leg, of the sole and greater portion of the dorsum of the foot, but the leg may escape, perhaps through the intermediation of other nerves. Frequently there is wasting of the muscles and other trophic symptoms. In lesion of one sciatic the leg is fixed in extension by the action of the quadriceps extensor, and the patient can walk, even when all the muscles below the knee are paralyzed, the foot being raised by over-flexion of the hip.

The *small sciatic* is implicated only when the pelvic plexus is impinged upon, and it rarely suffers alone. The effect is palsy of the gluteus maximus, with difficulty in rising from a seat, and a strip of anæsthesia along the back of the middle third of the thigh and upper half of the calf.

Injury to the *external popliteal* or peroneal nerve results in paralysis of the tibialis anticus, long extensor of the toes, peronei, and extensor brevis digitorum. There results inability to flex the ankles, or extend the 1st phalanx of the toes, or to raise the foot from the ground in walking —there is foot-drop. Talipes equinus ultimately results, and may be attended with persistent flexion of the 1st, or proximate phalanges from contraction of the unapposed interossei. In walking, the whole leg must be lifted, and there is the steppage-gait of neuritis. In old cases there may also be wasting of the anterior tibial and peroneal muscles. There is also anæsthesia in the outer half of the front of the leg and on the dorsum of the foot.

Lesion of the *internal popliteal* produces paralysis of the popliteus, calf muscles, tibialis posticus, long flexors of the toes, and muscles of the sole. The symptoms are loss of plantar flexion, inability to extend the ankle joint, and, if the disease is high enough to involve the branch to the popliteus, loss of power to rotate the flexed leg internally ; the foot cannot be adducted, nor can the patient rise on tiptoe. Talipes calcaneus results, and the toes may assume a claw-like position from secondary contraction, due to over-extension of the proximal and flexion of the 2d and 3d phalanges. There is also loss of sensation on the outer lower part of the back of the leg and on the sole.

Treatment.—The treatment of lesions of the nerves of the legs is similar to that of lesions of nerves of the arms. Secondary contractures are to be guarded against, being favored by position. Exposure to cold and fatigue should be avoided, as they favor fresh attacks of neuritis.

DISEASES OF THE MEMBRANES OF THE BRAIN.

Although, anatomically considered, the brain is enveloped by three distinct membranes,—the tough dura mater, the delicate arachnoid, and highly vascular pia mater,—the diseases of the membranes are practically confined to the dura on the one hand, and the arachnoid and pia conjointly on the other; the latter two always suffering together. The dura is, however, separable into two layers—a thin internal layer with its endothelial covering, and a looser external layer which serves as a periosteum to the bones; these two layers may suffer separately.

The term pachymeningitis is applied to inflammation of the dura mater, and leptomeningitis to that of the pia and arachnoid; the latter is commonly meant when the word meningitis is used alone.

PACHYMENINGITIS.

External Pachymeningitis.

External pachymeningitis is always acute, and is commonly circumscribed. It usually results from injuries to the head, especially fractures; from caries of the petrous portion of the temporal bone itself, the result of middle-ear disease; or from syphilitic disease of the bone with pus formation. Rarely no cause is discoverable. Rarely pus infiltrates between the two layers of the dura mater. More frequently there is pus between the dura and the bone. This may occur in syphilis, which, too, may cause thickening of the bone.

Symptoms.—These are indefinite and are often obscured by those of its causal disease. They are pain, delirium, sometimes but not always fever, sometimes convulsions, and signs of pressure. Such pressure may or may not be sufficient to cause paralysis of the opposite side.

Treatment.—The treatment is that of the causing disease, with surgical interference to remove pressure and give vent to pus.

Internal Pachymeningitis.

This is usually chronic. Three forms are commonly noted—purulent, pseudomembranous, and hemorrhagic.

Purulent and Pseudomembranous Pachymeningitis are not recognized before death. The former may follow an injury primarily, but commonly it is an extension from inflammation of the pia. Pus between the dura and arachnoid is rare. The latter may occur as a secondary process in infectious diseases.

Hemorrhagic Pachymeningitis.—Hemorrhagic pachymeningitis, or hæmatoma of the dura mater, is a rare but well-recognized condition first described by Virchow; much more common in infirmaries and hospitals connected with almshouses and asylums.

Etiology.—It is probably most frequently a result of chronic alcoholism, though it has been found in chronic insanity without association with alcoholism, especially in general paralysis of the insane; also acute fevers, when it is associated with profound anæmia. Syphilis is a possible cause. It chiefly occurs in males over fifty, but also between thirty and forty.

Pathology and Morbid Anatomy.—The original dictum of Virchow continues for the most part to be held, viz., that it begins as a hyperæmia in the area of the middle meningeal artery, extending thence forward, backward, and downward. The arteries become tortuous, dilated, and surrounded by thickened adventitia, while the capillaries, being overfilled, produce a rose-colored flush on the under surface of the membrane. To this succeeds a delicate web-like tissue containing wide, thin-walled capillaries three or four times the natural width, between which is a delicate reticulum of spindle cells, extending over the greater part of one or both hemispheres. This becomes afterward paler and firmer. Upon this succeeds another delicate vascular layer, succeeded by another and even another. From three to seven layers are thus superposed until a product of from $\frac{1}{8}$ to $\frac{1}{5}$ in. (three to five mm.) in thickness results. The delicate-walled capillaries, however, easily give way, causing hemorrhages, which vary in extent from mere points to large collections of blood—the smaller being interstitial and the larger between the youngest vascular layer and the next older. The proportion of blood and organized membrane varies greatly, now one predominating and now another. At times there seems to be blood only. Huguenin claims that the hemorrhage is the initial event.

Both products are subject to degenerative changes, the effused blood being disintegrated and partially absorbed, while the blood-vessels become obliterated and substituted by lines of pigment deposit along their course. There may also be serous infiltration, cystic degeneration, and even diffuse suppuration.

Symptoms.—*Hallucinations* are a characteristic symptom, but whether the result of the condition itself or of the disease of which it is a part is uncertain. Beyond this the symptoms are indefinite. There may be *apoplectiform seizures* coincident with fresh hemorrhages, *drowsiness, or coma. Muscular weakness* was very marked in a case under my own observation. *Headache* in the region involved, *vomiting, nystagmus, convulsions*, generally unilateral, and even hemiplegia, may be present; and, toward the close, *optic neuritis;* extensive disease may, on the other hand, exist without any symptoms whatever.

Diagnosis.—In the absence of distinctive symptoms the possibility of the presence of hæmatoma should be remembered where there are other signs of general paralysis or chronic alcoholism. If to such symptoms great muscular weakness is added further suspicion is justified.

Treatment.—With a prognosis absolutely unfavorable as to recovery, it remains only to treat symptoms as they arise. Indications of hemorrhage should be treated by elevation of the head, an ice-cap, and rest in bed.

LEPTOMENINGITIS.

Of leptomeningitis there may be an acute and chronic variety. In addition, other adjective terms are used to indicate its seat and the nature of its cause; such are basilar meningitis, meningitis of the convexity, tubercular meningitis, etc. Epidemic meningitis has received separate consideration.

Acute Leptomeningitis.

Definition.—An acute inflammation of the pia and arachnoid membranes, attended by exudation between the two membranes.

Etiology.—All ages are subject to meningitis, that of the convexity being more frequent in adults because they are more subject to traumatic agencies which cause it, while the basilar form is more common in children. It is rather more frequent in males, and there is a hereditary tendency to one form—tubercular meningitis.

Of the direct causes:—

(1) An eruption of miliary tubercles is the most frequent. This cause may operate at all ages, but is most active in children. In adults it generally starts from a recognized tuberculosis elsewhere; in children the process is almost always part of a general tuberculosis. Tubercular meningitis takes place generally at the base of the brain, constituting the chief form of basilar meningitis.

(2) Adjacent disease, which may be outside of the dura mater, such as caries, especially in the petrous portion of the temporal bone. Even disease outside the skull, like erysipelas or suppurative disease of the scalp, may be a primary focus. In these cases it is usually unilateral, and may be accompanied by thrombosis of the sinuses and abscess. Or the disease may result in abscess within the brain.

(3) The bacterium or toxin of the acute infectious diseases—pneumonia, ulcerative endocarditis, measles, scarlet fever, small-pox, typhoid fever, acute rheumatism, and septicæmia. Care must, however, be taken not to confound the simple intense delirium in some of these affections with meningitis, remembering, too, that the latter complication is, under any circumstances, a rare one. The toxin of pneumonia is the most common, and perhaps after this, that of small-pox. The inflammation thus caused is chiefly of the convexity, except in septicæmia, when it is general.

(4) Chronic Bright's disease, and other cachectic conditions. In these the inflammation is commonly basilar.

(5) Sunstroke—a doubtful cause.

(6) Mental excitement and brain work—also doubtful.

(7) Rarely in acute inflammation, syphilis, whose product is also basal.

(8) Finally, unknown causes may produce meningitis of the convexity or of the base. Possibly, as Gowers suggests, organisms otherwise powerless may become sufficient causes during ill health. Thus may be caused some undoubted though rare cases of non-tubercular basilar meningitis of children—*leptomeningitis infantum.*

Morbid Anatomy.—The early results of leptomeningitis are the same in all varieties. They consist, first, in a hyperæmia of the capillaries, producing a diffuse pinkish tinge. The next visible change is a turbidity and opacity of the arachnoid, which extends to the pia, where it is especially distinct along the blood-vessels, consisting, in fact, in an infiltration with leucocytes of the lymph spaces and lymphatic sheaths. As the cellular accumulation increases, the exudate beneath the arachnoid assumes a yellowish-white, creamy appearance. The subarachnoid fluid increases, constituting *hydrocephalus externus*. In suppurative cases it becomes pus, which forms a greenish-yellow layer at the convexity or base, or both.

In tubercular meningitis, which is chiefly basilar, the eruption of tubercles precedes the inflammation. There may even be tuberculosis of the pia without inflammation. In this form the inflammation is never actually purulent, though the lymph has often the appearance of pus. The tubercles are most abundant about the optic chiasm, between the pons, and the beginning of the fissures of Sylvius.

Ventricular effusion is present in the majority of instances, about four out of five, constituting *hydrocephalus internus*, generally associated with closure of the opening of the 4th ventricle. The effusion is usually limited to a few ounces, but it may be large in amount, distending the ventricles and compressing the cortex. The lining membrane of the ventricles and the choroid plexuses may be inflamed, and the ventricular effusion may be the result of such inflammation.

In all varieties of meningitis, and especially the tubercular, the superficial layer of the cortex is also involved, being at least hyperæmic, and sometimes softened, it may be also the seat of punctiform hemorrhages, constituting *red softening*. This is especially prone to occur in tubercular meningitis, because of the extension of the tuberculosis along the blood-vessels which dip into the cortex. In pulling off the pia these blood-vessels are dragged with it, leaving a ragged appearance of the cortex.

The leptomeningitis infantum presents an appearance very similar to that of tubercular meningitis. It involves chiefly the posterior part of the meninges and cerebellum, closing sometimes the foramen of Magendie, whence the term *occlusive meningitis*. It may also cause an acute, sometimes purulent, hydrocephalus.

Symptoms.—These are very varied and not always distinctive of the different forms. First, it is important to remember that all except those which are peculiar to inflammation of the base may be present in any of the serious infectious fevers without meningitis, especially pneumonia and small-pox. When secondary to these affections they are accompanied by the symptoms of the disease to which they succeed.

Meningitis is usually ushered in by *premonitory symptoms*, which, again, are not distinctive, being those usual to acute disease. Perhaps *irritability* is more constant than in other acute diseases. In case of children *vomiting* with a slight cause, or without discoverable cause, is a symptom of more suspicious nature. Generally there is *high fever*, *coated tongue*, and *constipation*, although fever is not invariable. The usual temperature is 103° to 104° F. (39.5° to 40° C.), but it may reach 105° to 106° F. (40.5° to 41.1° C.), and toward the close of fatal cases,

108° F. (42.2° C.). It is especially apt to be mild or absent in the meningitis of Bright's disease, or of debilitated children. The *pulse* is increased in frequency at first, but later may be slow and irregular.

Of the symptoms the direct result of the disease, *pain in the head* is the most constant. Commonly frontal, it may be general. Its constancy and severity are characteristic. Yet it is subject to such exacerbations as may cause the patient to cry out, constituting the *hydrocephalic* cry of children. The headache is invariable, followed sooner or later by *unconsciousness*. *Delirium* is an early symptom and soon follows the headache; at first wandering, it soon becomes active, and may alternate with drowsiness or stupor.

Vomiting is a very frequent symptom, especially in basilar meningitis, of which it is more or less characteristic. It has this peculiarity, that it is not usually accompanied by nausea and retching. General *convulsions* are also another symptom, occurring in all forms and all ages, but more frequently in the tubercular meningitis of children. When the inflammation is at the base *rigidity of the neck* with retraction of the head is very marked, especially when the inflammation extends down the membranes of the spinal cord. *Optic neuritis* is another symptom, usually late in occurrence—at the end of the first week—and due to involvement of the sheath of the optic nerve within the skull. *Strabismus* is also common. There may be weakness of the eye muscles and slight *ptosis*. The *pupils* are usually *contracted* in the early disease from intolerance of light; later, they are *dilated*. *Inequality* of the pupil is even a more characteristic symptom, though transient and variable. It occurs in connection with inflammation of the convexity as well as of the base.

The *facial nerve* may be involved in basilar cases, producing slight paralysis, as may also be the *5th nerve*, producing anæsthesia and trophic changes in the cornea if the Gasserian ganglion is involved. On the other hand, hyperæsthesic skin is often present; also hyperæsthesia of the special senses, especially hearing and sight.

Symptoms in the limbs may present themselves, such as *muscular rigidity*, *unilateral convulsions*, and even *hemiplegia*, but the latter is rare. When they occur, they are late symptoms.

Diagnosis.—The diagnosis is not always easy because so many symptoms may be simulated by simple congestion due to the poison of the infectious diseases. The basilar symptoms are the most distinctive, and it is a real help to know that a possible cause is present, either predisposing or exciting, such, for example, as a tubercular taint or tubercular disease or middle-ear disease. Retraction of the head, so marked in this form, may result from rheumatism of the muscles of the back of the neck. Sir William Jenner pointed out a difference between the relation of headache and delirium in general disease and meningitis: In general disease the headache ceases when the delirium begins; in meningitis the headache continues and co-exists with the disease. Convulsions, too, when present, occur at the beginning of a general disease, as witness scarlet fever particularly, while they occur late in meningitis. Optic neuritis and other eye symptoms are confined to meningitis.

A rapidly growing intra-cranial tumor often gives rise to difficulty

in the diagnosis between it and meningitis. In tumors which may be tubercular or gliomatous, limb symptoms,—such as weakness,—hemiplegia, and convulsions are manifested only after the tumor once begins to interfere with function, which it may not do at first; the loss of power, moreover, comes on gradually, while in meningitis all these symptoms are rapidly developed. Higher degrees of optic neuritis, as observed by the ophthalmoscope, are found in connection with tumor rather than with meningitis. The duration of the disease will settle the question ultimately, as meningitis is of short duration—from two to three days to as many weeks—while tumors last for months.

Meningitis, especially tubercular, is sometimes mistaken for hysteria, but the almost invariable presence of fever in meningitis and its total absence in hysteria should prevent error. In children the symptoms even of tubercular meningitis are sometimes closely simulated in bad cachectic states, when there is no meningitis whatever. What is regarded as meningitis after sunstroke is a prolonged state of mental hebetude with symptoms usually aggravated on slight exposure to the sun.

Prognosis.—The prognosis in leptomeningitis is unfavorable, although not necessarily hopeless. In meningitis of the convexity recovery is possible; in undoubted tubercular meningitis it is not. But I have so many times known a diagnosis of tubercular meningitis with corresponding prognosis followed by complete recovery in children, that I have grown very cautious in prognosis, knowing, of course, full well that an error of diagnosis was the condition of recovery. Especially in general tuberculosis should we avoid too unfavorable a prognosis because mistakes here are quite frequent. In meningitis from adjacent bone disease much depends on the accessibility of the bone lesion, but as this is generally difficult of access the prognosis is correspondingly serious. This is especially the case in ear disease. In syphilitic meningitis, if the diagnosis is made early, chances of recovery or improvement are better.

Treatment.—The treatment of adjacent disease which may cause the meningitis is of the first importance. Surgical interference should be promptly resorted to in middle-ear disease. In the absence of such disease the treatment is mainly symptomatic. Extreme quiet and the avoidance of all causes of excitement are paramount. It is the one disease, outside of ophthalmia, in which the darkening of the room may be justified. The head should be raised. Leeching is a most valuable means of affording cure, if possible, and temporary relief when cure is impossible. Leeches should be applied to the back of the ear and to the temple. Ice should be kept applied to the head. Counter-irritation, by blisters to the back of the neck, is also very useful, and not as painful or annoying as its appearance suggests. It has even been applied to the whole scalp after shaving the head, but I have never felt justified in doing this, especially where the diagnosis of tubercular disease is pretty clear. The bowels should be kept free.

The diet should be liquid—milk and animal broths of a light kind are the best food. Such drugs as meet the symptoms should be given. Phenacetin to relieve pain in the head, if the ice and abstraction of blood

do not do it. The temperature is kept down by sponging and even cool bathing. Mercury is still an acknowledged drug in meningitis not tubercular; and as chances of error of diagnosis always exist it may be employed in any case. It should be administered to the production of slight salivation, preferably by inunction because the effect is more rapidly produced. The mercurial ointment should be used.

Chronic Leptomeningitis.

Etiology and Morbid Anatomy.—This comparatively rare affection affects chiefly the convexity of the brain, and is the result of alcoholism, syphilis, or tuberculosis.

In milder degrees, seen in alcoholics, the pia-arachnoid is opaque, as seen over the sulci, the opacity and thickening being more marked along the borders of the blood-vessels. In syphilis there are often foci or thickened patches, thickest in the centre and receding toward the edges. These may reach dimensions to justify the term gummy outgrowth or tumor. The blood-vessels are the seat of endarteritis. In the tubercular forms, in children, the base of the brain is affected, as in acute tubercular meningitis. Internal hydrocephalus may be a consequence where there is obstruction to the orifice of the 4th ventricle.

Symptoms.—These are those of the acute form in a milder and more prolonged manner—headache, vomiting, mental symptoms, sometimes convulsions, rigidity, retraction of the head, optic neuritis, more rarely strabismus, and nystagmus. They may last from a month to a year or more. Fever is more frequently absent in chronic meningitis, but careful observation will generally find some elevation of temperature.

Diagnosis.—It is, in fact, the chronic variety of leptomeningitis which is separated with the greatest difficulty from tumor. Loss of motor power is more characteristic of tumor. Optic neuritis is also a more decided symptom in tumor, and goes on increasing, while it seldom reaches an advanced stage in chronic meningitis. Other eye symptoms —strabismus, irregularity of pupil—are more distinctive of meningitis. Strabismus occurs in hysteria, but it is always convergent and there is total absence of fever as shown by the absence of elevation of temperature.

Prognosis.—This is not so unfavorable as in the acute variety. The syphilitic form is quite amenable to treatment, the alcoholic less so; the tubercular must sooner or later be fatal. Caution in prognosis is demanded by occasional error in diagnosis.

Treatment.—The cause must be carefully sought. If syphilitic, iodides and mercurials must be used, as for this disease. In alcoholism and tuberculosis the symptoms must be treated by measures already indicated.

AFFECTIONS OF THE BLOOD-VESSELS OF THE BRAIN.

HYPERÆMIA.

SYNONYM.—*Cerebral Hyperæmia, or Congestion of the Brain.*

Definition.—A condition of the brain in which the blood-vessels are surcharged with blood. The congestion is *active* as the result of increased flow of blood to the brain, as in alcoholic hyperæmia; *passive* where there is obstruction to its outward movement, as in constriction of the vessels in the neck.

While, from the standpoint of morbid anatomy, our ideas may be very definite as to what should constitute active and passive hyperæmia, it cannot be said that a definite set of symptoms is associated with either in the case of the brain. In the first place, the amount of blood in the brain varies greatly within the limits of health, and while it might be said that physiological hyperæmia ends where abnormal mental phenomena present themselves, it is undoubtedly true also that an over-fulness of the vessels of the brain may exist for some time without the symptomatic expression which finally, however, appears. With the appearance of such symptoms we commonly date the clinical beginning of the pathological state known as chronic hyperæmia.

Morbid Anatomy.—The difficulties are increased by the fact that in acute active and passive hyperæmia, at least, no post-mortem evidences of it remain, the congestion having disappeared with death, although an unusual distinctness of the puncta vasculosa has long been regarded as post-mortem evidence. The difficulty of recognizing such condition makes this sign an unreliable one. In chronic hyperæmia there results, sometimes at least, a turbidity and even opacity of the pia mater, with slight thickening, together with elongation and tortuosity of the vessels, which is regarded as characteristic.

Etiology.—The causes of *active* hyperæmia are prolonged mental activity, excitement, and over-work, preëminently alcohol and the causes of the acute fevers; the hypertrophy and over-action of the heart which attends aortic regurgitation may be a cause. The causes of *passive* congestion are mainly mechanical, including mitral valvular heart disease, emphysema, straining or other cause obstructing the return of blood from the brain,—such tumors pressing on the vessels of the neck,—or tight clothing.

Symptoms.—These are not very distinctive. The symptoms of active hyperæmia, so far as recognizable, are a sense of fulness or pressure, headache, mental excitement, irritability, confusion of ideas, insomnia, vertigo, ringing in the ears, and in extreme cases hallucinations, delirium, and mania. These symptoms are increased when the head is downward or there is straining. The phenomena of so-called "rush of blood to the head," are probably the result of active hyperæmia. They include a suffusion of the skin of the face and head and feeling of warmth in these situations, strong beating of the carotids, headache, tinnitus aurium, spots before the eyes, vertigo, and sometimes actual falling.

It is not easy to separate the phenomena of passive hyperæmia from those of active congestion. They are, however, less pronounced and slower in their development.

Treatment.—The indications for treatment are, however, plain. The head is to be kept raised. Purgation is the first measure to be thought of. The saline and hydragogue cathartics are especially indicated because of their depleting effect. The ice-cap should be used. In extreme cases even blood-letting may be necessary, the efficiency of which is sometimes seen in the relief afforded by a bleeding of the nose. Leeches applied behind the ears often afford magical relief to the symptoms commonly ascribed to congestion of the brain. Wet cups may be put to the back of the neck for the same purpose. The diet should be spare and easily assimilable, in acute cases liquid only.

Of medicines, the bromide of potassium theoretically fulfils the indications, and in full doses of 15 to 30 grains (one to two gm.) every three hours to adults, is often useful though it should not be allowed to substitute the other measures mentioned. Phenacetin is an admirable remedy for the headache, a single dose of ten grains (0.66 gm.) being often sufficient. It may be repeated if necessary, or smaller doses may be given more frequently.

ANÆMIA OF THE BRAIN.

Definition.—The more usual application of the term anæmia of the brain is to conditions in which the quantity of blood in the organ is diminished, though depraved states of the vital fluid without loss of bulk may also produce the same symptoms.

Morbid Anatomy.—This is more distinctive than in hyperæmia. The membranes are pale, the blood in their vessels, except the larger ones, is scanty, and over the convolutions the vessels are quite empty. The grey and white matter are both pale on section, and the puncta vasculosa are less distinct and less numerous. The cerebro-spinal fluid is increased.

Etiology.—The causes leading to this condition are for the most part those which withdraw blood from the brain, but they include also such as prevent its access. Among the former are hemorrhages, profuse and rapid ; bowel fluxes, such as those of cholera in adults and cholera infantum in children ; and the opening of vascular areas by the removal of pressure caused by large tumors or ascitic fluid. Thus is explained the fainting which sometimes succeeds the removal of a large abdominal dropsy. In the second set of causes are feeble action of the heart, ligature of the carotid artery, or other obstruction in vessels carrying blood to the brain. Such obstructions are thrombi and emboli. The brain substance adjacent to the dilated ventricles in hydrocephalus interna is anæmic from compression. The fainting due to sudden emotion, such as fright, is ascribed to a withdrawal of blood from the brain.

Symptoms.—Some of these are definite and the direct result of loss of blood to the brain. Such are the dizziness, confusion of ideas, flashings of light, roaring in the ears, nausea, and ultimate loss of consciousness

and even death which succeed hemorrhages or emotion. In other cases the skin is cold and clammy, and a cold perspiration starts to the surface. Other symptoms are less distinctive. They are ascribed to chronic anæmia, but may result also from other causes. Such are mental apathy, disinclination to work, a sleepy feeling during the day, and insomnia at night. Headache, tinnitus, vertigo, hallucinations, and delirium are also consequences more particularly of lowered composition of the blood, the result of long illnesses, like pulmonary consumption and Bright's disease. The convulsions characteristic of the latter disease have been ascribed to anæmia and also to œdema of the brain.

The hydrocephaloid symptoms, described by Marshall Hall as the direct result of prolonged diarrhœa and of cholera infantum in children, are regarded as results of anæmia. They include semi-stupor with eyes unclosed, later dilated pupils, strabismus, convulsions, rigidity, and death.

Treatment.—The immediate consequences of the acute form of anæmia are diminished or averted by placing the patient on the flat of the back with the head low; by diffusible stimulants, of which alcohol and ammonia are the types; also cardiac stimulants, and nourishing and easily assimilable foods. The chronic forms of brain anæmia are treated by nutritious, easily assimilable foods, and tonics, especially iron and arsenic. In the hydrocephaloid condition in infants alcohol is the pre-eminent remedy, associated with warm baths and general restorative measures.

ŒDEMA OF THE BRAIN.

Definition.—The term includes two conditions, the most definite and easily recognizable of which is an abnormal accumulation of cerebro-spinal fluid between the arachnoid and the pia mater, and in the meshes of the pia. In the second condition there is added to the first an abnormal moistness of the substance of the brain.

Etiology.—The most common cause is mitral stenosis, though any cause obstructing the return of blood from the brain as well as recurring irritative hyperæmias, such as are produced by alcoholism and the psychoses, are also causes. Bright's disease is also a cause of œdema of the brain, local or general.

Local œdemas of the brain are also caused by obstruction of single sinuses of the dura mater, or compression by tubercular or other tumors of the veins of the velum interpositum, known as the *vena Galeni.*

Morbid Anatomy.—The membranes are turbid, their vessels are distended and serpentine in their course, and the subarachnoid space is filled with clear fluid. The substance of the brain is anæmic, moist, and glistening. In extreme cases there is compression of the cortex, with resulting flattening of the convolutions and widening of the sulci. The fluid in the lateral ventricles may also be increased.

Symptoms.—These are illy defined. There may be hallucinations and even mania, very similar, in fact, to those of anæmia. Traube and Rosenstein ascribed the convulsions of Bright's disease to œdema of the brain, while certain unilateral convulsions and paralysis in connection

with this disease have been assigned to the same cause. Even death has been ascribed to sudden serous effusions of this kind constituting acute œdema of the pia mater, or *apoplexia serosa.*

Treatment.—The treatment is that of the conditions to which the symptoms are secondary. The effects of cardiac stenosis must be overcome by cardiac stimulants; Bright's disease must receive appropriate treatment. Thrombosis of the sinuses admits of no treatment, though its effects may diminish by gradual contraction and possible liquefaction and removal of the thrombus. The psychoses should receive treatment appropriate to them.

APOPLEXY.

Definition.—The term apoplexy is applied to a sudden and prolonged loss of consciousness and motor power not due to heart failure. Its most frequent cause is cerebral hemorrhage, but sudden arrest of blood supply to a part of the brain by thrombosis, embolism, or ligature will produce the same effect. Laceration of the brain without hemorrhage is also a cause.

Unconsciousness may also be produced by simple congestion, and it was formerly thought that a simple serous transudate could produce similar symptoms in a milder form and of shorter duration; whence the term "serous apoplexy." It is doubted at the present day whether such a condition is sufficient. Concussion of the brain, however, causes similar symptoms.

I. Cerebral Hemorrhage.

Distribution.—It is a rare event to find a rupture in any of the large arteries of the circle of Willis, although one of the commonest things to be seen at an autopsy are the white patches of atheroma upon them. But the free anastomosis of this circle scarcely allows of increase of intravascular pressure sufficient to cause rupture. Further, it is the "central" rather than the "cortical" branches of this circle which rupture, and especially the central branches of the *middle cerebral* which, entering the brain at the anterior perforated space, pass to the corpus striatum and internal capsule. One of these is the so-called artery of "cerebral hemorrhage," thus named by Charcot because of the frequency of its involvement. It passes to the internal capsule and lenticular nucleus, where the majority of the massive hemorrhages of the brain occur.

Etiology.—Disease of the artery is at the bottom of the vast majority of cerebral hemorrhages. Indeed, except in the case of traumatic hemorrhages due to fracture of the skull, it is very doubtful whether hemorrhage ever occurs without such disease. The simplest form is the fatty degeneration and "erosion" of the intima, characteristic of advanced age. Endarteritis, however produced, is, perhaps, the most frequent cause. Its ultimate result, as shown by Charcot and Bouchard, as far back as 1868, is the miliary aneurism, which, in almost every instance, precedes the rupture. It is a spindle-shaped, rarely lateral, dilatation, $\frac{1}{25}$ to $\frac{1}{5}$ inch (one to five mm.) in diameter. The inflammatory

process preceding it consists in a proliferation and degeneration of the intima cells, followed by atrophy, which extends also to the muscular layer and the scanty adventitia. These, yielding to the intravascular pressure at the weak points, dilate to form the little aneurism, which is later ruptured by some further increment of pressure.

The "fatty erosion" of the intima which is the next most frequent cause of vulnerability is favored by age, by chronic interstitial nephritis, and the over-strain of the vessels due to the hypertrophy of the left ventricle so often associated with that disease as well as with valvular heart-disease.

While by far the larger majority of hemorrhages are preceded by miliary aneurism or fatty erosion—fully nine out of ten—there still remain a number of instances in which careful search fails to find anything but diffuse degeneration; whence these conditions cannot be regarded as indispensable. The infectious fevers, leucæmia, and anæmia are also causes of hemorrhage, which is independent of miliary aneurism.

Age is also a predisposing factor, most ruptures occurring after fifty though apoplexy has occurred under ten; while the occupations and dissipations of men furnish an additional predisposing element, which accounts for its greater frequency in the male sex. Other predisposing causes are those usually responsible for endarteritis, viz., gout, alcohol, syphilis, Bright's disease, the apoplectic habit as seen in the stout, short-necked, full-blooded individual; and, finally, heredity, which is, strictly speaking, a hereditary tendency to the favoring diseases. Embolism is also a cause of endarteritis which may result in aneurism.

The exciting causes are such as temporarily increase intra-vascular pressure, as violent exertion, straining, debauch in eating and drinking, and mental emotion.

Morbid Anatomy.—The large central ganglia in the neighborhood of the lateral ventricles, *i. e.*, the optic thalami, the caudate and lenticular nuclei, and the adjacent white matter of the internal capsule and centrum ovale are the favorite seats of the miliary aneurism and consequent hemorrhage. These aneurisms are found also, but much more rarely, in the smaller branches of the cortical vessels, in the pons, cerbellum, crura cerebri, or medulla. On section of the large ganglia they may be seen as small dark points, as large as a pin's head, and are often very distinct in arteries drawn out of the substance of the brain, especially the anterior perforated space. Coarser aneurisms are also found on the branches of the circle of Willis.

In accordance with the distribution of vessels hemorrhage is said to be meningeal or central. Meningeal hemorrhage may be outside of the dura mater, between it and the bone, or between the dura and the arachnoid, or between the arachnoid and the pia. The extra-dural and sub-dural meningeal hemorrhages are both traumatic, one variety of which is produced during birth, but the subarachnoid are due to the causes already considered. Central hemorrhages may also burst into the membranes as well as into the ventricles of the brain. Meningeal hemorrhage may occur in the infectious fevers, leucæmia, and anæmia.

Given a massive hemorrhage, what is its effect on the brain substance,

and what are the changes in the extravasated blood? The former varies somewhat with its situation. If extra-dural the dura mater is torn away from the bone to a varying extent. If subdural or subarachnoid, it separates these membranes from the brain substance, but in either event the convolutions are more or less flattened and the sulci more or less obliterated.

As already stated, central hemorrhage most frequently occurs in the neighborhood of the corpus striatum, through which, if large, the blood finds its way toward the outer section of the lenticular nucleus, pushing inward the optic thalamus and bursting into the lateral ventricle or into the white matter of the centrum ovale. The pressure exerted is often such as to flatten the convolutions, empty the parietal veins, and press the falx aside, sometimes even to produce a sense of fluctuation over the membranes. Hemorrhages rarely begin in the central white matter, but they may occur in the crura or pons, or 4th ventricle and also into the cerebellum, not infrequently from the superior cerebellar artery. Osler alludes to two cases of death in women of twenty-five from cerebellar hemorrhage. Very rarely hemorrhages into the ventricle may start in the choroid plexus or the ventricular walls. Large quantities of blood may be poured out at the base of the brain, and it may flow down into the cord from a rupture of any of the arteries going to or from the circle of Willis.

If the patient survives, changes take place in the extravasated blood, which promptly coagulates into a dark-red mass. This almost immediately begins to contract, permitting often the return of a certain degree of function by removing pressure. As time elapses, the dark-red mass passes into a chocolate-brown pulp, composed of liquefying clot and disintegrated nervous matter. The microscope, at this stage, recognizes numerous hæmatoidin crystals, and fatty granular cells, which are probably fatty by imbibition of fat drops. The adjacent nervous tissue is stained yellow by the imbibed hæmatoidin. The clot itself becomes encapsulated by fibrin, and gradually absorbed, being often substituted by a semi-transparent, or completely transparent, fluid, forming the apoplectic cyst. If smaller, the walls approach and unite, leaving only a linear pigmented scar. Especially is this the case with small clots on the surface of the convolutions, which may leave only a staining of the membranes. In other cases of abundant cortical effusion, especially in infants, there may be circumscribed wasting of the convolutions and a cyst of the meninges. The position and extent of the permanent lesion determine the presence of secondary descending degeneration. If the motor-cortex or a motor-patch is involved there may be found, in persons dying some years after an apoplexy with hemiplegia, degeneration in the fibres of the anterior part of the pons, in the pyramidal fibres of the medulla, in the direct pyramidal fibres of the cord of the same side, and in the crossed pyramidal fibres of the opposite side.

Symptoms.—*Premonitory* signs are occasionally present. There may be a feeling of fulness in the head, headache, tinnitus, vertigo, or numbness, tingling, pains in the limbs on one side, loss of memory of words, or choreiform movements—prehemiplegic chorea—possibly due to miliary aneurism or otherwise diseased vessels.

With the bursting of a vessel of sufficient size there occurs the *apoplectic "stroke,"* or apoplectic shock. Its most striking feature is *sudden loss of consciousness.* If complete the patient falls heavily to the ground, and there may be slight convulsive movement, but it soon ceases. More rarely a true convulsion ushers in the attack. The patient cannot be aroused, the face is suffused, cyanotic—sometimes, however, pale; the breathing is slow, noisy, stertorous, often attended with a puffing sound during expiration, corresponding with a blowing out of the relaxed cheeks on the paralyzed side; it may also be of the Cheyne-Stokes type. In contrast with the above, the development of unconsciousness is sometimes much more gradual, requiring several hours or a day, corresponding to which it is presumed that the hemorrhage is slow, constituting the "ingravescent form."

The second major symptom of apoplexy is *motor paralysis,* of which hemiplegia is the most conspicuous form. In most cases the motor pyramidal tract, as it crosses the internal capsule, is either directly destroyed or indirectly affected. Hence, most patients who survive the primary shock present a hemiplegia—paralysis of half the body opposite that of the hemorrhage. It is most noticeable in the arms and legs. These are thoroughly relaxed, falling limp when allowed to drop, as the limb of one thoroughly etherized. More rarely there is early *rigidity,* especially on the side opposite that of the hemorrhage. This symptom appears to be more frequently associated with hemorrhage into a lateral ventricle. *Reflex action* is either totally suspended or only brought out in response to a deep pin thrust or severe pinching.

The signs of hemiplegia are not always easily elicited at first, because a certain degree of consciousness is necessary to stimulate attempt at motion, but it may be that the angle of the mouth hangs down lower on one side—the paralyzed side—while the puffing of the cheek alluded to may be present on the same side, or the limbs of one side may be appreciably more flaccid than those of the other, or a small amount of reflex response may be elicited on the sound side. The *pulse* is usually slow, full and strong, tense. The *temperature* may be subnormal at first, rising to normal and even above, and in basal hemorrhage may be higher. In a rapidly fatal case it remains subnormal to the end. The *pupils* are *irregular, i. e.,* sometimes contracted, at others dilated, unequal. They respond to light either slowly or not at all.

Quite often one of the early symptoms in hemiplegia is *conjugate deviation* from the paralyzed side and toward the side of lesion; that is, in right hemiplegia the head and eyes look toward the left side. This symptom usually passes away, but sometimes continues for weeks, and, Gowers suggests, is perhaps occasionally represented by nystagmus or movement in the direction concerned. Should, however, convulsion, or spasm, or early rigidity develop, the head and eyes are rotated toward the paralyzed side, *i. e.,* away from the side of lesion. This is true only of cortical lesions, especially so in the neighborhood of the supramarginal and angular gyri.

In lesions of the internal capsule or the pons, on the other hand, where the conjugate deviation may also occur, the phenomena are

reversed,—the patient looks away from the lesion, in the absence of spasm,—but if the convulsion or spasm or rigidity occurs, the eyes and head look toward the lesion. These facts are a little confusing at first and may be expressed in the following :—

In lesion of cortex.

Without spasm, conjugate deviation is *toward* lesion.

With spasm or convulsions or early rigidity, *from* lesion.

In lesion of internal capsule or pons.

Without spasm, etc., *from* lesion.

With spasm, etc., *toward* lesion.

This is owing to the fact that these movements in health are innervated from both sides, and when a lesion occurs on one side of the brain, the innervation is given over to the other side until the injured one resumes its function, or until irritation in it causes it to assert or exceed its function.

The *fæces* and *urine* are passed involuntarily, and the latter is sometimes slightly albuminous.

In a few cases there is no reaction from the above-described condition. The symptoms all deepen, the breathing becomes rapid and rattling, the skin cool, the pulse weak and rapid, and the patient dies. In most cases, however, there is a certain abatement of the symptoms, even if the patient does not recover more fully. Consciousness returns partially or completely, the patient can be aroused by a loud voice, and one can recognize which side is paralyzed. There may, at this time, be a febrile movement, due to cerebral inflammation, during which the patient may die, or there may be another hemorrhage which carries him off.

On the other hand, improvement may continue to a further degree. The consciousness and intelligence may return completely, and the signs of paralysis gradually grow less, more rapidly in the legs than arms. They, however, almost never disappear completely, the patient continuing lame and requiring the use of a cane for the rest of his life. A remnant of face paralysis can almost always be recognized, while articulate speech may also continue defective.

Such marked improvement is, for the most part, reserved for the milder attacks, in which there is great variety as to degree. In such, the loss of consciousness is of short duration or it may not occur at all. Such attacks are not infrequently ushered in by nausea, vomiting, vertigo, or sudden headache. The paralytic symptoms may still be marked, and permit a study rather more satisfactory than the fulminating cases. In such study it will be found that all muscles are by no means equally paralyzed. Thus it will be seen that the lower division of the facial nerve, which supplies the muscles of the cheek, nose, and mouth, is plainly paralyzed ; while the upper division, distributed to the muscles of the eyes and forehead, is almost, if not entirely, intact. The forehead may be wrinkled with equal ease on the two sides, but an attempt to draw up the nose, or purse the mouth, fails, while one labio-nasal fold may be obliterated, and one angle of the mouth lower than the other. The natural wrinkles of the forehead are commonly less distinct on the paralyzed side than on the other. These events may be explained by the

fact that while both sides of the face receive fibres from each cerebral hemisphere this is especially true of the muscles of the upper part of the face which are always exercised bilaterally.

The *tongue* may not be paralyzed, but when it is, if protruded, it goes toward the paralyzed side, being pushed out by the genio-hyoglossal muscle of the other side, the innervation being by the hypoglossal nerve. Occasionally paralysis of the tongue contributes to difficulty in articulation.

Of the trunk muscles the trapezius is almost solely involved, and that but slightly, permitting the shoulder to drop a little, and very rarely there is somewhat diminished excursion of the side in deep breathing, owing to paralysis of the respiratory muscles.

Sensation is but slightly impaired in most cases of hemiplegia due to cerebral hemorrhage, and such impairment grows rapidly less as time elapses. It would be hemianæsthesia, and on the side opposite that of the lesion. There may also be trifling paræsthesia at first. Any marked disturbance of sensation means that the posterior extremity of the internal capsule is involved. There is sometimes temporary and even permanent hemianopia, which implies some lesion of the fibres of the optic nerve in the internal capsule or the posterior tubercle of the optic thalamus,—the pulvinar. The muscular sense is not usually affected.

The *tendon reflexes* are increased in nearly all cases on the paralyzed side, though at the very beginning of a severe shock they may be briefly abolished. In cases of any duration even the periosteal reflexes are increased, and to a less degree the reflexes of the sound side are increased. There is even, at times, ankle clonus. These events are explained by supposing a suspension of the inhibitory reflex cortical centres, due to the cerebral lesion. The *skin reflexes*, on the other hand, are diminished on the paralyzed side, remaining normal on the sound side.

The rapid improvement mentioned as occurring in some cases is usually confined to a few weeks or days, after which improvement goes on more slowly, the lower extremities recovering more completely than the upper. The gait resulting from partial recovery is peculiar. Short steps are taken by the affected leg, and the toe is dragged more or less, while sometimes locomotion is accomplished by sweeping the leg around in a semicircle by the iliacus and psoas and the vastus externus, while it is held stiff, as in a splint, by the quadriceps extensor. In the arm the shoulder muscles are the last to recover.

Later in the history of the case *contractures* may come on in the paralyzed muscles, shown especially in flexures of the fingers, contraction of the forearm in a position of pronation, and partial flexion, with the upper arm adducted. In the lower extremity the extensors or flexors may be affected, the calf muscles more frequently. This contraction is explained by some, and notably by Strümpell, as a "passive contraction," the position assumed being the natural one in a state of rest. On the other hand, Charcot and his pupils hold that the contractures are due to secondary degeneration of the pyramidal tract. There are also sometimes "associated movements" of the paralyzed muscles, to which Hitzig has called attention. In these, movements of the sound side excite

associated movements in the corresponding muscles of the other side, and attempts to move the affected side result in motion of corresponding muscles of the sound side. Sometimes, also, involuntary movements of the lower extremity occur when the patient attempts to move the corresponding arm. A post-hemiplegic chorea, first described by S. Weir Mitchell, should also be mentioned. It is seen not so much in the hemiplegia resulting from cerebral hemorrhage as from focal disease of the posterior end of the internal capsule and optic thalamus.

Trophic symptoms may appear late in the disease, seen at first in elevation of temperature, increase of color on the paralyzed side of the face, swelling of the eyelids, and contraction of the pupil; also swelling of the hands. It is to be remembered, however, that slight swelling may result from sluggish circulation of blood and lymph, contributed to by diminished muscular contraction and absence of use. In a more advanced stage the extremities become cooler and are often constantly moist. Among these vaso-motor events Charcot has placed what he calls *acute malignant decubitus*—a disposition to rapid gangrene of the tissues over the sacrum. It may appear in a few days after the shock, beginning with a circumscribed redness and formation of vesicles, succeeded by deep-reaching necrosis. While this is probably, as Charcot regards it, a vaso-motor phenomenon, that it is also invited by the usual causes of gangrene in dorsal decubitus, such as irritation by urine, fæces, and even inequalities in the bed-clothing, is also likely. An occasional arthritis, acute or chronic, Charcot also considers a neuropathic event.

General nutrition is well maintained, the patient even gaining in flesh at times. More rarely there is rapid wasting.

The mental condition of patients who recover partially from the effects of hemorrhage, is, for the most part, good, but it not infrequently happens that after awhile mental weakness manifests itself in loss of memory, defective intellection; while imbecility sometimes ultimately supervenes.

Diagnosis.—The greatest difficulty lies in the distinction between cerebral hemorrhage, embolism, and thrombosis. Sometimes, indeed, the distinction is impossible at once. In embolism the patient is younger, we look for valvular heart disease. An apoplectic seizure after parturition is apt to be embolic. In embolism, too, there is less disturbance of temperature, the paralysis is more apt to precede the coma and convulsions if the latter are present; the turgid face, the hard pulse, loud breathing, and greater general disturbance of a serious stroke of apoplexy from hemorrhage are wanting.

In thrombosis the difficulty in diagnosis may even be greater, especially from the "ingravescent" form of apoplexy, where the hemorrhage, and therefore the development of symptoms, is gradual, requiring sometimes a day or two. In thrombosis there are more frequently prodromata in the shape of slight seizures, quickly recovered from. Such events occurring in the aged, where there is evident atheroma of the blood-vessels and weak heart's action, are much more apt to precede thrombosis, where, too, there is absence of stertorous breathing, of variations in temperature, and pupillary disturbance. Lesions in the pons

and cerebellum are more apt to be hemorrhagic. According to Charles L. Dana, in any case between the ages of thirty and fifty, where there is no heart disease, the chances are six to one in favor of hemorrhage. Thrombosis and embolism with softening affect the vertebrals, basilar and posterior cerebrals, more frequently than hemorrhage.

In fulminating cases the coma is sometimes so profound that it is difficult, or impossible, to ascertain the presence of hemiplegia. The symptoms which aid in determining this have been mentioned on page 988. To these may be added the increase of reflexes on the affected side, conjugate deviation of the head and eyes, and rigidity of limbs on one side. It is these cases that are sometimes confounded with epilepsy, opium poisoning, severe alcoholism, or uræmia. In epilepsy there is the history of previous convulsions, and it is only where this has been overlooked that mistakes occur. In opium poisoning the coma is slow in its onset, the pupils are uniformly contracted, and the odor of laudanum is often on the breath. But here, too, the victim is often only discovered after coma has thoroughly developed. In alcoholism there is the odor of whiskey, but many an innocent fellow has been treated as a drunkard on whose brain lay a clot pressing him to death. The young ambulance or police surgeon is wise who defers his opinion. Sometimes alcoholism and apoplexy are combined, when a conservative course will be no less astute. The coma of uræmia in Bright's disease very strongly simulates that of apoplexy, especially in the rare cases of the latter in which there are convulsions. The presence of dropsy, or, in its absence, of the peculiar anæmia of Bright's disease should suggest this disease, while the finding of albuminuria and casts would settle the question in its favor. It is to be remembered, too, that uræmic convulsion may terminate in hemorrhage, while Bright's disease is also associated with a state of the arteries which disposes them to rupture. Coma in a puerperal woman associated with dropsy and albuminuria means uræmia.

Prognosis.—To have had a stroke of paralysis is justly regarded as having received a blow which marks the beginning of inevitable decline in health and usefulness, though cases are constantly occurring where a "slight stroke" is followed by complete recovery. Some of these are probably errors of diagnosis, yet not all. The cortical hemorrhages are those most frequently followed by recovery. After these come a large number of cases of first attack, from which the patient recovers a very considerable degree of health. Second attacks are prone to occur, which are more severe, and few survive a third attack.

The unfavorable cases are those in which the coma is profound and lasting. Such are hemorrhages into the ventricles and corona radiata, which are rapidly fatal. Meningeal hemorrhages are serious, but less so when traumatic than when due to disease of the vessels. Cases attended by early and persistent fever and delirium are unfavorable, as are also cases complicating renal disease and alcoholism. Hemorrhages into the corpus striatum and internal capsule produce persistent hemiplegia, followed by contracture. When cases survive the primary stroke and improvement sets in, this is much more rapid in the first few weeks than

later. In explanation of this it has been held that the symptoms thus rapidly removed are indirect focal symptoms, due to pressure of the clot on adjacent nervous tissue, while those more slow to yield are the result of destructive lesion.

Treatment.—The patient should be promptly placed in a horizontal position *with the head raised.* This is of the greatest importance, as it constantly happens that a patient in whom consciousness is returning immediately becomes comatose when the head is lowered. He should then be bled, unless the pulse be small and feeble. The bleeding should be accompanied by a laxative, which should be given alone if there be any reason why phlebotomy be not practised. In view of the unconscious state of the patient the best laxatives are croton oil and elaterium. Two drops of the former should be mixed in a little glycerine or oil and carried to the back part of the throat, or ¼ grain (0.0165 gm.) of elaterium dissolved in a small quantity of water may be given in the same way. The rectum should be at once cleaned out by an enema of warm water. An ice bag should be placed on the top of the head, hot water and mustard to the feet, while counter-irritation may also be applied to the back of the neck.

Compression of the carotid artery, formerly recommended and practised on empirical grounds, has recently received the endorsement of Horsley and Spencer, these experimenters having found that bleeding from the lenticulo-striate artery ceases when the carotid is compressed. It is especially in the ingravescent form that it has been recommended. F. X. Dercum and W. W. Keen * report two cases of ingravescent hemorrhage treated by ligation of the common carotid, of which one recovered.

If, after bleeding and purgation, the pulse continues bounding, the tincture of aconite or veratrum viride may be given in doses of a drop every half hour until the pulse is influenced. Iodide of potassium can hardly be expected to promote absorption of the clot, but may be given if syphilis is suspected.

The above treatment is for the period immediately succeeding hemorrhage. The remainder of treatment consists in measures to protect the patient against the effect of decubitus if this is prolonged, and in maintaining the nutrition of muscles, and protecting against contractures. The former is accomplished by attending to the secretions, preventing the irritation of the body by putrid urine and fæces or foreign substances like bread crumbs, by bathing and drying the body thoroughly, by frequent changes of posture. The latter will also guard against pneumonia, which is rather prone to occur on the paralyzed side. This disease may also be caused by the inspiration of particles of food, which is liable to happen if there is paralysis of the muscles of deglutition. The second indication is met by massage, faradization, and gymnastics, but they should be deferred for two or three weeks. Warm, salt baths, three or four times a week, are useful to the same end. Tonics in the shape of iron in small doses, quinine, and strychnine may be given, but alcohol in more than very moderate amounts is contra-indicated.

* Jour. of Nervous and Mental Diseases, September, 1894.

Operative treatment has been suggested to relieve the pressure of a clot in cerebral hemorrhage, and where it is certain that the clot is meningeal, especially after fracture, satisfactory results show that it is justified. Careful attention should be paid to the facts mentioned under topical diagnosis with a view to determining the seat of hemorrhage and the place to trephine. Deep hemorrhage is, however, beyond reach.

II. Embolism and Thrombosis of the Cerebral Vessels.

Synonyms.—*Cerebral Softening; Acute Softening.*

Definition.—By embolism is meant the plugging of an artery by a foreign body shot into the circulation from some point in the vascular system and carried in the blood-current to a point beyond which it cannot pass.

Etiology.—*Nature and Source of Embolism.*—The embolus is most frequently a vegetation from a diseased valve in the left ventricle. Less commonly it is a fragment of a clot in the same ventricle or in the auricular appendage or in an aneurism. Or it may be a calcareous particle from an atheromatous vessel or a piece of thrombus from the same. Even the territory of the pulmonary veins may contribute an embolus. Embolism is very much more frequent in chronic valvular disease than in primary acute endocarditis. It is prone to occur in recurring valvulitis, and especially mycotic endocarditis. Pregnancy with or without heart disease, the infectious fevers, and blood dyscrasiæ may be predisposing causes.

The embolus commonly enters the brain by the carotid, especially the left—which furnishes the most direct course—thence through the internal carotid to the left middle cerebral in the fissure of Sylvius; more rarely by the vertebral and its posterior cerebral branch.

Thrombosis.—In thrombosis there is also plugging of a living vessel, but by a clot formed in situ, which is either primary at the point plugged, or secondary about a previous embolus. Some favoring cause commonly exists. This is most frequently roughening due to endarteritis, with or without atheroma. Weak heart and blood dyscrasiæ are also predisposing causes. Ligation of the carotid artery is sometimes followed by thrombosis of cerebral vessels.

The vessels most frequently affected in thrombosis are the middle cerebral and the basilar, but the posterior cerebral and the vertebral and the branches of the circle of Willis may be plugged, and the basilar at its bifurcation.

The sinuses of the dura mater, especially the cavernous and transverse, may also be the seat of a thrombus, which is sometimes found post-mortem without symptoms during life. In the former vessel thrombosis is probably favored by the trabeculæ of connective tissue which span it and produce the slowing of current which is always a necessary condition of coagulation. Thrombosis of the cavernous sinus may be caused by caries of the petrous portion of the temporal bone. Such thrombosis may extend to the internal jugular vein, causing, in its turn, pulmonary embolism with the symptoms characteristic of it.

Relative Frequency of Thrombosis and Embolism.—Embolism has been thought to be more frequent in women, but of 79 cases lately collected by Newton Pitt, at Guy's Hospital, 44 were in men and 35 in women. Thrombosis is considered more common in men. Embolism is rare in children, being more frequent at from twenty to fifty ; thrombosis at from fifty to seventy.

Morbid Changes Due to Thrombosis and Embolism.—Degeneration and softening of the brain are the direct result of obstruction of its arteries, and occur sooner or later where the shutting off of the blood supply is sufficient. The process generally begins within twenty-four hours, and the minimum time required to complete it is one to two days. The local effects of embolism are much less distinctive in the brain than in the lungs or spleen. Thus there is almost never a distinct hemorrhagic infarct, though often a condition resembling it, the area cut off being infiltrated with blood. At other times the region is paler than in health and slightly softer. In either event the area becomes gradually infiltrated with serum and a more or less complete liquefaction results, presenting a reddish-yellow or white color, whence the terms *red* softening, *yellow* softening, or *white* softening. These variations are not the result of any essential difference in the nature of the process, as was formerly thought, but are rather accidental. In red softening the area happens to contain an unusual amount of extravasated blood due to punctiform hemorrhage or capillary apoplexy. This blood melts away and stains the softened mass. In yellow softening the proportion of fattily degenerated cells is larger, and it is found, therefore, chiefly in the cortex where cells prevail. In white softening there are few or no cellular elements, hence the white softening is found in the white or fibrous nervous matter. It is most characteristically seen about tumors and abscesses. As the grey matter of the cortex is also the most vascular part of the brain, it is here also that we find the red softening. Certain superficial yellow spots are found at times on the surface of the cortex in old persons known as *plaques jaunes.* They are sharply circumscribed, measure 0.4 to 1.6 inches (two to four centimeters), are made up of a yellow turbid material, sometimes crossed by trabeculæ, and represent fatty degeneration of peripheral cortical arteries.

Minutely examined, the softened areas consist of fatty granules and oil drops, myelin drops, fragments of swollen nerve fibres, fatty granular cells representing fatty neuroglia and nerve cells, or leucocytes and neuroglia cells, and perhaps endothelial cells which have *imbibed* the oil drops, it may be, from the disintegrated nervous matter. In the yellow softening these constitute the sum of altered materials. In red softening there are added in the early stages blood discs, later pigment granules or hæmatoidin crystals, or there is general staining by dissolved hæmoglobin. In the white softening the bits of fibres and myelin drops make up the chief bulk, as already stated. The minimum time required for these changes is one to two days. If within that period collateral compensatory circulation is set up, the destruction may not go so far and the nervous elements may resume their function. Or if this does not occur and the patient lives, the dead and disintegrated tissue may be gradually absorbed

and eventually be replaced by a cyst, while a minute focus of softening may be replaced by indurated cicatricial tissue. If the embolus is derived from an infective focus, as ulcerative endocarditis, an abscess may result.

Symptoms.—Neither thrombosis nor embolism of the cerebral arteries is always followed by recognized symptoms. All the large arteries of the base and the smaller arteries of the surface anastomose so freely that the effects of obstruction are promptly equalized. Nay, more, it is not usual to find at the necropsies of elderly persons yellow spots of fatty degeneration, the *plaques jaunes* referred to, scattered over the convolutions where nothing was suspected before death. Moreover, softening may take place in the "silent regions" without exciting suspicion. Very different is it with obstruction of the middle cerebral artery—the artery of the fissure of Sylvius. The clinical aspect differs, however, according as this vessel is plugged at its origin or a little further on in its course. The smaller arteries that alone enter the brain substance form two separate systems: the "cortical arteries" (Durst), passing to the cortex, and the "central" arteries, passing to the central ganglia. The central arteries are the first given off by the cerebral branches of the circle of Willis and are terminal arteries, unprovided with anastomoses. The cortical arteries spring from a network of branches of the cerebral arteries in the pia mater, in which tolerably free communication exists between the tertiary branches of the same trunk, and even between the branches of different trunks. These two systems of cortex and centre are, however, altogether independent of each other, and no anastomosis takes place between them, the zone at which they meet within the cerebral substance being situated about an inch and a half below the cerebral convolutions. In the case of the cerebral artery, when it is obliterated beyond the point at which its "central" branches come off, the superficial parts of the brain alone suffer, and since its branches in the pia mater anastomose with those of the anterior and posterior cerebrals, there may be no softening at all and but a temporary loss of function. At other times softening does occur, the exact situation and extent of which vary with the arteries plugged. The blood supply of the two central, the three frontal, and the three parietal convolutions being more or less cut off there is motor paralysis of the opposite side of the body, and as the lesion is most frequent on the left side there is right-sided hemiplegia and aphasia; the same phenomena, in fact, as follow hemorrhage, and which may be permanent or transient. Or it may be still more limited. The embolus may lodge in the artery, passing to the 3d frontal convolution, or in that of the ascending frontal or ascending parietal. It may lodge in the branch passing to the supra-marginal or angular gyrus, or the lowest branch, which is distributed to the upper convolution of the temporo-sphenoidal lobe. If, on the other hand, the seat of the lesion is at the point where the Sylvian artery arises from the internal carotid the central ganglia are involved, and there is almost certain to be softening of the corpus striatum and thalamus, because the arteries have no anastomoses, while the cortex escapes entirely because its vessels are distinct.

Summary of the Effects of Plugging of the Cerebral Vessels:—

Internal Carotid.—There may be no symptoms or there may be

transient hemiplegia or permanent hemiplegia and coma ending in death in a week or ten days. In the first alternate the circulation is maintained by the communicating vessels of the circle of Willis, which ordinarily dilate rapidly. If these vessels are small or absent, permanent hemiplegia and death must result, as a small part of the hemisphere only receives blood by the posterior cerebral. Thrombosis is very apt to extend from an initial focus in this vessel to its branches and may extend to the ophthalmic artery.

Anterior Cerebral.—Because of the right-angled direction at which this vessel is given off from the internal carotid it is rarely obstructed by embolus unless the parent trunk is plugged before this branch is given off, and then the mischief is trifling or *nil*, since branches of the middle cerebral may supply the same territory.

Middle Cerebral—the most frequently plugged of all cerebral vessels.—The result is hemiplegia, permanent if the embolus lodges before the central arteries are given off, since softening of the internal capsule ensues; if beyond the point the hemiplegia involving the arm and face is more apt to be transient. If on the left side there is also aphasia, there may also be impairment of sensibility for a time, and sometimes, also, involvement of the special senses and ptosis of the opposite side. The symptoms vary somewhat according as one or more of the cortical branches are obstructed by a plug at the point of division of the vessel in the island of Reil. Occlusion of the 1st branch may produce softening of the 3d frontal convolution and aphasia if on the left side; of the 2d and 3d, softening of the ascending frontal or ascending parietal convolution and hemiplegia, partial where the softening is incomplete; of the 4th branch, softening about the posterior limb of the fissure of Sylvius, and if on the left side, sensory aphasia, defective perception of words, with corresponding impairment of speech.

Posterior Cerebral.—Plugging of this branch distributed to the occipital and temporo-sphenoidal lobes rarely causes softening, because of anastomoses with other branches to the same area. So far as ascertainable, the phenomena are mostly sensory, including hemianæsthesia from softening of the tegmentum of the crus or internal capsule, or hemianopia from softening of the cuneus, though the same symptoms may result from interruption of the optic tract when the cortex is intact. Complete but temporary loss of sight has resulted from plugging of one posterior cerebral.

Basilar Artery.—Total occlusion of this vessel may produce bilateral paralysis from involvement of both motor paths in the pons, with other symptoms of apoplexy of this center, viz., bulbar palsies, irregularity of heart and breathing, spasm, and rarely convulsions; the temperature may rise rapidly to 109°F. (42.6° C.), or thereabouts, after an initial fall.

Vertebral Artery.—The left is more frequently plugged, rarely alone, commonly along with the basilar. The nuclei of the medulla are affected, and we have symptoms of acute bulbar palsy.

Cerebellar Arteries.—Obstruction of the isolated cerebellar arteries is rare as compared with plugging of the parent trunk, in the basilar. Even then the area of cerebral softening is limited by reason of collateral anasto-

moses, and the symptoms are obscured by those due to damage to the pons and medulla. Incoördination of movement has been reported in one or two cases where the region supplied by the posterior cerebellar was cut off.

Diagnosis.—The diagnosis between embolism and thrombosis, on the one hand, and hemorrhage into the brain, on the other, has been considered in the section on hemorrhage; but it is important to be able to decide whether the obstruction is embolic or thrombotic. In the former the onset is sudden, without premonitory symptoms; in the latter it is gradual, and there are often premonitory symptoms. In the former there may be convulsive twitchings, but hemiplegia quickly follows, with or without temporary loss of consciousness. In the latter the patient has previously complained of headache, vertigo, or tingling in the fingers; then paralysis may begin in one hand or foot, and extend slowly, the hemiplegia often remaining partial. Speech may have been embarrassed for some days previous, and the memory defective. In thrombosis due to syphilis, especially, the hemiplegia may come on gradually without loss of consciousness. The same is true of the so-called senile softening, which is generally due to thrombosis after atheroma of the cerebral arteries. In a few cases the onset is more sudden, and may happen during sleep. The temperature usually has a slight initial fall, followed by rise, as in hemorrhage. In embolism aphasia is quite a characteristic symptom, as it seems to occur more frequently on the left side than the right.

In both embolism and thrombosis the hemiplegia tends to improve rapidly, unless the vessel obstructed be a large one or there be rupture of a collateral branch. It is true that acute softening may terminate fatally within twenty-four hours, but usually the patient survives the onset, and at the worst dies after several weeks, the phenomena of the chronic stage being almost identical with those of hemorrhage. Spastic symptoms are also likely to occur, and there is a tendency to the characteristic mobile spasm.

Prognosis.—A patient rarely dies of a first attack of cerebral embolism, unless a very large vessel is obstructed, such as the internal carotid or basilar, whose occlusion is fatal; next in seriousness after these is plugging of the middle cerebral and vertebral, while obstruction of the two vertebrals is always fatal. Every succeeding attack increases the danger. Embolism promises more favorably than thrombosis; and thrombosis due to syphilitic disease is more hopeful than senile softening. Sudden severity in thrombosis is serious, and deranged breathing is an unfavorable symptom. Convulsions may be a result of syphilitic thrombosis. Where the embolism is due to valvular heart disease, it is apt to recur; when due to other causes, not. Thrombosis is prone to occur, especially when due to atheroma.

Treatment.—Neither thrombosis nor embolism demand blood-letting, as does hemorrhage. Indeed, it is strongly contra-indicated. Rest in bed, with head raised, is important. If syphilis is the cause of thrombosis it should receive the usual treatment—the iodide of potassium in ascending doses until doses of a drachm or more are reached. There

is no treatment for atheroma. Attention should be paid to the heart, kidneys, and bowels. The heart is commonly feeble, and digitalis and strophanthus are needed to keep its action uniform and strong, by which one condition of thrombosis is removed. The secretion of urine is scanty and high colored, but the treatment for the heart is also the treatment for the scanty secretion, which also calls for diluents. The bowels should be kept free to aid in promoting the circulation. To this end nitroglycerine is also indicated and may be given in doses of $\frac{1}{100}$ grain (0.0066 gm.) every two hours.

Moderate stimulation is beneficial. The aromatic spirit of ammonia, and alcohol are the most useful for this purpose. Mental excitement is to be especially avoided after a return to consciousness, and physical rest should be continued. Stimulants are then best discontinued, or continued in great moderation. Care should be taken to protect against the effects of decubitus.

Unfortunately there is no treatment which will restore softened brain matter, although a certain amount of function may be vicariously assumed. The same measures calculated to maintain nutrition and muscular integrity should be taken as are recommended in the treatment of hemorrhage.

INTRACRANIAL ANEURISMS.

Definition.—Intracranial aneurisms are of two kinds, miliary and of larger size. The former have been sufficiently considered when treating of hemorrhage.

Distribution.—Larger aneurisms affect the larger arteries at the base of the brain in the following order :—

(1) Middle cerebral.
(2) Basilar.
(3) Internal carotid.
(4) Anterior cerebral.

The anterior or posterior communicating and vertebral arteries are also occasional seats ; the posterior cerebral and inferior cerebellar rarely. William Osler found 12 of these aneurisms in 800 autopsies, and Newton Pitt 19 in 9000. The aneurism varies in size from that of a pea to a walnut.

Etiology.—Intracranial aneurisms are found rather more frequently in the male sex, and most frequently between the ages of ten to sixty. Osler and Pitt each found one at the age of six. Heredity exercises some influence. Endarteritis and embolism, both of which weaken the vessels, are the chief causes. The former may be syphilitic or simple. The presence of endocarditis should especially invite to examination for them at autopsies.

Symptoms.—*Death* from *apoplexy*, owing to rupture of the aneurism, may be the first intimation. Not only are there often no symptoms but when present they are vague. They may be those of tumor at the base of the brain, optic neuritis, and paralysis of the 3d and other cranial nerves. There are rarely *convulsions*. There may be *headache*, *vertigo*, *nausea*, *hebetude*, and even *coma*, *hemiplegia*, and *hemianopia*. A *murmur*

may be heard on auscultating the skull, while occasionally the patient himself is conscious of a murmur or recognizes the pulsation in his head.

Diagnosis.—This is usually impossible, but the above symptoms, associated with endocarditis, may excite suspicion. Syphilitic disease being as likely to produce tumor gives no assistance in diagnosis.

Treatment.—None exists which can be specifically directed to the disease.

THROMBOSIS OF THE CEREBRAL SINUSES AND VEINS.

Description.—Thrombosis chiefly attacks the sinuses, and is primary or secondary. Primary thrombosis is the result of a state of the blood and circulation; secondary, a consequence of disease adjacent to the sinuses. The former is much the rarer, occurring half as often.

Primary thrombosis is met in connection with general malnutrition and prostration, more frequently in children during the first six months of life as the result of exhausting maladies, especially diarrhœa. It is met also in older children. Recently Brayton Ball and others have shown its association, in young girls, with chlorosis and anæmia. It occurs in the old also as the result of exhausting disease, like pulmonary consumption and cancer.

Coagulation is favored by the trabeculæ which cross the cavity of the sinus, and by irregularities in the shape and lining of the latter. It is mostly in the longitudinal sinus, more rarely in the lateral, sometimes in the cavernous. Phlebitis is not always produced.

Very little is known of thrombosis of the cerebral veins except that it may occur in veins of the convexity as the result of meningitis, and the same causes that produce thrombosis of sinuses.

Secondary thrombosis occurs at any age, and is the result of disease adjacent to a sinus, commonly caries of bone, and is especially frequent as the result of disease of the internal ear. It spreads more frequently from the posterior wall of the middle ear but also from the mastoid sinuses. Fracture, suppurative disease outside of the skull, especially erysipelas, and tumor compressing the sinus may produce it.

Symptoms.—There may be no symptoms in primary thrombosis, or there may be *nausea* and *vomiting*, *headache*, and *hebetude* increasing to *coma*. Dilatation of the pupils, choked discs, and paresis have been reported.

Secondary thrombosis is a septic process. It is commonly announced by a *chill*, followed by *fever* and *occipital pain*, succeeding on earache with suppurative otitis. The sinuses occluded are those near the ear, but the blood escapes by other channels, and the brain substance is not seriously invaded. The *symptoms of meningitis* are soon added. They are headache, somnolence, and stupor, or there may be active delirium and convulsions, rigidity, or optic neuritis, all the results of meningitis. Death is most frequently due to suppurative pulmonary pyæmia, as was the case in 70 per cent. of Newton Pitt's cases, and the appearance of the latter disease under the circumstances is almost conclusive evidence of previous sinus-thrombosis.

Prognosis.—This is always grave. The average duration of the secondary disease is about three weeks, and its termination is almost always fatal. Pitt reports a case of recovery in a boy of ten who had otorrhœa for years, after removal by operation of a foul clot from the lateral sinus.

Treatment.—For primary thrombosis there is no treatment, except that for its cause. For secondary, operative treatment is indicated by trephining or other measures to give exit to pus. Quinine and restorative measures are indicated. Gowers lays particular stress on the use of tincture of the chloride of iron.

THE CEREBRAL PALSIES OF CHILDREN.

Definition.—Referring to the division already made of the motor path into an upper cortico-spinal segment, extending from the cells of the cortex to the grey matter of the cord, and a lower spino-muscular, extending from the ganglia of the anterior horns to the motorial end plates, the diseases now to be considered have their anatomical seat in the former, and are characterized by paralysis, with spasm or disordered movements, exaggerated reflexes, normal electric reactions, without rapid or extreme wasting. They result from a destructive lesion of the motor centres, or of the pyramidal tract in the hemisphere, internal capsule, crus, or pons. They are hemiplegic, diplegic, or aplegic.

Infantile Hemiplegia.

Synonyms.—*Hemiplegia spastica cerebralis* (Heine); *Hemiplegia spastica infantilis* (Bernhardt); *Acute Encephalitis der Kinder* (Strümpell); *Die Atrophische Cerebrallähmung* (Henoch); *Agénése cérébrale* (Cazauvieilh); *Sclérose cérébrale, atrophie partielle cérébrale* (other French writers).

Historical.—In 1884 Strümpell, by a paper, "*Ueber die Acute Encephalitis der Kinder*," called attention to this subject. Since then numerous papers have appeared in Germany, France, and America, among which may be especially mentioned those of Gaudard, Wallenberg, Jules Simon, Morse, Ross, Gowers, Sarah J. McNutt, Weir Mitchell, Wharton Sinkler, H. C. Wood, J. Lewis Smith, and William Osler.

Etiology.—The disease is somewhat more common in girls than in boys, 63 out of 120 cases studied by Osler at the Nervous Infirmary in Philadelphia being of this sex. Of these cases 15 were congenital, 45 in the first year, 22 in the second, 14 in the third, one in the fourth, three in the fifth, sixth, and seventh; one in the eighth, ninth, tenth and above. In ten the age of onset was not given. The hemiplegia was right sided in 68, and left in 52, cases.

Among the causes may be mentioned abnormal conditions of the mother during pregnancy, including accidents, possibly disease, especially syphilis, in a few cases fright or distress, the effect of the last being doubtful. Especially frequent causes are difficult or abnormal labor, in-

jury with forceps producing flexures and fractures of the cranial bones during delivery. After birth are penetrating wounds of the head, ligation of the common carotid, and infectious diseases, including whooping cough, diphtheria, scarlet fever, measles, meningitis, typhoid fever, vaccinia, and mumps. Previous convulsions may cause the lesion on which the paralysis depends, and in a few cases embolism may be responsible.

Morbid Anatomy.—The morbid states of the brain found at autopsy are mainly sclerosis and porencephalus. Embolism and thrombosis of vessels, especially the Sylvian artery, and hemorrhage into the ventricle and substance of the brain, are found in a few cases. The sclerosis involves either groups of convolutions, an entire lobe, or even an entire hemisphere. The skull may be flattened on the affected side, broad and prominent above the mastoid processes, sometimes thickened. The dura may be thickened and adherent, and in one case contained extensive osseous plates. The arachnoid is turbid and thickened and the amount of cerebro-spinal fluid increased. The pia mater may be thickened and adherent and drag portions of the cortex away on its being removed, leaving a roughened surface, while there may be nodular projections of sclerosed tissue. The reduction of weight of the sclerosed hemisphere may be very great, in one case, referred to by Osler in his monograph, the atrophied hemisphere weighed 5.5 ounces (169 gm.), the normal being 20 ounces (653 gm.). The lateral ventricle may be greatly dilated, and the brain tissue over it very thin, while cysts have been found in the sclerosed areas,—the remnants of old hemorrhages. The Rolandic area is that most frequently involved.

In porencephalus there is a localized atrophy or congenital defect of development, resulting in a cavity or depression in the cerebral hemisphere, extending generally into the lateral ventricle.

In 90 cases studied by Osler the lesions in 50 were atrophy and sclerosis, in 24 porencephalus, and in 16 embolism, thrombosis, or hemorrhage.

Symptoms.—The symptoms are complex and varied, but may be divided into three classes : those of the onset, those pertaining to the paralysis, and the sequences.

The *hemiplegia* is usually preceded or accompanied by *convulsions* and *coma*, although the disease may come on suddenly, without spasms or loss of consciousness, in children apparently healthy. In the majority of cases, however, the disease begins with convulsions, partial or general. Loss of consciousness almost always accompanies the convulsions, and may last from a few hours to many days. Rarely coma occurs without convulsions. Among other symptoms may be mentioned *fever*, transient or persistent; according to Strümpell and Gaudard it is an invariable accompaniment of the convulsions. *Delirium* is a common symptom, as is also *soreness* of the general surface. *Vomiting* and *screaming spells* are also noted.

The *hemiplegia* which is noted as soon as the child recovers consciousness is usually complete. Less commonly there is, first, *paresis*, which gradually extends to complete loss of power ; and in some instances a total *paralysis* is established after repeated convulsions. The face is

not always involved, and, as a rule, in facial paralysis of cerebral origin, the superior muscles are intact, and the child can close the eyes and elevate the brows. The facial palsy usually disappears rapidly and completely. As in adults, also, the residual paralysis is most marked in the arm, which is also subject to slow wasting, and is commonly useless for the ordinary purposes of life. The atrophy is moderate, but there may be arrested development, leaving a wasted and withered member. In extreme cases the arm is held close to the side, the forearm strongly flexed at right angles and in a semi-prone position, the hand flexed and the fingers contracted, the palm usually embracing the thumb. Motion may be almost lost in the arm and completely in the fingers, though in most cases there is considerable power of movement, the patient being able to lift the arm above the head, while flexion and extension can be made at the elbow and wrist. The finer and more delicate movements of the hand are rarely recovered. The leg, as a rule, recovers more rapidly and completely than the arm, and the palsy may completely disappear in it, while it rarely does in the upper extremity. In the leg the wasting is also less, while arrested development is also less frequent. A persistent halt is apt to remain—indeed, almost always does—as evidence of impaired power. This may consist in simply favoring the affected side, noticeable only on rapid walking. A decided dragging of the limb is, however, more usual, and there may be tremor of the leg while moving.

The frequency with which *rigidity* is present has given rise to one of the names of the disease, spastic infantile hemiplegia. It is not, however, an invariable symptom, and the paralyzed limbs may be relaxed a long time after paralysis sets in. When rigidity is present it disappears during sleep, and is increased by emotion and forcible attempts to overcome the spasm. Contracture may ultimately result, after which relaxation is no longer possible.

The *reflexes* are almost always increased in the affected limbs, ankle clonus being often obtainable in addition to exaggerated knee-jerk. The reflexes may even be increased on the sound side. Rectus clonus and clonus of the flexors of the fingers are even rarely present, while in a very few cases the reflexes are absent.

Sensation is rarely affected, but vaso-motor derangements are sometimes present. Electrical reactions are normal as a rule.

Post-hemiplegic *chorea*—hemiataxia—is not infrequent. More uncommon are *mobile spasm* and *athetosis* and post-hemiplegic *tremor*. These interesting symptoms were first described by S. Weir Mitchell in a study of cases of cerebral palsy.

In a majority of cases there is *aphasia*, usually transitory, most commonly with right hemiplegia, very rarely with left.

Defects of intelligence are very common, the degree of feeble-mindedness ranging from low-grade imbecility to total idiocy. Psychoses may occur late in life even where there have been no defects in childhood. *Epilepsy* is very frequent, usually confined to the paralyzed side, but also tending to become general. The attacks usually begin within two or three years, sometimes within a few weeks, after the onset of

the hemiplegia, but may be delayed eight to ten years or even longer. The seizures may present three well-defined degrees,—the first, in which the child is simply dazed for a moment or two, or longer, without any motor involvement; second, Jacksonian epilepsy, without consciousness, in which the spasms are confined to the affected side; and third, general convulsions, beginning in the paralyzed limbs and usually accompanied by loss of consciousness. The Jacksonian epilepsy is most common, but all forms may occur in one case.

Diagnosis.—Infantile *spinal* paralysis has most frequently to be excluded, usually without difficulty. The history of the case, including the presence of some of the causes named, the frequent onset with convulsions, the hemiplegia, the absence of rapid wasting of the affected muscles, the retained electrical reactions, are characteristic of infantile hemiplegia in its early stage; while rigidity of muscles, increased reflexes, the peculiar gait, and residual palsy with mental imbecility and epileptic seizures distinguish the later stage.

Tumor of the brain sometimes produces similar symptoms. Tuberculosis and glioma are the forms most common in children. Pressure paralysis by obstetrical forceps affects the face and upper extremities, but other symptoms are wanting, and it is scarcely likely to be confounded with infantile hemiplegia.

Prognosis and Treatment.—The prognosis is favorable so far as life and the recovery of considerable locomotive power are concerned; unfavorable as to recovery from mental defect and epilepsy. An institution for feeble-minded children, where the subjects have the benefit of training and watching, is the safest permanent home for them.

Bilateral Spastic Hemiplegia.

Synonyms.—*Spastic Rigidity of the New-Born* (Little); *Tonic Contraction of Extremities; Essential Contractions; Permanentes Kinder-Tetanus* (Stromeyer); *Spastic Paralysis of Children* (Adams); *Spastic Diplegia* (Gee); *Spasme musculaire Idiopthique* (Delpech); *Birth Palsies* (Gowers); *Little's Disease.*

Historical.—Delpech was probably the first to describe the disease fairly correctly. To the German orthopædic surgeon, Heine, belongs the credit of first appreciating these conditions and their cerebral origin and separating them from common infantile paralysis. His paper was written in 1860. Little's paper, published two years later in England, so attracted attention that his name became applied to the disease. Stromeyer, Adams, and Rupprecht have furnished careful accounts.

Etiology.—A majority of cases of bilateral hemiplegia in children date from birth, and the condition is the result of injury during birth. The infectious fevers are responsible for a certain number, and a few are direct results of convulsions.

Morbid Anatomy.—As may be inferred from the name, the lesions are bilateral and involve motor areas of the cortex almost solely. They consist in sclerosis or porencephalous defect, of which the most fre-

quent primary cause is compression by a blood clot due to meningeal hemorrhage from the veins or longitudinal sinus. A meningo-encephalitis may, however, be responsible for the sclerosis. Descending degeneration of the pyramidal tracts has been found in a few cases. Where examined the cord has been found unchanged.

Symptoms.—These are to be distinguished from those of the next form, cerebral spastic paraplegia,—which the disease closely resembles when the arms are so slightly affected that the palsy is scarcely appreciable. In the diplegic state *all the extremities* must be more or less *spastic*, although the legs almost always are worse than the arms. These cases are further characterized, as are those of spastic paraplegia, by their occurrence *at* or *very soon after birth.*

There may be *convulsions* or a prolonged succession of convulsions immediately after birth. After this or without it there may be noted a *limpness* or flaccidity of *muscles*, an expression of *paresis*, often overlooked, because present at a time when the muscular development of the child is so slight that little is expected of it. Soon, however, the inability to hold up its head may be observed, and when the time comes for it to walk it is noted that the limbs are clumsily used, and when examined are found stiff. In a few instances these symptoms may have been preceded by *convulsions.* As the child grows older it slowly acquires some power so as to be able to sit, but the *legs* are *crossed* and the head is not well supported by the neck muscles. If it is held up, the legs are extended and strongly adducted, and crossed with the feet in the pes equinus or equino-varus position. Occasionally the legs are partially *flexed*, while the stiffness varies greatly, involving, in extreme cases, the whole body, sometimes one side more than the other. It is sometimes constant, at others not. It may be greater on one side than another. The *arms* are usually *stiff* in flexion.

To the spastic symptoms described are added, in certain cases, *spasm* and certain movements known as *athetoid.* In the former, in an attempt at voluntary movement, as taking hold of an object, the fingers are thrown out in a stiff, spasmodic, or irregular manner, or there may be constant irregular movements of arms and shoulders, movements which are usually characterized as choreic. In fact, such cases have been named *chorea spastica*, being differentiated from the congenital choreas by the spastic feature. Spasm rarely affects the muscles of the face, though it does occasionally, causing a continual *grimacing* which does not always disappear during sleep.

The *athetosis* is double or bilateral, resulting in the most grotesque and distorted movements. They consist in a constant contraction and relaxation of muscles, more particularly of those of the fingers of one hand and forearm. Flexion of the fingers of one hand may take place, while those of the other may be extending, and the same may be true of different fingers of the same hand. The shoulder and trunk muscles may be also affected, producing a rhythmical and orderly twisting and bending of the body; or those of the neck, producing a turning of the head from side to side. These movements are all increased under excitement or the effort to do anything.

Spastic Paraplegia.

Synonyms.—*Paraplegia cerebralis spastica* (Heine); *Tetanoid Pseudo-paraplegia* (Seguin); *Spastic Spinal Paralysis* (Erb); *Tabes dorsalis spasmodique* (Charcot).

Definition.—Spastic paralysis of the legs in children.

Historical.—A common affection of children, though only recently understood, was fully described and correctly named by Heine in 1849. Delpech and Stromeyer in Germany, Adams and Little in England, described it. Erb and Seeligmüller in Germany, and Gee in England, brought the subject to the notice of physicians, and Erb contrasted it with the spastic paraplegia of adults. Ross, Hadden, Gowers, d'Heilly, Gilbert, and Osler, have more recently treated the subject as one of the cerebral palsies of children.

Etiology.—The causes are those of spastic diplegia.

Morbid Anatomy.—This is less known than the morbid anatomy of the other forms of cerebral palsy, only two records of autopsy being known to Osler, in each of which was noted a moderate grade of general cortical and descending degeneration of the lateral columns of the cord.

Symptoms.—These are almost identical with those already described as belonging to the spastic paraplegia of adults, with which the earlier writers classed it. Spastic paralysis of the lower extremities, dating from birth or appearing within the first few years of life, with talipes equinus or equino-varus, adductor spasm, rigid stiff gait, the patient walking on his toes or by crossed-legged progression—all without wasting—these are, in a word, the symptoms. The order of sequence of events is very similar to that described under spastic diplegia. In attempts to walk the heels are everted and knees approximated, because of spasm of the adductors, which may be so strong as to make it impossible to separate the thighs. The spastically extended legs may, however, be gradually forced into flexion after the manner of the "lead-pipe" contraction. If, however, the attempt be made to extend the leg the spasm returns. If the extension be gradually insisted upon it often happens that when the extension is nearly complete the spasm suddenly completes it, as the spring acts on the blade of the pocket-knife, whence the name "clasp-knife" rigidity.

Mental imbecility is not so serious as in spastic diplegia or even as in infantile hemiplegia.

Diagnosis.—The distinction between spastic diplegia and paraplegia is usually easy with a good history. There is an affection of children known as pseudo-paralytic rigidity, idiopathic contraction with rigidity, or tonic contraction of the extremities, with which it is sometimes confounded, but the following table of differences from Osler's monograph will aid in separating the two conditions:—

Pseudo-paralytic Rigidity.	*Spastic Paralysis; Di- and Paraplegia.*
Follows a prolonged illness. Is often associated with rickets, laryngismus stridulus, and the so-called hydrocephaloid state.	Usually exists from birth. History of difficult labor, of asphyxia neonatorum, or of convulsions.
Begins in hands as carpo-pedal spasm; often confined to hands and arms.	Arms rarely involved without legs and not in such a marked degree.
Spasms painful and attempts at extension cause pain.	Usually painless.
Intermittent and of transient duration.	Variable in intensity but continuous.

The spasm in the pseudo cases is altogether more extreme and difficult to overcome. The disease is associated with rickets and other constitutional diseases.

Tetany is characterized by a different history and causation. Bilateral rigidity may also be produced by tumors of the pons and cerebellum.

Treatment.—The treatment varies with the stage at which the physician is called. If in the stage of initial convulsion there is no remedy like chloral, which should be given in doses sufficient to control the fits. In the mild degrees, or with a view to keeping up an effect first obtained by chloral, the bromides may be used. If chloral fails chloroform may be inhaled.

In established paralysis medicines do not avail much and recoveries are rare. Hygiene and good food, gymnastics, manipulation, massage, passive motion, and surgical appliances. Baths and electricity should not be forgotten.

The epileptic convulsions should be treated as when occurring under other conditions though the cortical lesions occasioning the disease preclude any expectation of permanent relief. Operative procedure has been suggested in certain selected cases and carried out, but with results which have been disappointing.

The mental deficiencies are best treated in an institution for feeble-minded children, where all such cases should be taken whatever the circumstances of the parents.

SCLEROSIS OF THE BRAIN.

Multiple Sclerosis of the Brain and Spinal Cord.

Synonyms.—*Insular Sclerosis; Disseminated Nodular Sclerosis; Sclérose en plaques.*

Definition.—A chronic affection of the brain and spinal cord, consisting in the presence of numerous sclerotic patches scattered through the nerve centres.

Etiology.—Its precise cause is unknown. The infectious diseases, especially scarlet fever, are alleged causes; so are cold, exposure, mental emotion, and syphilis, but without definite foundation. Hereditary predisposition has been noted. The disease is more common between the ages of eighteen and thirty-five, though Strümpell met a case which came to autopsy at sixty. Both sexes are equally subject. Prichard states that more than 50 cases have been reported in children. It has been thought that the disease depends on anomalies of the vessels, which are generally the seat of chronic endarteritis.

Morbid Anatomy.—The sclerosed patches are widely scattered through the brain and cord, rarely in the cord alone. They may generally be recognized by the naked eye by their grey color and unnatural firmness. On section they appear as greyish-red areas. Histologically they consist of a reticulated fibrillary connective tissue traversed

by a few healthy nerve fibres. In the vessels there is an increase of the nuclei and later a thickening of the walls. Fatty granular cells are present in fresh cases. The axis cylinders are preserved in the nodules for quite a long time after destruction of the medullary sheaths. The favorite seats of the plaques in the brain are the centrum ovale, the walls of the lateral ventricles, the corpus collosum, and the cerebellum; while they are quite numerous in the pons, less so in the medulla, but numerous in the cord, especially the white substance. The cortex, it is said, is not often invaded, but Osler describes the distribution of sclerosis in ten specimens in the Museum of the Institution for Feeble Minded Children at Elwyn, near Philadelphia,* all of which were in the convolutions.

Symptoms.—By no means every case of multiple sclerosis can be recognized, so often are the symptoms united with those of other lesions whose effects predominate, while the slowness of the onset necessitates delay in the recognition of even typical cases. Typical cases do, however, occur, and they present a set of symptoms whence their recognition is more or less easy.

The first of these symptoms is *tremor*, known as "*intention* tremor," because associated with any voluntary effort to perform an act, as picking up an object, raising a glass of water to the lips, or apposing the ends of one's fingers. This does not prevent the ultimate attainment of purpose. When the patient is quiet the tremor ceases, and in this respect can be differentiated from the trembling of paralysis agitans. It is not confined to the arms but occurs also in the head and trunk, so that the head trembles when it is raised from the pillow. It is increased by excitement.

The second characteristic symptom is what is known as *scanning speech*, a slow, measured, yet indistinct and obscure utterance, depending upon disturbances in the innervation of the tongue and larynx, probably due to the presence of sclerotic nodules in the pons and medulla. There may be tremor in the tongue and lips when speaking. The third symptom is *nystagmus*—oscillatory or lateral movements of the eyeball when the eyes are directed to an object. In addition there may be *spastic symptoms* manifested chiefly in the presence of *increased reflexes*—including periosteum as well as tendon—in both upper and lower extremities; but the skin reflexes remain normal. There is ankle clonus, and the gait is often spastic. *Paresis*, at first absent, ultimately appears, amounting at times to complete *paralysis*. The sphincters remain intact, at least until toward the close. There are no disturbances of sensibility in the majority of cases. *Optic atrophy* is sometimes present, less commonly than in tabes, and associated with such *derangements of vision* as amblyopia, achromotopia, and even blindness. *Optic neuritis* occurs with subsequent atrophy, especially in the temporal halves of the optic nerve; also derangements of innervation with diplopia.

Mental weakness and imbecility are sometimes present, more rarely *melancholia or exaltation*. *Apoplectiform* attacks also occur, following

* "The Cerebral Palsies of Children," Philadelphia, 1889, p. 49.

prodromal symptoms such as *vertigo* and *headache*, and succeeded by hemiplegia which, however, subsequently disappears.

Diagnosis.—This is not difficult in typical cases. The intention tremor, the scanning speech, and nystagmus are characteristic, and when associated with spastic weakness can scarcely mean anything else. The apoplectiform seizures and mental weakness are also valuable signs in association. When the symptoms are mixed with those of other nervous lesions, diagnosis is not so easy. In paralysis agitans tremor occurs during rest as well as motion; in multiple sclerosis only when motion is attempted. Strümpell says, "The circumstance, indeed, that the anomalous cases will not properly fit the molds of any other form of disease should make us think of the possibility of multiple sclerosis."

The disease known as *pseudo sclérose en plaques*, described by Westphal, seems to have the same symptoms precisely except nystagmus. The tremor movements are said to be more violent. No lesions are discovered in necropsy.

Prognosis.—This is unfavorable after a long and tedious course, terminating in the bedridden state.

Treatment.—This is unavailing. The end may be delayed by galvanism and tepid bathing. The internal use of nitrate of silver long continued has been recommended.

DEMENTIA PARALYTICA.

SYNONYMS.—*Chronic Diffuse Meningo-encephalitis; Paretic Dementia; General Paresis; Progressive General Paralysis of the Insane.*

Definition.—A chronic progressive meningo-encephalitis, or meningo-rachitis, with resulting mental and motor derangements, terminating in dementia and paralysis.

Historical.—Boyle in 1882, and Calmeil in 1826, by their descriptions, first separated paralytic dementia from other diseases which run a like course. The minute anatomical changes lying at the bottom of the symptoms are a matter of comparatively modern study, and have received valuable contributions from Bevan Lewis.

Etiology.—Seventy-five per cent. of all cases are caused by syphilitic infection. Starting out with this assumption we have at once an explanation of its greater frequency in the male sex, though many women have it; while it is rather a sad commentary on the fidelity of man that it is much more frequent among married men. The fact that it occurs most frequently between the thirtieth and fiftieth year, that it is a disease of the better classes,—especially army officers and artists,—and that it is preëminently a disease of the cities, should be added. Although other factors apparently enter into the causation of general paresis, it is the disposition of those who have most closely studied the subject to assign to them a predisposing rôle. Such influences are heredity and exhausting mental work, such as comes of public political life and ambitious financial ventures. Intemperance, venereal diseases, chronic lead poisoning, and traumatism are generally regarded as causing influences.

Morbid Anatomy.—An atrophy of the brain, and especially of the frontal lobes, may be put down as the most important morbid change. The convolutions are wasted and pale in color, the fissures wider, and the weight of this portion reduced to one-fourth or one-third the normal, while the consistence is firmer and more resisting to section. Other macroscopic changes are a thickening of the dura mater, pachymeningitis interna, œdema of the pia with thickening, opacity, and adhesion to the cortex. Minute examination of the cortex recognizes thickening of the vessel walls due to nuclear infiltration of the adventitia of the arterioles and lymphatic sheaths,—in other words, the effects of mild inflammation. To these are added demonstrable destruction of nerve elements, especially of the fine medullary nerve fibrils running parallel with the surface, known as "association fibres" in the frontal convolutions and island of Reil; also atrophy of the ganglion cells. Associated with this is an overgrowth of connective tissue and numerous Deiter's spider cells. Here enters a contested question as to whether these nerve changes are primary or secondary to an interstitial encephalitis. Tuczek, Wernicke, and Strümpell hold to the former view; while Rindfleisch and Mindel adopt the latter, making the destruction of nervous tissue secondary to the overgrowth of connective tissue.

The white matter is also involved, the central ganglia as well. Coincident changes in the spinal cord,—first described by Westphal,—consisting in fascicular systemic degeneration of the lateral columns and posterior columns, either alone or jointly, are quite constantly present. To these are ascribed a large part of the ataxic and spasto-paralytic symptoms. From this brief statement of the character and situation of the morbid changes it will be seen that they are widespread in their distribution through the nervous system, while they are also degenerative.

Symptoms.—So widely scattered a distribution of morbid phenomena naturally brings about corresponding differences in the variety and degree of the symptoms. As further characteristic, no absolute constancy is observed in the order of their development. As a rule, however, the *first stage* is characterized by abnormal mental processes and these are at first what may be comprehended under the single expression *peculiarity or "queerness" of conduct.* The patient will perform acts wholly unnatural to him, and will surprise his friends and family by breaches of decorum and morality. An apathy and loss of memory, causing the omission of obligations, are also constant. At first these may pass unnoticed as temporary, but their permanence is gradually established. In lieu of this may be present an irritability and intense *restlessness*, so that the patient cannot remain in one spot, but walks constantly to and fro. Not often in this stage is there much volubility, but rather a *morose silence* is observed. In this stage, too, the patient may make rash and ruinous financial ventures, and lose his own money and that of his friends, or he may become very generous, giving away freely all he possesses, and more, too. The power of arithmetical calculation is defective or gone. He may be self-satisfied and intensely egotistical. On the other hand, he may be conscious of these ills and be anxious

about them, as well as experience a discomfort or malaise, for which he may consult the physician.

Nor are *motor disturbances* wholly wanting in the first stage. They are chiefly derangements of speech and handwriting, and are of no small diagnostic value. The speech is slow and hesitating, yet the patient stumbles over syllables, especially where the word is complex or rather difficult to enunciate. As to the handwriting, it is tremulous, characterized by the omission of letters and substitution of wrong ones, as well as erroneous spelling,—all motor defects.

Other symptoms of the first stage are inequality of the pupils, ocular paralysis, and absence of patellar reflex, in tabetic cases often reflex immobility of the pupils, and in spastic cases increase of reflexes. There may be neuralgic pain and attacks of migraine.

The *second stage* is characterized by more *exalted mental symptoms* and excitement, with a *higher degree of motor disturbance.* The former consist in exaggeration of all previously maintained mental symptoms, amounting to noisy, boisterous, and maniacal excitement, and even uncontrollable violence. In this stage belong, too, those extraordinary delusions of grandeur—expansive delirium—in which the patient imagines himself or herself to be a person of great consequence and unlimited wealth. This is not, however, invariable, and there may be an exaggerated degree of the opposite condition of melancholy sometimes present in the first stage; or the two conditions of delirium and depression may alternate. Sleeplessness may be added to restlessness and mental excitement, causing rapid decline of strength.

Motor disturbances are greatly increased, but a uniform order of invasion is by no means always observed, while remissions and temporary improvement are often noticed. *Speech* becomes almost impossible and incomprehensible. There is paraphasia, persistent repetition of words, and reading and writing are impossible. The voice can no longer be modulated, is weak and rough from imperfect innervation of the vocal cords.

The gait becomes defective and the patient often trips in walking. There may be *ataxia* and other tabetic symptoms; apoplectic seizures with *paralysis,* or epilepsy with grand or petit mal and aura, sometimes one-sided and followed by monoplegia or hemiplegia. There may be loss of *sensibility,* with bladder and rectum paralysis. The tendon reflexes may be lost and the pupil be immobile, or the opposite condition of spasm with increased tendon reflexes prevail. The paralytic attacks may occur in the earlier stages, though in mild degree, consisting in vertigo or obscuration and loss of consciousness, lasting for a short time and then passing away. There may be local twitching in the face and extremities and even typical Jacksonian epilepsy. Finally, *bulbar* symptoms may appear with invasion of the medulla. Ultimately, the patient becomes helpless, bedridden, and completely demented, dying from exhaustion or intercurrent disease.

In a few cases none of the mental symptoms described are present, but a gradual decline of mental power takes place until complete dementia supervenes. An *acute variety* is also sometimes met with, properly

termed "galloping," in which the disease runs its whole course in a few months, and is especially characterized by emaciation and rapid loss of strength due to restlessness, sleeplessness, and insufficient food. The pulse and temperature are essentially normal, or at least there are no characteristic variations.

Diagnosis.—To recognize paretic dementia *ab initio* is perhaps impossible, but to watchful observation the disease commonly reveals itself after the symptoms have existed for a short time. The early symptoms resemble those of neurasthenia, but differ from those of the latter disease in their steady progression. Other affections possibly mistaken for it are cerebral syphilis, tumors of the brain, and multiple sclerosis. In cerebral syphilis the onset is usually more sudden, and paralytic symptoms appear earlier. Headache is more frequent and severe, and there may be convulsive seizures, but affections of the tongue and speech are wanting, while the train of mental symptoms is less complete and characteristic, and expansive delirium, as a rule, does not occur. The epilepsy is more commonly Jacksonian. It is to be remembered that the syphilitic virus produces both, and it is not unnatural that the two should sometimes merge. Tumors of the brain produce symptoms more localized, and often also optic symptoms including choked disc. The symptoms of insular sclerosis, which include dementia, are often identical with those of paralytic dementia, and the two diseases cannot then be distinguished. Intention tremor is more characteristic of sclerosis. The cerebral symptoms of some forms of plumbism, it is said, also sometimes closely resemble those of paralytic dementia.

Prognosis.—The prognosis is almost always unfavorable, although the course of the disease varies somewhat. The most rapid cases of the galloping form may terminate in a few months, but two or three years is the more usual duration, sometimes much longer, it may be ten years or more. Death ensues from exhaustion, hastened by the complications and secondary conditions which naturally supervene on an illness so prolonged and in which nutrition is so interfered with; or it may be due to intercurrent disease.

Treatment.—In view of the general acknowledgment of the syphilitic origin of chronic diffuse meningo-encephalitis and the acknowledged efficiency of anti-syphilitic treatment over tertiary manifestations of the disease, it is rather surprising that attempts at curative treatment are so futile. There is, however, little doubt as to what should constitute the treatment by medicines. It is confined mainly to iodides and mercurials. Mercurials are best used by inunction, and the iodides in ascending doses.

As to the rest, treatment must be symptomatic. The bromides and chloral, with quiet, wholesome surroundings, and sometimes enforced retirement, are the measures demanded for the relief of the nervous excitement.

For the opposite condition of depression and melancholia change of scene by travel and residence in different localities should be enjoined. Further than this the use of a proper hygiene, with bathing, frictions, wholesome out-door life, and an abundance of nourishing and easily assimilable food constitute about the sum of the means we can bring to bear against the disease.

TUMORS OF THE BRAIN.

SYNONYMS.—*Neoplasmata cerebri; Intracranial Tumors.*

Definition.—Cerebral tumors, clinically considered, include not only tumors of the meninges and substance of the brain, but also all intracranial and even such extracranial tumors as ultimately invade the brain. Among the latter are tumors of the orbit or nasal cavity, of the antrum, and the spheno-palatine fossa.

Varieties.—The principal varieties of cerebral tumor, approximately in the order of frequency, are :—

(1) Tyroma.
(2) Gumma.
(3) Glioma.
(4) Carcinoma.
(5) Sarcoma.
(6) Psammoma.
(7) Myxoma.

And after these, in irregular order, any of the remaining histioid tumors,—cholesteatoma, lipoma, angeioma, fibroma.

Of these tumors psammoma and glioma are peculiar to the brain. Even dermoid cysts and parasitic products—including the echinococcus—or hydatid cyst and the cysticercus cellulosæ, are met with.

Etiology.—Except sarcoma, tumors are found in males more frequently than in females. C. L. Dana has tabulated the relative frequency of tumors at different ages as follows :—

Childhood,	tubercle, parasites.
Early life,	gumma, glioma, parasites.
Early and middle life,	sarcoma, glioma, gumma.
Middle and late life,	sarcoma, gumma, cancer.

Brain tumors of any kind are rare after sixty. Heredity appears to have slight, if any, influence. A few brain tumors are metastatic, especially carcinoma, and to a less degree sarcoma. Eichhorst relates several remarkable cases in which trauma seemed to be the exciting cause.

Certain tumors seek by preference special localities. Thus tubercular tumors are most numerous in the cerebellum and about the base of the brain. Syphiloma elects the hemispheres or the pons and vicinity; it is often multiple, generally superficial, grows from the meninges, or is attached to arteries, attaining sometimes a large size. Glioma starts from the neuroglia of the white matter in any part of the brain, but more frequently the cerebrum, and may also attain a large size,—larger than any other brain tumor; it is further characterized at times by its great vascularity, leading sometimes to rupture and apoplectic symptoms. Sarcoma develops also most frequently in the membranes of the brain and sheaths of the vessels; it has been found in the pons; it may be primary or secondary; it is often encapsulated. Myxoma and fibroma occur in the same localities. Carcinoma is usually secondary, but may be primary; arises more frequently in the membranes, but may be found in the sub-

stance of the hemispheres; it is especially secondary to primary cancer of the breast, lungs, or pleura. The hydatid cysts developed by the echinococcus are usually on the surface of the brain; the cysticercus, usually multiple, on the surface or in the ventricles.

Symptoms.—The symptoms of cerebral tumor are in no way specialized by the kind of tumor present, and depend entirely upon the effect they exert on the surrounding brain substance, chiefly by pressure. They do, however, vary somewhat with the part of the brain involved. It occasionally happens that a brain tumor may produce no symptoms whatever, being thoroughly latent, and disclosed only by the autopsy. On the other hand, apparently insignificant tumors cause very decided symptoms. Such differences may depend in part on the location of the tumor, and in part on the rapidity of its development.

As in all local diseases of the brain, two sets of symptoms usually present themselves:—

(1) Diffuse, and

(2) Focal symptoms.

(1) *Diffuse or General Symptoms.*—The most constant of these is perhaps *headache*, which varies in intensity and constancy. Probably the severest headaches it is given human beings to suffer are caused by brain tumors, exhibiting every variety of pain, sharp, cutting, shooting, boring, or dull and pressing. At times it is moderate, producing a sense of discomfort only. It may be intermittent or constant. It may be over the entire head, or half of it, or be still more localized in the forehead or back of the head, extending also from the former over the face and the latter down the neck. It may be increased by mental excitement of any kind, by noise, or by alcoholic drink, or intense light. There may be tenderness on pressure, or pain in percussing the head. The seat of pain is, however, for the most part, no indication of the seat of the tumor, though the presence of pain limited to the occiput and back of the neck suggests a tumor in the posterior fossa of the skull, the occiput, or the cerebellum. Localized pain on tapping the skull is a more reliable index.

Vomiting is another characteristic symptom of brain tumor. It may occur independent of headache, but is often associated with it. It is further characterized by being independent of food-ingestion, and without nausea, and is apt to be worse in tumors of the cerebellum and pons.

Dizziness is also a very frequent symptom, and often an early one. It is at times intermittent, at others constant, and it may be so severe as to make it impossible for the patient to walk. It is most serious in tumors of the posterior fossa and of the cerebellum. Along with vertigo may be *slowing of the pulse*.

Mental symptoms may be present. They may be intermittent, and variously manifested in peculiarities of temper, such as sullenness, indifference, absent-mindedness, and loss of memory; or the opposite condition of maniacal excitement or delirium. Or there may be drowsiness and even coma. Such mental states may, indeed, be the only manifestations of tumor. The patient may talk slowly, and the facial expression is sometimes altered.

Apoplectic seizures and *epileptiform attacks*, especially of the Jack-

sonian variety, are distinctive symptoms. The former may be due to hemorrhages in the tumor or around it, and may be followed by transitory paralysis and paresis. Epileptic convulsions, especially if unilateral, point, though not unmistakably, to tumors in the hemispheres impinging on the cortex. *Choreiform movements* are sometimes present.

Choked disc or *papillitis* is the most constant and most valuable diagnostic symptom of brain tumor. It consists, in brief, in a swelling of the optic nerve, with over-distention and congestion of the retinal veins, and narrowing of the retinal arteries. It is usually bilateral. There is still some difference of opinion as to the mechanism of choked disc, but it is probably the result of intracranial pressure forcing the cerebrospinal fluid from the subarachnoid space into the lymph sheath of the optic nerve, causing compression of the nerve and the vessels which flow through it. The vision is not necessarily deranged in choked disc, nor are its defects uniform, varying from slight amblyopia to total blindness. The swelling may diminish, and improvement in vision ensue, but retinitis or neuro-retinitis may set in with consequent nerve atrophy, producing permanent impairment of vision. The choked disc is sometimes the only symptom of brain tumor, and its subject first consults the oculist for relief. On the other hand, it is not caused by brain tumor alone, but it may result from meningitis or abscess, in fact anything which produces increased intracranial pressure.

The *senses of smell and hearing* may be impaired by tumors impinging on the olfactory or auditory nerves, and there may be *pruritus* and other modifications of cutaneous sensibility; also neuralgic pains. If the tumor is on the floor of the 4th ventricle, there may be *polyuria* and *glycosuria*. Finally, sooner or later the *appetite* may fail and the *nutrition* suffer, although the opposite condition may obtain of large appetite and good nutrition. In the terminal stage there may be *irregularity* of *breathing* (Cheyne-Stokes) and slowing of the pulse, while the final issue is often preceded by a *febrile movement*. The local temperature in brain tumor is usually raised from 92° to 95° F. (33° to 34.9° C.), and even 98° F. (36.7° C.).

(2) *Focal Symptoms.*—These are the result of irritation or destruction of certain parts of the brain and become, therefore, of value in diagnosis. They are either irritative or destructive, the former producing contractions and spasm, the latter paresis and paralysis. For convenience in localization the brain may be divided, as in figure 89 after C. L. Dana, into :—

(1) The prefrontal area, including all anterior to a line starting from the upper end of the ascending branch of the fissure of Sylvius at right angles to a line drawn between the frontal and occipital ends of the brain.

(2) The central region, bounded in front by the line just named and behind by a line limiting the posterior central convolution prolonged downward to the Sylvian fissure.

(3) The parietal lobe.

(4) The occipital lobe.

(5) The temporal or temporo-sphenoidal area.

(6) The corpus callosum.
(7) The great basal ganglia and capsules.
(8) The corpora quadrigemina, deep marrow, and pineal gland.
(9) The crura cerebri.
(10) The pons and medulla.
(11) The cerebellum.
(12) The basal ganglia of the brain.

The boundaries of these territories are shown in the accompanying figure :—

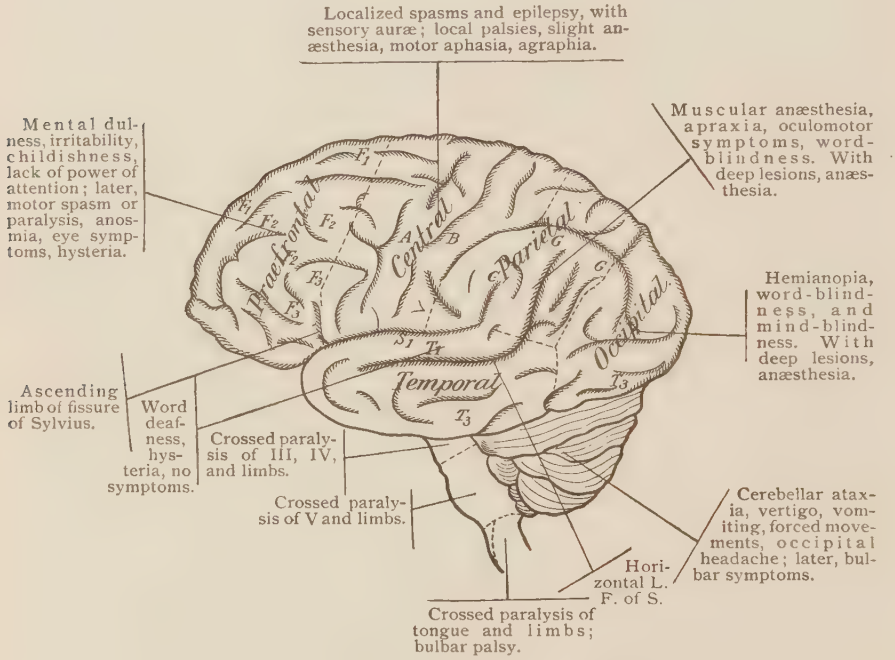

Localized spasms and epilepsy, with sensory auræ; local palsies, slight anæsthesia, motor aphasia, agraphia.
Mental dulness, irritability, childishness, lack of power of attention; later, motor spasm or paralysis, anosmia, eye symptoms, hysteria.
Muscular anæsthesia, apraxia, oculomotor symptoms, word-blindness. With deep lesions, anæsthesia.
Hemianopia, word-blindness, and mind-blindness. With deep lesions, anæsthesia.
Ascending limb of fissure of Sylvius.
Word deafness, hysteria, no symptoms.
Crossed paralysis of III, IV, and limbs.
Crossed paralysis of V and limbs.
Cerebellar ataxia, vertigo, vomiting, forced movements, occipital headache; later, bulbar symptoms.
Horizontal L. F. of S.
Crossed paralysis of tongue and limbs; bulbar palsy.
Prefrontal
Central
Parietal
Occipital
Temporal

FIG. 89.—SHOWING FOCAL SYMPTOMS OF BRAIN TUMOR.—(*After Dana.*)

(1) *Tumors of the prefrontal area* often give no localizing symptoms whatever, motor or sensory, while general symptoms may also be absent and the tumor truly latent. Then again, general symptoms may be well marked, including mental torpor and imbecility, childishness, irritability, and emotional phenomena. If it extends downward into the inferior frontal convolution, the tumor may cause aphasia; or if backward, it may occasion irritative spasm or destructive paralysis. Involvement of the optic nerve may cause hemianopia and optic neuritis; of the olfactory nerve, anosmia; if it invades the orbit, oculomotor paralysis and protrusion of the eye.

(2) *Tumors in the central region* may cause irritative lesions in the upper third of this area resulting in spasm, beginning in the toes, in the ankles, or muscles of the leg; in the middle third, spasm beginning in the fingers, in the thumb, in the muscles of the wrist or shoulder; in the lower

third in the muscles of the face, the angle of the mouth, or tongue. All of these may be preceded or associated with sensory disturbance, such as numbness and tingling, and may be limited to one muscle group before extending to another, constituting the "signal symptom" of Seguin. There may be an aura, and the muscular sense is also sometimes affected.

Destructive lesions cause paralysis, and this may have the same distribution as the convulsions which sometimes precede. If on the left side, aphasia and agraphia may result.

(3) *Tumors of the parietal area* may produce no symptoms or very slight sensory and motor phenomena. With the involvement of the angular gyrus and lower lobule may come word-blindness and mind-blindness; and with that of the supra-marginal gyrus the obliteration of the muscular sense. If the tumor is higher up, near the longitudinal fissure, the muscles of the lower limbs may be involved; and if upon the central area, spasms and paralysis of the various muscular groups described under (2). Paralysis of the 3d nerve has occurred in connection with tumors in the neighborhood of the angular gyrus.

(4) *Tumors of the occipital lobe*, if in the cuneus and first occipital convolution, may produce homonymous hemianopia; and if double, total blindness; if elsewhere on the left side, there may be mind-blindness; and if they extend also into the angular gyrus, word-blindness, along with hemianopia; if obtruding further forward into the parietal lobe, hemianæsthesia, hemiataxia, and perhaps some hemiplegia from involvement of the internal capsule may occur.

(5) *Tumors of the temporo-sphenoidal area on the right side* rarely produce symptoms; on the left side, in the posterior part of the 1st and upper posterior part of the 2d gyrus, they cause word deafness. When extending well down they produce vertigo or forced movements, owing to irritation of the internal ear. Disturbances of the sense of smell and taste may result from involvement of the hippocampal convolution.

(6) *Tumors of the corpus callosum* are rare. The symptoms are much the same as of tumors in the 3d and lateral ventricles of the brain, extending peripherally. They cause general symptoms of brain tumor, with gradually developing hemiplegia, and later paraplegia. With this there is mental dulness and drowsiness and indisposition to speak. The cranial nerves are not involved.

(7) *Tumors of the basal ganglia and the capsule* produce symptoms similar to those that occur in the corpus callosum. They are mainly pressure symptoms. There is progressive hemiplegia, with which there is apt to be anæsthesia and stupidity less marked. Sometimes there are choreic movements if the tumor involves the optic thalamus and adjacent parts of the internal capsule. Tumors of the caudate nucleus alone, as of the lenticular nucleus alone, are generally latent; so are those of the anterior three-fourths of the optic thalamus, except that sometimes choreic and athetoid movements are noted, due to irritation of fibres of the internal capsule. A large tumor of the thalamus may involve the fibres of the optic radiation and cause hemianopia or sometimes

hemianæsthesia. This may be differentiated from hemianopia due to lesions of the occipital lobe by the presence of the hemianopic pupillary reaction, in accordance with which a ray of light thrown on the insensitive part of the retina will not produce a reflex contraction of the pupil. Optic neuritis is apt to be an early symptom of tumors in this vicinity.

(8) *Tumors of the corpora quadrigemina* usually involve the crura as well. They are characterized by incoördination, forced movements, and oculo-motor palsies, to which may be added hemianopia or blindness due to destruction of the primary optic centres; the pupillary reflex is lost and there is nystagmus.

(9) *Tumors of the crus* from involvement of the 3d nerve are especially characterized by oculo-motor paralysis on one (the same) side and hemiplegia on the other. Tumors of the crus are, however, rare.

(10) *Tumors of the pons and medulla* produce two sets of phenomena:—

(*a*) Irritation or destruction of fibres in the pons and medulla.

(*b*) Pressure on the nerves emerging into this region.

Either may occur alone or both jointly. Lesions here are especially apt to produce alternate paralysis; that is, involvement of the cranial nerves on one side and the limbs on the opposite side.

If the tumor is high up in the pons, there will be a palsy of the 3d nerve on the same side and a hemiplegia on the opposite side; if lower down, a palsy of the 5th on the same side and hemiplegia on the other; if still lower down, it may involve the 6th nerve, producing internal strabismus, the 7th producing facial paralysis, and the auditory causing deafness. If the tumor is very large, it may produce a hemianæsthesia as well; if seated superficially on the lateral edge of the pons, involving the peduncles, there may be forced movements of the body, either toward or from the side of lesion. Conjugate deviation of the eyes away from the side affected may also occur. This is in direct contrast to the conjugate deviation sometimes noted in cerebral lesions, where the head and eyes are turned toward the side of lesion.

Tumors of the medulla will produce hemiplegia and hemianæsthesia, and, if the tumor is large, symptoms of bulbar paralysis. From irritation of nerves on the same side, the 9th, 10th, 11th, and 12th, difficulty in swallowing, irregular action of the heart, irregular breathing, and vomiting may arise. Sometimes also there is retraction of the head, or sensory symptoms including numbness and tingling and finally convulsion. If the cerebellum is impinged upon, there may be unsteadiness of gait.

(11) *Tumor of the cerebellum* produces very characteristic symptoms, though here, too, there may be latency if the growth is limited to the hemispheres. If the middle lobe is invaded, vertigo, vomiting, headache, and cerebellar ataxia are present; more rarely optic neuritis, nystagmus, and neuralgic pains in the neck and occiput occur. The irregular and staggering gait of cerebellar ataxia is very striking, the patient reeling like a drunken man, or he may be thrown sideways or forward, rarely backward, by forced movement.

If the medulla is compressed by the tumor, vomiting from this cause may ensue, also bulbar symptoms and glycosuria; while there may be

distention of the lateral ventricles, causing hydrocephalus and bilateral rigidity from pressure on the motor paths.

(12) *Tumors of the base*, if of the anterior fossa, produce symptoms much like those of tumors of the prefrontal area, adding, however, anosmia from destruction of the olfactory lobe, while there may be also involvement of the optic and oculo-motor nerves and of the orbital contents. Tumors of the middle fossa and of the interpeduncular space produce pressure on the optic chiasm with consequent neuritis and the peculiar forms of hemianopia by which lesions of this area are distinguished from those in the anterior fossa.

Diagnosis.—This consists first in the recognition of the presence of tumor from the general symptoms, and then the determination of its location in either hemisphere from the focal symptoms. The same symptoms may be produced by any agency causing pressure on these structures. It may be meningeal thickening, hemorrhage, aneurism, abscess.

The nature of the tumor may be determined in part by what has been said of the preference for certain localities and the age of the patient, and in part by the history, say of tuberculosis or syphilis or primary growths elsewhere. The surface temperature is of uncertain value in diagnosis. Death may be sudden, especially from growths near the medulla. It is usually the result of increasing pressure.

Prognosis.—This is generally unfavorable. It is true that in some rare instances the brain tumors cease, after a time, to grow. In something less than five per cent. of the cases they are removable by operation, but four-fifths of all persons operated on perish. When due to syphilis they may be melted away by mercurials and iodides. Calcification is a rare but happy termination of tubercular growths. The duration of tumor averages two or three years ; the extremes from a month to many years.

Treatment.—This is medicinal, hygienic, and operative. The first is limited in its purpose to the cure of syphilitic tumors and, perhaps, in a slight degree, to tubercular. The astonishing effect of the mercurial and iodine treatment upon syphilitic new formations is nowhere so well shown as upon cerebral gumma. Unless syphilis can be excluded with absolute certainty, the iodide of potassium should be given in any case in ascending doses, limited only by their effects. In the absence of syphilis the larger doses are not well borne. In addition, mercury should be used, at first preferably by inunction until the specific effect is produced, after which it may be discontinued, to be renewed as indicated. Instead of inunction the bichloride may be given internally in doses of $\frac{1}{12}$ grain (0.005 gm.) three times daily, or until the physiological effects are produced. When the tumor is once under control, it is still necessary to keep up the treatment in such doses as experience may determine to be necessary. Usually the iodide of potassium is sufficient for this purpose. Thus I have a patient in whom for twenty years the disease has been kept in check by a dose of 60 grains (four gm.) a day, which has sometimes to be doubled for a time. If mercury is necessary in this stage, the biniodide may also be used in doses of $\frac{1}{24}$ to $\frac{1}{12}$ grain

(0.0025 to 0.005 gm.), as required, though I never have that feeling of confidence in it that I have in the separate use of the iodide of potassium and the bichloride of mercury.

In tyroma the usual constitutional treatment of tuberculosis by cod-liver oil, iron, and other tonics, with nourishing food and healthful in-door and out-door life is to be carried out. The usual remedies indicated to relieve pain are to be used, bromides, if necessary, in large doses, phenacetin, antifebrin, and antipyrin, and, if necessary, morphine. The ice-cap may be used, and, above all, leeching tried. The most magical effect is sometimes produced by free leeching, though it is unfortunately of but temporary duration. Other symptoms should be treated by appropriate remedies.

The hygienic treatment is of the greatest importance. Excesses of every kind should be avoided, alcohol should be rigidly excluded, as well as all sexual excitement, and mental excitement of any kind, for the slightest increment of blood in the brain may bring on a convulsion and cause death.

Exploratory operation, being comparatively harmless with the aseptic precautions of the present day, should be made wherever the tumor can be localized with any approach to accuracy. Although cerebral localization has been developed to a very high degree, it must still happen that we frequently fail to locate a tumor accurately.

SUPPURATIVE ENCEPHALITIS.

Synonyms.—*Inflammation of the Brain; Encephalitis; Cerebritis; Abscess of the Brain.*

Definition.—By encephalitis is meant inflammation of the substance of the brain as contrasted with its membranes. What is spoken of as inflammation of the brain in popular parlance is really inflammation of the membranes of the brain, or meningitis. A literal application of the term is here intended.

Etiology.—The causes of cerebritis are—

(1) Traumatic.

(2) Extension from an adjacent focus of inflammation.

(3) Pyæmia.

Under traumatic causes are included blows upon the head or falls, more commonly attended by fracture or punctured wound; although it is not necessary that there should be even a scratch upon the skin.

Under adjacent disease, whence extension of inflammation is especially frequent, is to be included caries of the petrous portion of the temporal bone due to disease of the middle ear or labyrinth, the most common of all causes of abscess of the brain. Disease of the orbit is another focus of the same kind. The route of such a communication may be either through the sinuses of the brain or the lymph paths.

Pyæmic abscess of the brain is rare. Causal foci are malignant endocarditis, gangrene of the lung, chronic bronchitis with bronchiectasia, bone disease, suppuration of the liver, and the specific fevers, among which may be included *la grippe*.

Encephalitis occurs most frequently between the ages of ten and forty, and about three times as often in the male sex as in the female.

Morbid Anatomy.—Abscesses of the brain are usually solitary, though there may be two or three or even more. The abscesses may be from a half to three inches (one to eight cm.) in diameter, rarely more, though the entire lobe has been involved. The abscess itself is a very interésting product. Unless very recent, it is surrounded by a distinct wall which is composed of three layers. The inner is smooth, made up for the most part of fatty granular cells. Outside of this is a layer of germinal tissue containing spindle cells and more perfect fibrillated tissue. Externally again is another layer of fatty cells. The pus within the abscess is usually greenish-yellow in color, and acid in reaction, while its corpuscles are distinctly nucleated from the effect of the acid ingredient on the cell. The zone outside of the abscess is œdematous, the cells are swollen, sometimes disintegrated, with blood points scattered throughout, becoming sparser as the periphery is extended.

The abscess is preceded by the condition known as *red softening*, which is often spoken of as the first stage of the inflammation, but it is most important to remember that red softening is not peculiar to abscess. It consists simply of brain substance broken down into a reddish blood-stained pulp. In this substance are found fragments of nerve tubules, drops of myelin, pus corpuscles, and fatty granular cells. The termination of cerebritis is not always in abscess. It is barely possible, before the stage of abscess is reached, for a condition of *yellow softening* to supervene, and the so-called apoplectic cyst be the final result.

The cerebrum is involved four times as often as the cerebellum, the left hemisphere more frequently than the right, and the temporo-sphenoidal lobe more than any other. The cause has something to do with the location: Ear disease places the abscess in the temporal lobe or cerebellum; if in the tympanum, the cerebrum rather than the cerebellum; if the mastoid cells and labyrinth, the cerebellum.

Symptoms.—While inflammation of the brain is spoken of as acute and chronic, more strictly speaking it is rather primary and delayed,—the symptoms of the so-called chronic form being essentially the same as those of acute cerebritis, but characterized by their late appearance after the cause which precedes them. In acute cases the symptoms develop rapidly and may run their course in a few days; while in the forms known as chronic the symptoms are scarcely less rapid after they once set in, which may be weeks, months, and even longer, after the operation of the cause.

These symptoms are the result of pressure,—direct or indirect,—of destruction of the brain substance, or of poisoning due to absorption of putrid matter. They are much the same as those of meningitis, with which, indeed, abscess is often associated, especially if there is injury. The most striking are *headache*, often severe and persistent; *vomiting; vertigo; mental dulness*, succeeded sometimes by *delirium* and sometimes by *coma*. *Convulsions* are often present, and are epileptoid in character. *Optic neuritis* is also one of the symptoms. There is usually *fever*, as shown by elevation of temperature. At other times the temperature is

normal or subnormal. The *pulse* is usually slow, 60 to 70. The symptoms may set in with a *chill* after the latent period. The toxic symptoms are those usual to toxic states, viz., chill, irregular fever, prostration, emaciation, exhaustion. *Paralysis* in the form of hemiplegia sometimes occurs. The paralysis, however, is not always hemiplegic, and may be limited to the arm and face, especially in abscess of the temporo-sphenoidal lobe, which may compress the lower motor centers. If on the left side, there may be *aphasia*. Other cranial nerves besides the optic are sometimes involved.

Of the *chronic* form it has already been said that the symptoms, though long delayed, are the same as those of the acute form. Such delay, however, does not always cover all symptoms, since, during the latent stage the patient may have headache or vertigo in a mild degree, and especially may he be irritable and depressed, while he may even have a convulsive seizure during this preliminary period. It occasionally happens that there are no symptoms at all and cases have occurred, more particularly of abscess in the frontal lobe, where there were no signs or symptoms before death.

When the abscess is in the parieto-occipital region there may be *hemianopia*. It is especially in abscess of the cerebellum that *vomiting* occurs, and *staggering* if the middle lobe is affected.

Phlebitis of the superior petrosal and lateral sinuses is especially common when the abscess is caused by disease of the ear, since the former receives a vein from the internal ear, and the latter the mastoid veins. *Œdema about the ear and neck* and *hardness of the jugular veins* should suggest phlebitis, while *rigidity* of the neck and *cranial nerve paralysis* even more unerringly point to meningitis.

Acute cases last from eight to fourteen days, rarely thirty days; the delayed cases may not show their first symptoms for months. In the curable form referred to Strümpell says pronounced symptoms of focal disease exist for a time and suggest a tumor, but after some months or even a longer time they gradually abate and recovery is complete. The nature of the symptoms is such as to suggest a seat in the cortex, for there is usually paresis of some part of the body, often associated with symptoms of motor irritation and impairment of speech.

Diagnosis.—This is easy in acute cases, being substantiated by the history of injury, rigor, and fever, followed by the brain symptoms described. Almost as certain is the diagnosis when such symptoms follow chronic ear disease or localized putrid lung disease. It is to be remembered, however, that simple ear disease may produce some general cerebral symptoms, in which is included optic neuritis. In such cases it is simply impossible to make the diagnosis until the course and termination of the disease have excluded one or the other. In like manner meningitis and abscess may be confounded, and with reason, because, in the first place, meningitis may be produced by the same causes as abscess; and second, meningitis may be caused by abscess. Meningitis, however, affects the cranial nerves more than abscess, unless the abscess is seated in the pons, and usually meningitis succeeds more promptly upon its cause. It is to be remembered that tumor of the brain may

produce identical symptoms with those described. The chief distinctive symptom is the presence of fever in abscess.

Prognosis.—This, unless we admit a curable form described by Strümpell, is always ultimately fatal unless we have the rare good fortune to reach it with the trephine.

Treatment.—A certain prophylaxis may be exercised in the proper treatment of disease of the ear ; for it is often the neglect of this which leads to the abscess. Such prophylaxis includes measures which secure free discharge and antisepsis. Beyond this the only treatment for abscess which promises anything toward a favorable result is operation, on which account the surgeon should be promptly associated in the treatment of the case. The use of the trephine has saved a few cases. For the details of the operation the student is referred to surgical works.

Encephalitis Without Abscess.—When, on the one hand, inflammation of the surface of the brain accompanying meningitis is eliminated, and, on the other, softening of the brain, formerly thought to be the result of inflammation but now known to be due to the arrest of blood supply, it leaves a very small number of cases of possible encephalitis without abscess, which both Gowers and Strümpell admit as possible.

CHRONIC HYDROCEPHALUS.

Definition.—A collection of serous fluid either between the meninges,—which constitutes intermeningeal hydrocephalus, *hydrocephalus externus*, or *hydrocephalus ex vacuo*,—or in the ventricle of the brain, constituting ventricular hydrocephalus or *hydrocephalus internus*. The seat of effusion in hydrocephalus externus may be either in the subdural space, *i. e.*, between the dura mater and the arachnoid, or in the subarachnoid space. The first was formerly regarded as the more frequent, later its occurrence came to be denied, but more recently, by means of frozen brain sections, it has been demonstrated. Since the subarachnoid space communicates with the ventricles of the brain, the two forms of hydrocephalus may coexist. Both external and internal hydrocephalus may be diffuse or circumscribed, producing in the case of the former cystic spaces in the membranes, and in the latter vesicular distention of portions of the ventricles.

External Hydrocephalus occurs in connection with atrophy of the brain and is not of much clinical importance.

Internal Hydrocephalus.

This is divided into congenital and acquired.

Congenital Hydrocephalus.

This preëxists before birth, and may be present to such a degree as to obstruct the birth of the head. More frequently it is not recognized until some time after birth.

Etiology.—This cannot be said to be certainly known. Virchow early ascribed it to inflammation of the ependyma; Rindfleisch rather to an obstruction to the circulation in the choroid plexus, which in hydrocephalus is always hyperæmic and beset with vascular papillæ. Drunkenness and syphilis in parents, and accidents in pregnancy, are held responsible; occasionally also tumors of the brain. More than one child in a family is sometimes affected.

Morbid Anatomy.—The head is characterized externally by its spherical shape and large size, its smooth eyebrows and protruding eyes; the last being due to depression of the orbital plate of the frontal bone, and is often so great that the eyelids cannot close over the eyes. The size of the head thus obtained is often enormous, eight to ten inches (20 to 25 cm.) in diameter in a child of three or four years. On the other hand, the face appears very small. On closer examination the cranial bones are found separated and exceedingly thin, at times almost like paper. In the membranous interspaces are often found Wormian bones. The veins may be seen beneath the skin, and fluctuation may sometimes be obtained On incising the brain a variable quantity of limpid fluid passes out. The quantity is sometimes enormous, reaching 20 pounds (40 kilos.) or more. The cerebral cortex is greatly thinned, the thickness on the convexity being reduced to but a few millimeters. The gyri are compressed. The ventricles are dilated, and the basal ganglia are condensed. The commissures are stretched, and even torn. The foramen of Monro is a wide opening, and the 3d ventricle is dilated, and sometimes also the 4th. The ependyma is thickened, the choroid plexuses vascular, sometimes a little changed.

Symptoms.—These consist largely of the external morbid states just described, but in addition there is *slowness of physical and mental development.* The child learns to walk late, and is very feeble, and is apt to be mentally deficient, although sometimes bright. Convulsions may occur, and the reflexes be increased. The congenital case rarely lives to be more than four or five years old, though sometimes it may attain adult life. The rachitic head may be mistaken for the hydrocephalic, but the latter has not the broad forehead with prominent frontal eminences; it is rather spherical and smooth.

Acquired Hydrocephalus.

Etiology.—This is also commonly ascribed to some inflammatory process, although it is said to be sometimes idiopathic. Especially is it a consequence of suppurative and tubercular meningitis, when it is spoken of as acute acquired hydrocephalus, though chronic inflammatory processes may also cause it. Derangements in the circulation in the choroid plexus and in the ependyma of the ventricles may, however, be responsible. Especially may a tumor in the 3d ventricle, at the base of the brain, pressing upon the venæ Galeni or on the right straight sinus of the dura mater, be a cause; or closure of the foramen of Magendie by which the ventricles communicate with the membranous spaces. Even lung or heart affections and growths in the mediastinum and neck may produce the needed obstruction.

Morbid Anatomy.—In cases of acquired hydrocephalus, even though beginning tolerably early in life,—say the seventh year,—as well as in adults, the skull does not necessarily expand, and the head may not enlarge. Indeed, as in cretins, the head may even be smaller than natural. In these instances the brain substance must yield, and is reduced in thinness at times to a few millimeters only. In other cases the skull yields, its plates become thin, the fontanelles grow larger, and an appearance like that of congenital hydrocephalus may result.

Symptoms.—The symptoms of acute hydrocephalus are never distinctive, on account of the rapidity in the course of the disease which produces and obscures it.

Of chronic acquired hydrocephalus the most striking symptom is, as a rule, the *marked distortion in the size and shape of the head* already described. The weight of the head is sometimes so great that it inclines to fall to the side or backward or forward, and has to be supported by the hands of the patient. Other symptoms may, at times, be decidedly delayed, and the child may make some progress in studies. At times there is early *headache*. Signs of *mental imbecility* sooner or later make their appearance, manifested first, perhaps,—as in congenital hydrocephalus,—by absence of development, but progressing until the child lives an almost vegetative existence, having to be fed and cared for like an infant, even though several years old. There may be *convulsive contractions*, *tremors*, *ataxic gait*, *paresis*, and *paralysis;* in fact, all the symptoms which succeed on irritative and destructive lesions of the nervous system. The symptoms of tumor of the brain may be quite closely simulated, especially where the cranium does not enlarge with the growing distention of the ventricles. There may be choked discs, atrophy of the optic nerve, and total blindness. There may be prolonged attacks of drowsiness, or coma, with slow pulse, while sudden death is not uncommon during epileptiform convulsions or apoplexy.

Spontaneous evacuation of the fluid sometimes takes place by the nose, mouth, ear, or orbit.

Diagnosis.—This is commonly easy. It is only in cases where the cranium does not expand that the symptoms of brain tumor may lead to a diagnosis of the latter condition instead of hydrocephalus.

Prognosis.—This is usually unfavorable. Generally the child lives two to five years, though it may perish in a few months or live ten to fifteen years, or, as in a case of Bright's, to twenty-nine years. It has happened that spontaneous recovery has followed the evacuation of fluid above described. The absorption of small amounts of fluid is also possible.

Treatment.—This consists primarily in the treatment of the disease which is responsible for the hydrocephalus, if it can be discovered; secondly, in the treatment of the symptoms which may arise, and next, in attempts to cure the malady. Some favorable results have of late followed the removal of the fluid by puncture of the ventricles, although there has been failure in the majority of instances. Measures should be taken to make the removal gradual, if possible, thus attempting to imitate the spontaneous efforts of nature, which have occasionally been followed

by recovery. To this end the slow removal of the fluid—by puncture of the subarachnoid space between the 3d and 4th vertebræ—has been recommended and practised by Quincke. At this point, too, the spinal cord cannot be injured. It is more particularly in congenital hydrocephalus that operation is indicated.

If operation be deemed undesirable, attempts may be made to get rid of the fluid by diuretics and purgatives, although with little prospect of success. Iodide of potassium may be tried with the faint hope that the hydrocephalus is due to a syphilitic tumor which might thus be melted away. Blisters may also be applied.

GENERAL AND FUNCTIONAL DISEASES.

ACUTE DELIRIUM.

Synonym.—*Bell's Mania.*

Definition.—An acute and violent febrile delirium of unknown cause and undetermined lesion, running a course of two to three weeks, and usually fatal.

Historical.—The disease was first described by Luther Bell, of the McClean Asylum, in 1849.

Symptoms.—These set in suddenly and consist in violent, active *delirium*, in which the patient talks and moves incessantly, with a speech that is incoherent and unintelligible and movements which are aimless and irresistible or rhythmical as though with a purpose. This is kept up for hours and hours, notwithstanding the use of the most powerful anodynes, until the patient becomes exhausted,—the whole presenting a picture which is at once revolting and pitiable, as in the case of a poor woman, lately in the Philadelphia Hospital, whose image frequently recurs to me. At times sleep is obtained for an hour or two, but immediately on waking the active movements and delirium begin. The rhythmical movements may be like those of the salaam convulsions, up and down, as of one chopping wood or working a pump-handle. Throughout there is *high fever*, the temperature ranging from 102° to 104° F. (38.9° to 40° C.).

The tongue is dry, the pulse rapid and feeble, the skin, in like manner, dry and often covered with petechial spots or pustules and bullæ or bruises, the result of the violent acts of the patient. There seems, however, no pain or tenderness other than is due to these causes.

Morbid Anatomy.—As stated in the definition, there is nothing definite. There may be venous engorgement of the meningeal veins and of the cerebral cortex, with perivascular exudation and cellular infiltration of the lymph sheaths and perivascular spaces. There is often engorgement of the bases of the lungs, and deglutition pneumonia has been found.

Diagnosis.—At first the disease may be mistaken for any of the acute fevers which sometimes begin with violent delirium, especially

typhoid, but the course of the temperature and absence of other distinctive symptoms soon eliminates any doubt.

The same may be said of certain forms of puerperal mania, and more rarely pneumonia of the meningeal type and of cerebral meningitis itself. The *incessant* violence is, however, peculiar to Bell's mania.

Prognosis.—This is almost always fatal.

Treatment.—This must consist of measures to control the mania, of which hypodermic injections of morphine are almost alone efficient, and these often only feebly so. Chloroform or ether has sometimes to be employed, because of the dangerous doses of morphine which seem necessary. Blood-letting has apparently been of service in some cases, and there certainly is no contra-indication to it in the early stage of most cases, the patients being commonly very strong and vigorous. The cold bath may be employed, but is not an easy treatment to carry out, because of the difficulty in controlling the patient.

PARALYSIS AGITANS.

SYNONYMS.—*Chorea scelotyrbe sive festinans* (Sauvages); *Chorea procursiva* (Bernt); *Shaking Palsy; Parkinson's Disease.*

Definition.—A chronic nervous disease, characterized by muscular weakness, tremor or shaking in the extremities, muscular rigidity, and forward-bent gait.

Historical.—The disease was first fully described by Parkinson in 1817.

Etiology.—Shaking palsy is commonly a disease of the second half of life, but occasionally occurs between thirty and forty, and has been observed as early as the twentieth year. It is a little less frequent among women than men—11 to 14. Among the causes held responsible for it are exposure to cold and wet, fright, mental excitement, business worry, injury,—whether to nerves or other parts of the body,—alcoholism, sexual excesses, and the infectious diseases including malaria; while heredity is said to have a certain influence. The etiology of the disease is largely a matter of conjecture and inference. Perhaps exposure to cold is the best determined cause.

Morbid Anatomy.—This is unknown so far as essential lesions are concerned. Various lesions have been described, while the brain, spinal cord, and peripheral nerves of the most typical cases have been examined with negative results. Degenerative atrophy of the latter has been found on minute examination. As the phenomena are similar in kind, if not in degree, to those of senility it is held by Dubief that it has for its anatomical basis the lesions of cerebro-spinal senility, which differs from true senility only in its earlier onset. The question has justly been raised whether it is a disease of the nervous or muscular system.

Symptoms.—The disease is not a very rare one in this country, and it is not unusual to find a case in a morning's walk in the city, while the county almshouses almost always contain one or more,—easily recog-

nized by the characteristic shaking or tremulousness of the hand. Though commonly gradual in onset the symptoms may come on quite suddenly, and at first only after exertion. Indeed, there may even be a *prodrome* in the shape of neuralgic pains, paræsthesia, dizziness, and the like. The more sudden cases follow fright or trauma. The *tremor* is most marked in the fingers and hands, where it commonly begins, and whence it extends to the arms and lower extremities. The upper arm muscles are rarely involved. It most frequently passes from the right arm to the right leg, thence into the left arm, and thence into the left leg; or the course may be crossed, that is, from the right arm to the left leg. It may remain in one limb to the exclusion of the others. In the fingers the movement between the thumb and index finger is that of rolling pills. At the wrist that of pronation and supination. In the feet it is most marked at the ankle-joint; in the toes more than in the fingers. It affects the writing, making it trembling as in the aged, and ultimately it becomes impossible to write. The muscles of the head and face are last involved, sometimes not at all, and when present the motion is vertical and quite rhythmical, usually about five times in a second. At first the tremor ceases during sleep, but continues during the waking state even when the muscles are at rest, but ultimately it continues also even during sleep,—in fact, sleep is sometimes prevented thereby. It is increased in voluntary motion and by emotion. Should paralysis supervene, the motion ceases.

The rate of tremor varies greatly, being at first slower, and increases in rapidity as the disease advances. Roughly it may be put down at from three to five times per second. On the other hand, there may be intermissions of days and even weeks.

Muscular weakness is a less striking symptom, but may be estimated by the dynamometer, and increases with the duration of the disease and the intensity of the tremor. It is most striking at least in the extensor muscles, the *flexors* being disposed to *rigidity* and *spasm*, which early produce a slowness and stiffness of motion which is characteristic. It is this flexor spasm which brings the thumb and forefinger into the writing or pill-rolling position. At other times, hyperaction of the interossei muscles over that of the common extensors of the fingers results in the position so characteristic of arteritis deformans, that is, with the 1st phalanx bent, the 2d extended, and terminal phalanx also bent. Ultimately extension is impossible. Occasionally the opposite state of fixed extension exists.

The *attitude and gait* ultimately assumed by the subject of shaking palsy are also the result of rigidity, which sooner or later affects all the muscles. The head is bent forward, the back bowed, the arms held away from the body and flexed at the elbows, and the knees approximated so that they are often rubbed in walking; while the general appearance is that of a man in danger of falling forward. The position of the body due to flexion also gives rise to a "propulsive" gait, caused by carrying forward the center of gravity, so that when started the patient is apt to "get a going" and cannot stop until he comes up against some object. On the other hand, a push backward, bringing the centers of gravity behind the point of support, is apt to make the patient fall because he can-

not move back fast enough to save himself by "retropulsion." Charcot regards both these phenomena as "forced movements," but Strümpell prefers to explain them by simple physical laws as above described. Sometimes the characteristic position of the patient exists without the shaking, and Strümpell proposes for this the name "paralysis agitans sine agitatione."

The *facial expression* is also very strikingly altered. The face is indeed without expression, stiff and mask-like, giving rise to the name "Parkinson's mask." From the partially closed mouth there is often a dribbling of saliva. On the other hand, sometimes the mouth is kept closed and is found full of saliva,—a condition ascribed to delayed deglutition rather than increased secretion. The speech is slow and hesitating and monotonous, the voice piping and shrill. On the other hand, if the lips and tongue share in the tremor, the speech is stuttering as though the patient were in a hurry to speak, quite different from scanning speech of insular sclerosis.

The remaining nervous and organic functions are essentially normal. Sensation is not altered and the bowels and bladder are unaffected, as is also the temperature, although it is said that the surface temperature is sometimes raised. Charcot has noted an alteration of the temperature sense. There is sometimes a tendency to unnatural perspiration.

Diagnosis.—This is usually very easy and can be generally made at a glance. Multiple sclerosis resembles it in some respects. Both have tremor, but in multiple sclerosis this is shown more particularly when the patient attempts to do something, as to bring a glass of water to his lips or approximate his fingers. The speech is rhythmical, "scanning," instead of rapid and stuttering, as in shaking palsy; there is nystagmus, and the disease begins almost invariably in the lower extremities, while the attitude is not that of paralysis agitans. Chorea is characterized by movements, but these are irregular and more intermittent.

Prognosis.—A well-established case of paralysis agitans is not curable by medicines. On the other hand, the disease lasts indefinitely, the patient getting slowly worse, with perhaps the intermissions alluded to, until he dies of some intercurrent disease or from the effects of some accident growing out of his condition.

Treatment.—Under the circumstances this must, for the most part, be by tonics and general hygienic measures. As the disease advances the patient should be guarded against accident; and especially when in bed his position should be changed for him if he cannot change it himself, as is often the case.

Cases have improved under the use of the iodide of potassium and arsenic, and hyoscine has been especially recommended by Erb, hypodermically, in doses of $\frac{1}{200}$ to $\frac{1}{120}$ grain (0.003 to 0.005 gm.) of the muriate. Good results have also been reported from the use of atropine, of which $\frac{1}{1000}$ to $\frac{1}{600}$ grain (0.00066 to 0.0011 gm.) may be used subcutaneously or by the mouth.

Measures calculated to improve the general health are indicated, such as sea bathing, massage, electricity, fresh air, and out-door life.

Other Forms of Tremor.

Synonym.—*Ballismus.*

In addition to the tremor in paralysis agitans a similar tremor occurs under other circumstances, sometimes without assignable cause, when it is known as *simple* tremor, or it may be induced by fright or over-exertion. A *hereditary* tremor has been described by C. L. Dana. *Senile tremor* is the well-known form of tremor which comes on with advancing years, at times earlier than others, but usually not until after seventy years.

Toxic tremor is due to a number of toxic agents, among which tobacco and alcohol are the most frequent. Lead is another of these causes. Finally, *hysterical* tremor occurs as a part of hysterical phenomena in women. *Asthenic* tremor is due to simple weakness and is especially seen in exertion during convalescence from acute disease.

ACUTE CHOREA.

Synonyms.—*Chorea Minor; Mild Chorea; Sydenham's Chorea; St. Vitus's Dance.*

Definition.—A disease mostly of the young, characterized by irregular, involuntary muscular contractions, associated at times with psychical disturbance, often with rheumatism and endocarditis. The term chorea is derived from the Greek χορεια, dancing.

Historical.—The term *chorea sancti viti* was first applied, by Paracelsus (1493–1541), to an affection of a totally different nature, a sort of hysterical dancing mania which prevailed in epidemic form in the fourteenth, fifteenth, and sixteenth centuries in Germany and the Netherlands, for which the subjects sought relief by pilgrimages to certain shrines, among which was that of St. Vitus, in Zabern, whence the disease was called St. Vitus's dance. From other shrines it received other names, as St. John's and St. Anthony's dance. Chorea minor was first recognized by Sydenham in the sixteenth century, and was also called by him St. Vitus's dance, though a widely different affection from the St. Vitus's dance of Paracelsus.

Etiology.—The disease, though not confined to children, occurs far more frequently among them, notably from the time of the second dentition,—the sixth or seventh year,—to the fifteenth year. More than three-fourths of the entire number of cases occur during this period. Among adults it is relatively more frequent from the fifteenth to the twenty-fourth year. Occasionally it occurs in old age, when it is known as *chorea senilis.* Chorea is about twice as frequent in the female sex as in the male. Heredity has been always an acknowledged factor in its causation, but is probably less significant than was once supposed. It has even been claimed that the disease is sometimes congenital in the offspring of a choreic mother. It is more frequent in neurotic families. The so-called Huntingdon's chorea, which is hereditary, is not a true chorea. True chorea affects children of all social grades, but is more

common among the artisan and lower classes. It is rare in the negro. Wharton Sinkler, who has especially investigated this point, has seen but one case in a full-blooded negro, while William Osler, at the Johns Hopkins Hospital, out of 175 cases found five in the negro race. It is apparently unknown among Indians in their natural state.

As to temperament, it is well known that high-strung, excitable, nervous children, as contrasted with the dull and phlegmatic, are especially liable to the disease. It is especially in these that over-study is noted to have a predisposing effect. Psychical influences are undoubtedly potent; thus, fright causes a large number of cases, while grief causes many, and even joy some.

The season of the year appears to have an undoubted influence. Morris J. Lewis, whose studies have been most thorough in this direction, finds the fewest attacks occur in October and November, and the greatest number in March and April. Hermann Eichhorst, on the other hand, says the greatest number of cases occur in the autumn and winter months. The disease prevails more in towns than in the country.

Imitation, commonly regarded as an exciting cause, has been shown by modern studies to play a less important rôle than has been thought, many cases described as thus originating being really hysteria. Trauma precedes a certain number of cases. Reflex irritation, especially digestive disturbances, and intestinal worms were regarded as potent causes by the older observers; but here again Osler's studies have failed to find any causal relationship. The chorea of pregnant women has been referred to this category. The causal relation of eye-strain to chorea has been emphasized by Stevens, but is practically denied by George de Schweinitz, who concludes, from an examination of more than 100 cases, that, while ordinary chorea and many forms of facial spasm,—habit spasm, etc.,—are materially benefited by correcting refractive errors and anomalies of the ocular muscles, he does not believe there is any proof to show that eye-strain is of itself responsible for their origin, with perhaps the single exception of habit spasms affecting the orbicularis and immediate facial area.

The association of arthritis and chorea was observed by the earliest students of the subject, and was distinctly recognized in England as early as 1802, but the exact causal relation of the two diseases has, perhaps, not yet been made out. That they are frequently associated and that there is close connection between the two affections is admitted by English and French writers, but the Germans find the association much less frequent. Steiner, for example, found only four cases of rheumatism in 252 cases of chorea. English observers find from 20 to 70 per cent. of cases of associated joint affection, while in this country, where rheumatism is apparently less frequent in children, the range of percentage found by various observers is from 15.5 to 54 per cent. That the arthritis precedes the chorea in a large number of cases is generally conceded, the latter disease developing with the subsidence of the former, or not until convalescence has been well established. Hence, that the rheumatism is the cause of the chorea seemed at one time established, but recent views as to the probable infectiousness of rheumatism and the possible infec-

tiousness of chorea changed the conditions. As the nature of the virus of rheumatic fever is unknown, it may be that chorea is caused by a similar poison. This is further sustained by the fact that the infectious diseases play an acknowledged rôle in the etiology of chorea. Scarlet fever, diphtheria, measles, typhoid fever, gonorrhœa, secondary syphilis, puerperal fever, pyæmia, multiple suppurative polyarthritis, have all been followed by chorea, but, with the exception of acute rheumatic polyarthritis and some forms of septicæmia, the number of cases thus associated is not large. On the other hand, acute exanthemata developing in the course of chorea usually check the disease. Anæmia has been held to be a cause, and probably is a predisposing cause, though frequently also a result. The relation of hysteria to chorea is interesting from the close resemblance, at times, of the two conditions. It has already been said that the cases of so-called imitation-chorea are really hysteria, and, on the whole, the association of the conditions is rather co-incidental than causal. Poisons are acknowledged causes in a few instances. Carbon dioxide and iodoform are among those which appear to have caused acute attacks of chorea of short duration.

Morbid Anatomy.—There is no certainly ascertained morbid anatomy for chorea, and the lesions which have been found are the result of the complications or are incidental. The most constant of these associated lesions are endocarditis, in 85 per cent. of Osler's cases; pericarditis, 26 per cent.; combined heart lesions, 90.4 per cent.; pneumonia, 12 per cent.; less numerous were acute pleurisy, pyæmia, and phlebitis, also noted. As to the nervous system, in the cortex of which the symptomatology would lead us to expect the essential lesions, C. L. Dana has analysed the recorded autopsies, of which there were only 39 in which the state of the nervous system was accurately described. In 16 there was intense cerebral hyperæmia, peri-arterial exudation, erosions, softened spots, minute hemorrhages, and occasional emboli. The changes were most marked in the deeper parts of the motor tracts, particularly the lenticular nuclei and the thalami. These changes are essentially the same as those described by W. H. Dickinson, in 1876. Essentially similar were the lesions found in two of Osler's cases. In two reported by Bevan Lewis there was apoplexy, one cerebellar, one cerebral and extra ventricular. The so-called chorea corpuscles described by Ellischer are in no way characteristic. The same may be said of the swelling and turbidity of certain of the large pyramidal cells and the deeper layers of the cortex in the Rolandic region described by F. C. Turner. The changes in the ganglion cells of the spinal cord described by H. C. Wood in canine chorea have been found also by Triboulet, but he agrees with others who hold that canine chorea is a very different thing from human chorea.

Nature of Chorea.—This, it must be admitted, is as yet unknown. It has been intimated that the symptoms are of a kind which would naturally result from lesions in the motor cortical area. No constancy in such lesions is demonstrable. A cerebral seat for chorea is rendered likely by the existence of hemichoreas, the association of chorea with mild psychical derangements, and by the fact that choreiform movements are sometimes symptoms of undoubted brain lesions—*posthemiplegic*

hemichorea. The embolic theory which was suggested a few years ago by Senhouse Kirkes,—and supported by him,—Hughlings Jackson, Broadbent, Tuckwell, and others, was based upon the presence of foci of embolic softening found in a few instances in connection with endocarditis, but has gained no new supporters.

The theory which is at the present day naturally attracting most attention is the infectious theory, but the limits of a text-book do not permit its developmental consideration. Suffice it to say, that Pianese, of Naples, has apparently isolated a bacillus from the nervous system of a choreic patient, which he was able to cultivate successfully, and the cultures from which caused death in animals; also, that while the acuter forms present many, if not all, the conditions necessary to the conception of an infectious disease, the course of the milder forms, their etiology, notably their negative morbid anatomy, seem to demand that the disease be regarded for the present as a neurosis; that is, a disease of functional derangement without known anatomical basis.

Symptoms.—*Premonitory* symptoms, both motor and psychical, usually precede the onset of chorea. They include *restlessness* and inability to sit still, *altered disposition*, manifested by irritability and perversity. These symptoms, often misunderstood by parents, are sometimes the occasion of reproof and even severe punishment to the child,—a course which accelerates and aggravates the disease.

A close study of the symptoms permits of their division into three separate groups, determined chiefly by the severity of the symptoms:—

(1) A *mild* form, including the majority of cases where the affection of the muscles is slight, the speech scarcely involved, the general health slightly disturbed.

(2) The *severe*, in which the choreic movements are general, power of speech lost, and the patient unable to get about and help himself.

(3) The *maniacal*, or *chorea insaniens*, characterized by intense cerebral excitement.

It is, however, unnecessary to separate the symptoms of each variety.

The *motor phenomena* are those first observed. They consist in peculiar jerky movements which begin most frequently in the upper extremities, especially the right hand, rarely in the legs. They may even be general from the first, though the earliest symptoms often escape notice. *Speech* is affected sooner or later in one-fourth of the cases. The extent varies greatly from slight hesitancy to incoherency; the difficulty being in the muscles of articulation rather than phonation. As a rule, the movements cease during sleep, though they sometimes persist even then. It is not generally believed that the movements extend to the muscles of organic life, though associated irregular and rapid action of the heart has been ascribed to choreic spasm of the papillary muscles. As the disease continues *muscular weakness* becomes manifest in a general want of strength rather than paralysis, though the weakness may be distributed hemiplegically or even monoplegically. It may even precede the jerking movements.

Sensory symptoms are less conspicuous than the motor. Pain is

rare, though its presence has been enough characteristic in some cases to obtain for them the name "Painful Chorea" from S. Weir Mitchell. Painful points over the sites of emergence of spinal nerves have been pointed out, though they must be rare. Numbness, tingling, and pricking sensations are occasionally met and may be a part of the phenomena of multiple neuritis sometimes present. Headache, sometimes very severe and paroxysmal, may occur, while epileptiform seizures are also a rare symptom. The *reflexes* are variously affected, the knee-jerk being normal in about half the cases, in the remainder increased or absent. Trophic lesions are almost unknown. *Mental symptoms*, in the majority of cases, are not very conspicuous, though there are in some severe cases extreme manifestations, including melancholia, hallucinations, and even mania, which have their climax in chorea insaniens.

Most important are the *symptoms of cardiac disease*, in regard to which William Osler makes the startling statement "There is no disease in which endocarditis is so constantly found post-mortem as chorea. It is exceptional to find the heart healthy." The symptoms which are therefore to be always carefully sought include a *systolic apex murmur*, palpitation, and irregular heart action, although the child rarely complains of the latter or of pain about the heart. Very rarely the *pulse* may be slow in the feeble state which follows chorea. Organic *murmurs at the base* are very much more uncommon, most of the murmurs here being functional. They are heard with greatest intensity in the area of the pulmonary artery, but are audible sometimes in the aortic area as well. In a large proportion of all cases in which a murmur is heard at the base or along the left margin of the sternum in the 2d, 3d, and 4th interspaces it is functional, but a soft systolic murmur in this area with systolic pulsation in the cervical veins may be caused at the tricuspid orifice.

On the other hand, *endocarditis* sometimes occurs *without symptoms* or physical signs, while the disappearance of physical signs does not prove that endocarditis was not present. A *pre-systolic murmur* is also at times present, indicating mitral stenosis—in 19 per cent. of Osler's cases. On the other hand, the comparative rarity of simple aortic valve involvement is conspicuous, this being more uncommon than combined aortic and mitral disease, or even combined mitral and tricuspid. The tricuspid valves may be alone attacked.

A to-and-fro murmur, indicating *pericarditis*, may be present say in from eight to 25 per cent., and in more than half of these it is associated with endocarditis. It is to be remembered that both forms of organic heart disease, and especially endocarditis, may occur in chorea without rheumatism,—*e. g.*, in 66 per cent. of Osler's cases,—also that such endocarditis may lay the foundation of permanent organic disease.

Occasional *skin affections* make their appearance in chorea, the larger proportion being due to the prolonged administration of arsenic, so much used in the treatment of this disease. The forms for which the arsenic treatment is more or less responsible are erythematous and papillary eruptions, herpes, and the pigmentation frequently resulting from the prolonged administration of this drug. Eruptions also occur independent of arsenic administration. They are usually purpuric and associated with

arthritis, similar in form to the purpura so often associated with rheumatism, and include some of the forms of multiple erythema,—as erythema nodosum, purpuric urticaria, or simple purpura.

Fever is a rare symptom in chorea, except as the result of complications, of which arthritis is the most common, but endocarditis and pericarditis may also cause fever. The rare instances are cases of *chorea insaniens*, in which the temperature may rise to 105° F. (40.5° C.).

The *duration* of the disease may be put down from eight to ten weeks for the ordinary cases, from three to six months for the very severe ones. *Remissions* occur, and *relapses* as well, pointed out by Sydenham. A disposition to vernal recurrence has been noted.

Diagnosis.—This is usually easy. Simple tremor, athetosis, paralysis agitans, as well as alcoholic, senile, saturnine, and mercurial tremor are not likely to be confounded with the movements of chorea. The symptomatic choreiform movements due to cortical irritation by meningitis, tubercle, hemorrhage, softening, tumor, or parasites, are attended by other symptoms which mark them as something different from chorea. In multiple and diffuse cerebral sclerosis the so-called *chorea spastica* movements may be very similar, but the early onset,—usually in infancy,—impaired intelligence, increased reflexes, the rigidity, and chronic course of the disease characterize it. Friedreich's ataxia was formerly regarded as chorea, but modern studies easily separate it by the slowness and incoördination of its movements, the scanning speech, talipes, nystagmus, and family distribution.

Prognosis.—Except in *chorea insaniens*, which is always fatal, recovery is the rule in from eight to ten weeks. Chorea of the pregnant woman is more serious than the chorea of children.

Treatment.—All cases should be carefully examined for causes of reflected irritation which should be removed, then rest is essential. It is not necessary that the very mildest cases be put to bed, but they should be withdrawn from school and guarded from excitement and the curious gaze of friends and strangers, for the movements almost invariably increase when the patient is under observation. More serious cases should be put to bed—a more thorough exclusion as well as rest thus secured. Not only is recovery thus facilitated but a diminished liability to heart complication is also attained.

Of drugs, arsenic and iron hold the first place. The former is given in slowly ascending doses of Fowler's solution until its physiological effect is produced, after which the dose should be gradually diminished. Some one of the preparations of iron should be given continuously in moderate doses. The bromides are also indicated, especially where there is restlessness and want of sleep, when chloral may also be added, and in severe cases may be given continuously. Opiates should, however, never be employed. An old remedy in this country is black snake-root or cimicifuga racemosa, first recommended by the late Dr. Hiram Corson, who wrote me he had used it for fifty years without a failure. I have sometimes used it in the shape of the infusion in mild cases, with apparently satisfactory results, in doses of one and two fluid ounces (30 to 60 c. c.). Modern remedies are antipyrin and physostigma. The former is given to adults

in doses of seven to 15 grains (0.5 to one gm.), much reduced for children. Physostigma has been given in doses of $\frac{1}{70}$ to $\frac{1}{35}$ grain (0.0009 to 0.0018 gm.) hypodermically. Hyoscyamine in doses of $\frac{1}{100}$ grain (0.00065 gm.), three times a day, has apparently been followed by good results. The oxide of zinc, valerianate of zinc, nitrate of silver, and sulphate of copper, formerly much recommended, have fallen into disuse. In consequence of the close relations between chorea with its attending arthritis and rheumatic arthritis it is reasonable to expect that the salicylates might be useful, but such expectation has not, as yet, been realized.

CHOREIFORM AFFECTIONS.

There remain to be considered several forms of convulsive contraction included under the term "habit spasm" or "habit chorea" or "tic." The term *tic*, as usually understood, means *facial spasm*. It has, however, been extended by the French school (whose lead in these affections seems at present acknowledged), to include "an habitual, conscious, convulsive movement, resulting in the contraction of one or more muscles of the body, reproducing, most frequently in an abrupt manner, some reflex or automatic action of common life" (Guinon). It is characteristic of these motions that they are more or less under the control of the will, in which respect they differ from the contractions of chorea minor.

I. Simple Tic.

Synonyms.—*Habit Spasm; Habit Chorea.*

Simple tic may be localized or general.

Localized tic begins usually in young persons, most frequently in girls from seven to fourteen years of age, and may persist through life. The spasm is confined to a single muscle, a group of muscles, or a group of associated muscles, most frequently the muscles of expression. The mild forms are looked upon as simply peculiarities of the individual; but the more severe forms, in which nearly all the muscles of the face are affected and even the depressors of the jaw and the tongue muscles are often thrown into action while speaking, are manifestly pathological. It differs from the *idiopathic facial* spasm of adults in that the latter is rarely seen until after the fortieth year and is slower than is the habit spasm of the facial muscles. It is possible for the simplest forms to be a perpetuated childish trick; such may be a blinking of the eye or the act of sniffling. In other simple forms there is a drawing aside of the mouth or a jerking of the head to one side or a simple shaking of the head, while the eye is winked at the same time; or there may be a shrugging of one shoulder. More rarely the contraction occurs in the legs, as in the very characteristic "spring-halt" tic, in which at times the leg is suddenly drawn up. Localized tic is usually transient, gradually disappearing after a few months.

Generalized Tic, Electric Chorea (Henoch).—In this there is sudden electric-like spasm of the muscles of the trunk and limbs, but especially

of the neck and shoulders, causing an instantaneous start, which affects the patient for an instant only, when it passes off and leaves him quiet and motionless. The contraction is like that produced by a galvanic shock. It may be associated with facial spasm. It occurs especially in children, but also in adults, particularly women, and may persist for years.

Paramyoclonus Multiplex; Myoclonia.—This term was applied by Friedreich to a disease first observed by him, in which there are clonic convulsions in symmetrical muscle groups in the arms and legs without loss of consciousness. It is usually in males, and follows emotional disturbances like fright or straining. In addition, there is a considerable increase in the tendon reflexes. In order that a case may be one of true paramyoclonus it is necessary that the contractions in the single muscles should be sudden, lightning-like. The muscles affected are commonly those of the trunk and extremities. The contractions are usually bilateral and vary from 50 to 150 in a minute. There are no sensory symptoms. Between the attacks there may be tremors. These cases are allied, on the one hand, to the electric chorea just described, and, on the other, to the different forms of convulsive tic, clonic facial cramp, clonic accessory cramp. Some cases of so-called paramyoclonus are really cases of hysteria.

Dubini's Disease.—The term electric chorea is also applied to an acute infectious disease occurring in Lombardy and known as Dubini's disease, in which there are also sudden contractions, first usually in the arm, but passing thence into all the extremities, followed in several weeks or months by paralysis and muscular atrophy, occasionally also by epileptiform convulsions and fever. No morbid anatomy has been determined.

II. Tic with Explosive Utterances, Caprolalia, Echolalia, Fixed Ideas, Etc.

Synonyms.—*Maladie de la tic convulsif; Gilles de Tourette's Disease.*

Definition.—In addition to motor spasm this form of tic is characterized by explosive utterances of certain words and sounds, such as "fire," "murder," "hah," "bow-wow;" or profane words, such as "God damn," or "Jesus Christ;" or filthy and obscene words, when it is known as caprolalia. There may also be mimicry of words, when it is called *echolalia;* or mimicry of action, *echokinesis;* or the patient may be possessed of a fixed idea of the variety known as *ithmonomania*, *délire du toucher*, *onomatomania*, and *folie pourquoi*. In the first almost every action is preceded by performing a certain number of acts: as in a patient of Osler's who before she went to bed had to tap her heel upon the bedstead a given number of times; before drinking a tumbler of water, to rotate the glass nine or ten times, and the same thing when setting it down; before opening a door a certain number of knocks had to be given, and the greatest difficulty was experienced in getting her to brush her hair as it took so long to count before she began. In the *délire du toucher* there is an irresistible disposition to touch objects; in

onomatomania to repeat over and over again names which arise, and in the *folie pourquoi* to demand a reason for every one of the simplest acts. In other instances the patient imagines that some one is talking to her. All these are in addition to the convulsive acts.

The involuntary movements themselves vary greatly, from trifling tic of any one or more of the face muscles to contractions involving all the muscles of the body. This condition, which is neither chorea nor habit spasm, is at times taken for both. It is commonly easy of recognition, and although of uncertain prognosis recoveries take place.

III. Complex Coördinated Tic.

Definition.—This includes a number of forms of habit movement differing from ordinary tic in the more complex nature of the actions performed. It includes tricks and habits, such as those of one who in writing stops at every few words and looks intently at his finger-tips; the "head nodding" of children (not to be confounded with the *epilepsia nutans* of children), "thumb sucking," "rocking in bed," and similar actions. Of the same nature is the so-called "head-banging," in which the child, asleep or awake, while in bed, will turn over and bang the head violently into the pillow, repeating this act five or six times or for two or three hours at a time. Or the child may strike the head repeatedly with the fist—*krouomania*. Or it may rotate the head violently from side to side, balancing, or gyrating the body with great rapidity. The practice is sometimes communicated from one child to another. These movements are met with especially in feeble-minded children, in whom it may be accompanied by nystagmus, and is sometimes the result of injury after birth. Where these phenomena do not occur in the feeble-minded or after injury early in life the prognosis is said by Gee and Haden, who have especially studied the subject, to be favorable.

IV. Spasms of the Muscles of Respiration and Deglutition.

Definition.—The spasm affects the muscles of respiration and phonation, the muscular contraction being accompanied by more or less noise, as a "sniffle" or "hiccough" during inspiration, or some noisy or explosive sound during expiration. Such spasms are sometimes part of a hysterical state. Among those described as thus occurring is a sort of rumbling which comes from low down in the abdomen, passes up the stomach, and out of the mouth as an explosive loud noise—something like belching, but louder. In another instance there was a peculiar clucking noise in the throat accompanying motions essentially those of swallowing, which disappeared only during sleep. Again, there may be a loud inspiratory cry preceded by three or four deep inspirations and followed by a deep, hoarse, expiratory sound.

V. Chronic Progressive Chorea.

Synonyms.—*Huntingdon's Chorea; Chronic Hereditary Chorea.*

Definition.—A disease of adult life, commonly hereditary, characterized by irregular movements, deranged speech, and ultimate dementia gradually developing.

Historical.—The affection was first described by C. O. Waters, of Franklin, New York, in Dunglison's "Practice of Medicine," in 1842; again in the American Medical Times, 1863, by Irving W. Lyon; fully in 1872, by George Huntingdon, of Ohio, in the Medical and Surgical Reporter, in whose paper these three marked peculiarities were presented and dilated upon :—

(1) Its hereditary nature.

(2) A tendency to insanity and suicide.

(3) Its manifesting itself as a grave disease only in adult life.

Subsequent to Huntingdon's description little is found in literature until 1884, when C. A. Ewald reported two cases in Germany, and in 1885, when Clarence King, in this country, reported another family. Then followed numerous reports; and finally, in Paris, in 1889, the monograph of Huet, and another by Osler, in 1894, in which the history of two families is detailed. Numerous cases were reported between 1889 and 1894.

Etiology.—Its frequent hereditary origin has been mentioned. Indeed, heredity is one of its most striking features, 25 per cent. of certain families having been victims, and even more than 50 per cent. of the adults in families. It is especially when both parents are affected and seriously that one or more of the offspring almost invariably have the disease if they live to adult age. The two sexes are about equally affected, though in some families more males have it. It is further characteristic that if a generation is skipped, the disease never manifests itself again in that family, and that it rarely presents itself before thirty years of age. Huet has, however, collected seven cases of earlier onset. It is said, also, that it is not invariably hereditary, being sometimes due to emotional causes. In all the families in which this choreic tendency has been found the nervous temperament prevails.

Morbid Anatomy.—This is somewhat more definite than that of chorea minor. At least there has been found at necropsy quite frequently a condition of pachymeningitis and hæmatoma of the dura mater with atrophy of the cortex, and less frequently a disseminated encephalitis evidenced by subcortical foci of round cells. Nothing has, however, been found which can in any way be regarded as peculiar or as accounting for the disease occurring at a certain age or for its affecting certain individuals, though the lesions do explain the motor phenomena. It should be stated that Charcot and his pupil, Huet, do not separate this chronic progressive chorea from chorea minor, but all others do.

Symptoms.—The *onset is gradual* in hereditary cases, although it may be sudden in cases otherwise arising. As in chorea minor, *motor symptoms* are the first to appear; first usually in an unsteadiness of the gait or slightly irregular movements of the hands. Occasionally only,

the *mental symptoms* are the first to appear, not usually manifesting themselves until the motor are well established. Motor symptoms include also *spasm* of the muscles of the face. The movements differ from those of chorea minor in being *slower* and in *absence of coördination*, strikingly manifested in walking. The station may be good, except a slight swaying of the trunk, but an attempt to walk is followed by an unsteadiness characterized by marked lateral deviation from the straight line, by swaying of the body, and sometimes by precipitate falling movement from which the patient may, however, recall himself—in brief, a typical drunkard's gait. This unsteadiness ultimately makes locomotion impossible, and the patient takes to his bed. Yet before this stage is reached, although ataxic, he may be able to walk long distances. While at rest the movements cease altogether. They are aggravated by emotion and excitement, while in the beginning they may to a degree be influenced by the will. Thus a patient said to me lately, "If I put my mind to it I can stop it."

Speech is affected in most instances, being at first slow and hesitating and interrupted by interjections; later, it is indistinct. The handwriting is likewise involved, the letters being irregular and badly formed, running into each other and off the line, and ultimately writing becomes impossible. Sensation and the special senses remain intact, as does the muscular force, until the disease is advanced. The reflexes are usually increased.

The *tendency to insanity and suicide* has been referred to as an acknowledged symptom. Beginning as a simple irritability or moodiness with depression, it passes slowly over into feeble-mindedness. The suicidal impulse is sometimes carried out.

Diagnosis.—This is easy in the hereditary cases only. Friedreich's ataxia resembles it in heredity and other symptoms, but begins earlier. The absence of spasm is characteristic of Friedreich's ataxia, its presence is characteristic of progressive chorea. Idiopathic double athetosis also occurs in elderly persons, but in it the movements are associated with rigidity and the gait is also spastic, while neither rigidity nor spastic gait play any part in progressive chorea.

Prognosis is ultimately fatal. The progress of the disease is progressively and irresistibly from bad to worse.

Treatment is of no avail.

VI. Chorea Major.

Synonyms.—*Pandemic Chorea; Automatic Chorea; St. Vitus's Dance; Rhythmical or Hysterical Chorea; Latah; Myriachit; Jumpers; Jerkers; Holy Rollers.*

I prefer to include under this heading all the different varieties of saltatorial spasm of which the historical St. Vitus's Dance of Paracelsus, prevalent in the fourteenth, fifteenth, and sixteenth centuries, is the most familiar illustration.

All are varieties of tic, described by Bamberger, in which strong contractions take place in the leg muscles when the patient attempts to

stand, causing a jumping or springing action. All are endemic neuroses, illustrated by the "jumping Frenchmen" of Maine and Canada, the subjects of which are liable, on any sudden emotion, to jump violently and utter a loud cry or sound and obey any command or imitate any action without regard to its nature. The jumping prevails in certain families. Similar were "the jerkers," who appeared during the religious revivals in Kentucky in the early part of the present century; and the "holy rollers," in New Hampshire and Vermont. The disease known as latah among the Malays, and myriachit in Russia are similar. In the true St. Vitus's dance, chorea major, or *chorea Germanorum*, the paroxysm arises spontaneously; so also in the salaam convulsions of children, in which the muscles of the abdomen are affected, and in which there is a bowing forward of the head and body as many as a hundred times or more. The paroxysms may occur several times a day, lasting from a few seconds to as many minutes. In the others, as the American jumpers, etc., it is in response to some external impression. During the paroxysm the affected person sings, dances, jumps from the ground, rolls from side to side, hammers with his hands, stamps with his feet, or whirls madly around until he falls exhausted to the ground.

VII. Pre- and Post-Hemiplegic Chorea.

Definition.—By this are meant choreiform movements which are the result of cerebral disease, most frequently hemorrhage. They may immediately precede or follow the stroke. The pre-hemiplegic form is rarer and more serious in significance. The movements vary greatly, and the milder degrees can be recognized only on close examination. In this form the symptoms precede, usually by a few days, the apoplectic stroke, and cease as soon as paralysis appears.

Post-hemiplegic chorea, on the other hand, ordinarily appears in the limbs previously paralyzed, at the time when they again begin to be capable of motion. It is generally sudden, and either continues throughout life or disappears gradually. Often it is associated with contractures. Not infrequently the affected side of the body is anæsthetic, and even the organs of special sense may take part in the hemianæsthesia.

The lesion causing these symptoms is regarded as cerebral, and in that portion of the cerebrum within the internal capsule where the fibres of the pyramidal tract pass between the lenticular nucleus and the optic thalamus and the occipital lobe. Sometimes, however, similar phenomena are associated with disease elsewhere, as in the pons or even the spinal cord; but under any circumstances it would seem to be necessary that there should be irritation of the pyramidal tracts somewhere in their course.

EPILEPSY.

SYNONYMS.—*Morbus caducus sive sacer; Morbus divinus; Falling Fits.*

Definition.—Epilepsy is a chronic paroxysmal disease, characterized by sudden loss of consciousness, and in its typical form by violent general convulsions (grand mal); but both unconsciousness and convulsion may be so fleeting as to be barely recognized (petit mal); while convulsions may be localized and without loss of consciousness (Jacksonian or cortical epilepsy); finally, seizures may be substituted by conditions of uncontrollable violence or somnambulistic acts (psychical epilepsy).

Epilepsy is, strictly speaking, a syndrome or group of symptoms of which the morbid basis is not always the same. Formerly it was considered essential to the idea of epilepsy that the convulsions should be neither toxic, reflex, traumatic, the result of previous brain disease, nor heart failure. At the present day toxic convulsions, which are essentially covered in actual practice by uræmic convulsions, are not regarded as epileptic. Nor are pure reflex convulsions which are due to such causes as teething, constipation, worms, and other forms of peripheral irritation. On the other hand, certain convulsions due to cortical brain lesions, which will be further considered, are acknowledged to be epileptic in addition to those of which the anatomical basis is still unknown.

Etiology.—From one to six persons out of every 1000 have epilepsy. The tendency of modern studies is to diminish the importance of heredity, formerly so conspicuous as a supposed cause of epilepsy. Gowers's statistics, which may still be regarded as representing the older pathology, drawn largely from his own practice, ascribe to heredity a percentage of 35, while the range in the older statistics is from nine to 40. Osler's observations, on the other hand, on cases at the Infirmary for Nervous Diseases in Philadelphia, and in the Institution for Feeble Minded Children at Elwyn, Pa., give the percentage in the two institutions as a little over one per cent., and in five cases out of 435 in which the epileptics were children of epileptic parents it was traceable to the mother in every instance. The comparative unimportance of heredity as a cause is upheld by the modern French school, notably by Marie. On the other hand, the disease is of frequent occurrence in neurotic families, including those subject to insanity, hysteria, and neuralgia. So, too, vices of constitution and habit in parents, especially alcohol and syphilis, are acknowledged causes. The intermarriage of relations is also an element. More certainly responsible is local disease of the brain cortex, including tumors and traumatic disease such as are produced by fractures, and the conditions described as causing the cerebral palsies in children, post-hemiplegic epilepsy being one of these.

All of these are causes which may be both essential and exciting. Among the more purely exciting causes are fright, irritation by worms in the intestinal tract, dentition, constipation, and the like, all of which may provoke attacks in an epileptic. Some would regard the reflex epileptiform attacks excited by these causes as true epilepsy, and call it reflex epilepsy. But at present these cases should not be called epileptic, since

they do not recur after the exciting cause is removed. Such were two striking cases in my own experience, one of which disappeared entirely after the removal of a tape-worm in a girl of eighteen, and another after the cure of constipation in a young man of twenty. These are very different from others in which attacks are brought on by like exciting causes, but occur also independently of these causes.

True exciting causes are infectious diseases, alcoholism, and syphilis. The influence of infectious diseases is thus shown: Given an epileptic who is subject to seizures once a month, who acquires typhoid fever, the prodromal symptoms are almost always sure to include frequently recurring epileptic seizures. Masturbation is included among the true causes, but is probably only an exciting cause. Ocular and aural irritations are exciting causes. Cardiac epilepsy is a variety in which there is disturbance of the heart's action, either palpitation or slowing, prior to attacks; but such derangements are co-symptoms rather than causes, or they may be a mode of manifestation of the aura to be presently described.

Epilepsy is preëminently a disease of childhood and youth, and after twenty it is most unlikely to arise. Most cases begin between the ages of ten to fifteen. Yet it is said that idiopathic epilepsy may occur after sixty. It seems to be slightly more frequent in boys than girls, although all statistics do not point this way, whence it may be concluded that the numbers in each sex are nearly equal.

Morbid Anatomy.—The cortical lesions described as causing the cerebral palsies of children and some resulting from trauma are found in connection with most cases of Jacksonian or local epilepsy. Tumors, especially those involving the motor layer of the cortex, are among these causes; so are localized syphilis, pachymeningitis, and tyroma or tubercular tumor, and sometimes tubercular meningitis, pointed out by J. Hendrie Lloyd. Lloyd, would, however, exclude the gross deformities, such as parencephalon; and diffuse processes, such as lobar sclerosis, which manifest themselves by idiocy and arrest of development and are not infrequently provocative of epileptic seizures.

Sclerosis of different parts of the brain and medulla is also found in cases of epilepsy, especially in the hippocampus major, more rarely in the olivary body, these being probably conspicuous local seats of a more diffuse lesion. Similar sclerosis is sometimes found in the cerebellum. A nuclear degeneration and vacuolation of the angular cells of the second layer of the cortex has been claimed by Bevan Lewis as a distinctive lesion of epilepsy. Many cases of so-called idiopathic epilepsy are still without a demonstrable morbid anatomy.

Mechanism of the Convulsion.—The epileptic seizure itself is regarded in the light of our present knowledge as an explosion or discharge of nerve force the seat of discharge, at least in the severe seizures, being the large motor cells in the deeper layers of the cortex, the function of which is to store up and discharge nerve force. They are under the control of the angular sensory cells in the second layer which have inhibitory power. This, under the influence of disease, is lost, and the motor cells explode periodically. The same mechanism exists in sensory and psychical epilepsy.

Symptoms.—These vary in the four varieties known as grand mal, petit mal, Jacksonian, and psychical epilepsy.

(1) *Grand Mal.*—In a large number of cases the epileptic attack is preceded by what is known as the *aura*, a peculiar sensation, which differs greatly in different individuals. Most frequently, perhaps, it is like what the word literally means, a breath of air, which starts from a particular part of the body, as the extremities or a single finger or toe or a part of the surface of the body such as the neigborhood of the stomach or the heart. At other times the aura is a simple epigastric sensation, a sense of discomfort or uneasiness emanating from the stomach or the feeling of a ball arising therefrom. It may be a flash of light, which may be of different colors; an object, as a face or faces, and even a coffin—as in one of Allen Starr's cases. Auditory auræ are manifested through the sense of hearing, and may be subjective sounds of any kind, including musical tones, or even voices. Gustatory and olfactory auræ include subjective tastes and smells, mostly of an unpleasant character. Auræ are represented also by tingling, numbness, or simple flushing, or chilliness anywhere in the body. "Intellectual auræ," so called by Hughlings Jackson, are certain mental conditions, such as a "dreamy state," and the consciousness of a certain algebraic formula, which always presented itself to a patient of Allen Starr's.

In other cases there is more prolonged *prodrome*. For several hours or a day the patient may be the subject of sensations. He may feel generally miserable, dispirited, timid, irritable, or dizzy. Or he may be pale or quiet, and patiently waiting for the dreaded event, known to him rather by its consequences than its phenomena of which he is unconscious. There is pathetic sadness often in this patient expectation. The aura is by no means always present, indeed, perhaps in the majority of cases of epilepsy, there occurs no warning of the attack. The aura may be substituted by certain movements, such as running rapidly for a few minutes either forward or in a circle, the so-called epilepsia procursiva, or the patient may stand on his toes and rotate with great rapidity.

Following the aura or independent of it occurs the *convulsion* or "*fit*," of which the initial event is often the *epileptic cry*. This is succeeded by the *fall*, which may be sudden, as if the patient were shot, he often injuring himself in the fall. Following this, the phenomena of the fit may be quite sharply divided into *three stages*, that of tonic spasm, of clonic spasm, and of coma.

(1) The Tonic Spasm.—In this the head is drawn back or to the right and the jaws fixed; the arms are flexed at the elbow, the hand at the wrist, and the fingers clinched into the palm, while the legs and feet are extended. The muscles of the chest are involved and respiration is suspended and the face becomes dusky, livid, and swollen, contrasting with the initial pallor. The muscles of the two sides are not equally affected, so that the neck is twisted and the spine curved. This stage lasts but a few seconds and is succeeded by clonic spasm.

(2) The Clonic Spasm.—Now the muscular contractions become intermittent. At first tremulous and vibratory, they soon become strong and general, until the arms and legs are thrown about in the most vio-

lent manner, sometimes so violently as to produce dislocation, usually of the shoulder. The muscles of the face are also involved in distorting contractions, while the eyes roll and the lids open and close. The jaw muscles contract violently and the tongue is apt to be caught and bitten. A frothy saliva, often blood-stained, escapes, and the patient is said to "froth at the mouth." There may be involuntary discharge of fæces and urine. The lividity supervening in the first stage diminishes somewhat during this stage. The temperature rises ½° to 1° F. (0.28° to 0.55° C.). Very soon the contractions become less violent, finally abate, and this stage terminates, in one or two minutes, in the stage of coma.

(3) Coma.—In this the limbs are relaxed and there is profound unconsciousness, but the breathing is noisy and stertorous. The face remains congested, but is no longer cyanotic. He may, after awhile, be aroused, but if left alone commonly sleeps several hours, awaking after a time in a remarkably natural state, feeling bruised and aching, but otherwise quite himself; or there may be some mental confusion and even headache.

These are the phenomena of the attack in the vast majority of cases of grand mal. There may not be another for several days or a month or more. In severe cases, on the other hand, there may be daily recurrence, though not until the disease has lasted for several years. In a few instances the attacks may follow each other in rapid succession without a return of consciousness, lasting from twelve hours to a day or more, producing the *status epilepticus*, in the course of which the patient may die from exhaustion. In this state there is often decided fever.

After the attack the reflexes may be increased and ankle clonus may be obtained; at other times the reflexes are absent. The urine is also often increased and a small amount of albumin is quite common after the fit.

(2) *Petit Mal.*—The symptoms in minor attacks vary somewhat, but commonly the patient stops in the midst of what he may be doing, the eyes become staring and fixed, the pupils dilated, the countenance pale, there may be some twitching of the facial muscles or the limbs, and consciousness is lost, but there is no convulsion. Anything that is in the hand may be dropped, but in a minute or two consciousness returns and the patient resumes what he has been doing as though nothing had happened. Here, too, though rarely, there may be auræ of various kinds and even an epileptic cry; also forced movements—procursive epilepsy. There may be dizziness without unconsciousness and the patient may fall. As the disease continues the attacks of petit mal generally become grand mal, or the two forms of attack may alternate.

(3) *Jacksonian or Partial or Cortical Epilepsy.*—In this, consciousness is retained, while convulsions occur, though circumscribed to a single group of muscles or to a single limb. It is always symptomatic of some focal lesion in the cortical motor area, which may be a tumor, an injury or inflammatory process in the membranes or brain substance, softening, hemorrhage, abscess, or sclerosis. It is especially apt to be a sign of a growing tumor. Hence, it is also called symptomatic epilepsy.

Previous to the twitching there may be a numbness or tingling in the part to be involved, which has been called the "signal symptom" by Seguin, because it ushers in the attack. It may remain during the attack, and is of value in determining the seat of the lesion, and therefore the place for operation. Its seat is usually the same in the same patient in all the attacks.

The spasm or convulsion begins uniformly in one part,—it may be the face, the thumb, the toes,—thence slowly invades the entire limb. It continues sometimes for three or four minutes or longer. The movement is tonic and clonic, extending from the part in which it begins to other parts in a definite order of extension. Thus, if it begins in a part of the face it extends thence to the whole face, then to the shoulder, arm, forearm, and hand, and possibly the leg from the trunk down to the toes. Or it may start in the fingers and go in the opposite direction. Jacksonian epilepsy also occurs in uræmia and progressive paralysis of the insane, and it has already been spoken of as following the hemiplegia of children. After the convulsion, the parts convulsed and especially that in which the spasm begins are found to be partially paralyzed and awkward in movement, and quite often the numbness continues for some time, with a moderate degree of tactile or thermal anæsthesia.

(4) *Psychical Epilepsy.*—This occurs either as a later symptom following the more common forms of grand mal and especially petit mal, or as an independent state or as what is known as a "psychical epileptic equivalent," in which the usual seizure is substituted by a somnambulistic state in which the patient performs various acts, sometimes of great complexity, including driving, walking, and the like, of which he is totally oblivious after he passes into the natural condition. Some striking instances of psychical epileptic equivalent are related by Allen Starr in his book on "Familiar Forms of Nervous Disease." Other manifestations of psychical epilepsy are represented by violent maniacal excitement and uncontrollable violence, in which criminal acts, including even homicide, are committed.

Relative Frequency and Time of Attacks.—The major form of attack is the most frequent, after this mixed forms of major and minor, and then the minor and Jacksonian; the most infrequent are the psychical forms.

Two-thirds of the attacks occur between 8 A. M. and 8 P. M.; many attacks occur early in the morning soon after awaking, some between 3 and 5 A. M., and others in the night at unknown hours—nocturnal epilepsy. In true epilepsy the patient generally feels perfectly well between the attacks, indeed he not infrequently feels better for a time after the spell.

Diagnosis.—The epileptic fit is of itself in no way characteristic of the disease. The uræmic convulsion is identical, as is also the reflex convulsion with teething and the like. Even hysterical convulsion closely resembles it, but there are points in which it differs. Something more, therefore, than the convulsion is necessary to prove the disease. The aura is distinctive, and when present and repeated is conclusive. Scarcely less so is the epileptic "cry," although it is less constant

than the aura. The relaxation of the sphincters belongs rather to the epileptic fit, while the bitten tongue, the dilated pupil, and sudden unconsciousness belong to uræmia as well; and it is from uræmia that it is most important to distinguish epilepsy. The occurrence in the midst of apparent health of a convulsion with the features described, followed by prompt recovery, without albuminuria or casts, can hardly be anything but epilepsy. At other times, when other signs of Bright's disease are absent, it may be necessary to defer the diagnosis a little longer in order to study the urine.

The reflex convulsion in children is apt to be repeated until the cause is removed, and in this respect the condition resembles the status epilepticus, but in the former a little careful searching will probably find the cause. The isolated reflex convulsion may be more difficult to account for at first, but in these cases immediate decision is less important, and we may await time to help us. The very short duration of the petit mal separates it sharply from the uræmic fit. Nocturnal convulsions, occurring as they do often without the knowledge of the patient, are always epileptic.

The hysterical convulsion sometimes simulates closely the epileptic. But the hysterical patient rarely loses consciousness completely, the fall is not so sudden, the victim rarely if ever hurts herself, and never bites her tongue. Nor is there any rise of temperature, while even the pulse and respirations commonly remain quite normal. There is rigidity, but, unlike that of epilepsy, it is not more conspicuous in the beginning of the attack. Opisthotonos, or arching of the back, does not occur in the epileptic convulsion. Finally, the hysterical convulsion is of longer duration, lasts ten minutes or more, while the duration of the epileptic fit is not usually more than three or four minutes.

The *petit mal* is most frequently mistaken for fainting, but after two or three occurrences it should be recognized. The vertigo of Ménière's disease and of attacks of indigestion resemble it, but in the former there is deafness, while in neither is there actual unconsciousness, as is always the case in *petit mal.*

Jacksonian epilepsy is *sui generis* and is not simulated by any except the rare instances of circumscribed uræmic convulsions and similar spasms in general paresis. A further study of the cases in each instance must quickly dissipate error. While the approximate seat of the lesion may be inferred in many cases of Jacksonian epilepsy, the precise cause cannot generally, because all sorts of lesion produce the same symptoms. Recurring epilepsy in persons over thirty is probably due to organic causes, and in nine cases out of ten, according to H. C. Wood and also Fournier, is due to syphilis.

The highest refinement of diagnosis in the study of epilepsy attempts to determine from the character of the aura the seat of beginning cortical irritation. Thus a visual aura, it is claimed, might indicate that the nervous discharge began in the occipital lobes, a vertigo might indicate that it began in the cerebellum, a sense of numbness the sensory motor area of the cortex. The "intellectual auræ," as they are called by Hughlings Jackson, are regarded by him as affording evidence of a nervous discharge from the highest cerebral centres.

Prognosis.—The true epileptic rarely gets well. I believe I have seen two cases of recovery in my experience. In such statement epileptiform attacks due to peripheral irritation are rigidly excluded as not true epilepsy. These invariably get well with the removal of the irritation, while true epilepsy, in which attacks are readily excited by such irritation, is benefited but not cured. The chances of recovery are said to be greater in the young, and in the male sex than the female. One of my cases of apparent recovery was a man who had his last fit after forty; the second a woman who had no attack after fourteen. Both live. C. L. Dana places the recoveries at five to ten per cent., which appears to me large. Even cases of combined petit mal and grand mal of which the prognosis is most unfavorable are said to get well. The prognosis of petit mal is more unfavorable than that of grand mal; of the mixed forms still more unfavorable, and post-hemiplegic epilepsy most unfavorable of all.

On the other hand, an epileptic rarely dies of his disease. He may fall in the water during an attack and drown, or may choke to death if attacked while eating. Death sometimes occurs from exhaustion in the status epilepticus, but this is not frequent. But the health of epileptics usually deteriorates slowly, and life is shortened accordingly; few surviving the age of forty or fifty. They rather frequently die of tubercular phthisis. Especially frequent is mental deterioration; indeed, it may be said to be the rule where the patient lives long enough, and about ten per cent. become demented or insane. Much may be done by treatment to control the number of attacks, and the less numerous they are the less serious is the effect upon the health. Many cases earn a living, and more could if properly helped.

The more infrequent the attacks the better the prognosis. Pure nocturnal epilepsy and the pure diurnal form are each more easily cured than the mixed forms. Cases, too, which arise after twenty years of age are more likely to get well.

Treatment.—No fact in the therapeutics is better established than that the bromides control epilepsy in varying degree—it may be completely, it may be simply to render infrequent the seizures. There is probably no important difference in the efficiency of the various preparations, but the bromide of sodium is usually preferred on account of its greater solubility. Bromide of ammonium is slightly more stimulating. Causes of peripheral irritation should first be sought and if possible eliminated. Gastro-intestinal irritation should be removed; phimosis should be cured; the possible practice of masturbation should be inquired into. These eliminated, the bromide treatment may be commenced. The doses required vary greatly and must be determined by trial. Scarcely less than 15 grains (one gm.) four times a day are required for adults, and from this point the dose may be increased until the desired effect is produced. The massive doses sometimes given, amounting to ounces in a day, are ultimately harmful, but doses of a drachm, or four grams, are sometimes necessary and well borne. It is sometimes of advantage to combine the various bromides of sodium, potassium, and ammonium. Greater efficiency is secured if the drug is given on an empty stomach,

half an hour before meals or two hours after, and smaller doses suffice when thus administered. Bromism, shown by drowsiness, mental torpor, gastric and cardiac distress with acne, sometimes results. It is doubtful whether it can be obviated in any way except by omitting the drug. The bromidè eruption may be sometimes averted by combining arsenic, but this does not always succeed, and on this account, too, the drug must be omitted. In a few cases the bromides are absolutely useless, more especially in cases in which they produce gastro-intestinal derangements, perhaps in about five per cent. of the cases. Chloral adds to the efficiency of the bromides, and is sometimes necessary to produce sufficient effect. It may be given in doses of ten to 30 grains (0.66 to two gm.).

To the treatment by the bromides should, of course, be added proper hygienic measures. Suitable food, especial attention to the bowels, fresh air, and out-door life are indispensable. Bathing is important and cold baths,—particularly douches and shower baths, cold sponge baths or wet packs,—should be judiciously advised. The vaso-motor tone and circulation are thus strengthened.

The food should be simple and easily assimilated, over-eating should be especially avoided, while the too free use of meats is harmful. Rice, potatoes, fresh succulent vegetables like string beans, peas, and tomatoes, stale bread, wheaten grits and the like, with an abundance of milk are suitable. Water should be freely drunk by the patient, and a glassful advised between meals and at bed-time.

The mind should be kept occupied; nothing is more baneful to the epileptic than idleness, and it is said that cures have even been effected by giving the patient something to do.

Given a case that under the bromides has yielded to treatment, what course shall be pursued as to its interruption? The most experienced clinicians urge that the drug should be continued at least two years after the fits have disappeared, and Seguin even advises that there should be no reduction in the bromides until three years have elapsed without symptoms. My own practice has been after cessation of the fits to continue a dose of 15 to 20 grains (one to 1.32 gm.) at bed-time for an indefinite period. The friends of the patient should be impressed with the importance of such a course, as he himself is almost sure to grow indifferent after the long absence of attacks.

Of other recently recommended remedies may be mentioned antifebrin and antipyrin. A trial of the former, in the Vanderbilt Clinic in New York, by Allen Starr, was unsatisfactory. On the other hand, antipyrin, in the hands of Charles S. Potts, at the Dispensary of the University of Pennsylvania, was apparently useful. The drugs are similar and merit a trial where the bromides are for any reason unsatisfactory.

Starr has also used the tincture of simulo (*Capparis coriacea*) at the Vanderbilt Clinic with the effect of reducing the number of attacks in grand mal, but to no purpose in petit mal. It was used in doses as high as ½ ounce (13.5 c. c.) daily. In petit mal the same observer found nitroglycerine the only remedy of any service. He appears to have used it in doses of $\frac{1}{100}$ grain (0.00065 gm.) three times a day. In my experience this regulation dose fails in a large number of cases to produce the physiological

effect, and larger doses, $\frac{1}{50}$ to $\frac{1}{25}$ grain (0.0013 to 0.0026 gm.), may be given. It is to be remembered that epilepsy is one of the diseases which are nearly always influenced for a time by new remedies. The preparations of valerian may also be tried in the event of failure by the bromides. Others which have been used are borax, iodide of zinc, and sulphonal.

The nitrite of amyl has been employed to abort the attack in cases where there was an aura, and in a certain number of cases, about 25 per cent. in Starr's experience, has proven efficient.

Operation, usually trephining, is increasingly practised, and many successful cases have been reported, chiefly of Jacksonian epilepsy. When a well-defined lesion can be located, operation should promptly be done. Even in doubtful cases operation may be justified, as with modern surgical precautions it is attended with little or no risk. It should be remembered, too, that operation per se has proved curative; that is, cases have apparently recovered after trephining where no lesion was found after removing the disc.

Asylum Provision.—It is exceedingly important that some systematic provision should be made for epileptics either by the State or private charity. They are, as a rule, unwelcome inmates of hospitals because of their incurability. Doubtless the neglect to which they are subjected at home aggravates in many cases their condition, while it makes even more unhappy their lot. Provision should be made to enable them to pursue some vocation, the tendency of which has been shown to be curative. A hospital with such provisions has lately been inaugurated in Philadelphia.

Treatment of the Convulsion.—Of no small importance is the treatment of the eclamptic attack. The first measure is to secure protection against biting the tongue. Unfortunately, this is often the initial event in the convulsion. The end of a towel may be twisted and inserted between the teeth or a suitable piece of wood or a clothes-pin may be similarly used. A small object like a cork is unsafe, as it may be swallowed or drawn into the larynx and cause death by suffocation. Some patients carry such an appliance ready for use. The patient should be controlled simply enough to protect him from injury.

REFLEX CONVULSIONS OF CHILDREN.

Synonyms.—*Infantile Convulsions; Eclampsia; Epilepsia acuta.*

Definition.—Convulsions due to peripheral irritation in children.

Etiology.—There is some confusion in the use of the word eclampsia. Some would use it as simply synonymous with the word convulsion, an application, I think, altogether the best. Others apply it to convulsions due to peripheral irritation only; others seemingly to puerperal convulsions only; others, notably Hermann Eichhorst and C. L. Dana, define eclampsia as acute epilepsy. Eichhorst further says: "Epileptiform convulsions, which have the same genesis as true epileptic attacks, are excited by irritation of the cortical motor brain areas." He then names among the causes of these, toxic agencies, including uræmia

and lead poisoning, but also says he will treat only under eclampsia of the convulsions of infants (five to twenty months); among the causes of which he names heredity; psychical causes, as fright or anger; but most frequently reflex irritation, as of the skin or gastro-intestinal tract (dentition, intestinal worms, inflammation, and the like); foreign bodies; fecal accumulation; stone in the bladder, etc.; and, finally, the infectious fevers and rickets. This class of cases I have taken great pains to exclude from the epilepsies, and prefer to include at present under the heading of Reflex Convulsions of Children. The convulsions which attend diseases of the brain are a part of the symptomatology of these affections, and do not require separate consideration.

Debility and malnutrition may be considered as predisposing causes of the form of convulsion under consideration.

Symptoms.—These demand no detailed consideration, since the convulsion is epileptiform and has been described. It is much more often partial than the typical fit of true epilepsy, but it has the same stages of the tonic and clonic spasm followed by drowsiness. It is most frequently single, but the fits may follow each other in rapid succession and, though rarely, terminate fatally. As in epilepsy, the temperature rises slightly during the fit. It may come on suddenly without warning or be preceded by restlessness and fever.

Diagnosis.—This is usually easy, the convulsion coming on suddenly in the midst of health, yet traceable to some such event as the ingestion of some indigestible food, to teething, or other source of peripheral irritation.

The convulsion is distinguished from that of infantile hemiplegia by the absence of the latter symptom which is permanent. A transient paresis does, however, sometimes follow the reflex convulsion.

These convulsions most frequently occur between the fifth and twentieth month, and toward the end of the second year, though they may occur as late as the fifth year. Convulsions occurring after this period are more likely to be true epilepsy.

Prognosis.—Cases of infantile convulsions are always alarming, yet most get well, and doubtless many cases occur and recover among the poor which do not come under the notice of the physician. On the other hand, not a few deaths are caused by them,—according to Morris J. Lewis 8.5 per cent. of all deaths in children under ten; and according to West 22.35 per cent. of all who die under one year. Cases of infectious disease ushered in by convulsions are almost always serious, but the convulsions themselves are rarely fatal. The convulsions due to gastric derangement are generally followed by recovery.

Treatment.—The first step in the treatment must always consist in finding and removing the cause. If it be undigested food, an emetic and an enema are indicated; if dentition is at fault, the lancet should be promptly applied to the gums. The next step is immersion in a warm bath, say at 95° F. (35° C.) increased to 100° F. (37.8° C.), to which mustard may be added. At the same time cold should be applied to the head by means of an ice-bag or cold water. To control the convulsion chloral is the remedy *par excellence*, but while waiting for its effect

it may be necessary to permit the child to inhale a few drops of chloroform. The dose of chloral should be sufficient, 2½ to five grains (0.165 to 0.33 gm.) to a child of one year, frequently repeated until the effect is produced. It may be given in enema in double this dose, the buttocks being compressed until it is absorbed. The bromides may be given in combination with chloral, but they are altogether too feeble to be relied upon alone. Should these measures fail, opium may be used and even morphine, hypodermically, in minute doses; but these drugs should be used only as a last resort. Generally, the attack is relieved the moment the peripheral irritation is removed.

MIGRAINE.

SYNONYMS.—*Sick Headache; Bilious Headache; Hemicrania; Megrim; Migrän; Paroxysmal Headache.*

Definition.—Migraine is an intermittent, sensory neurosis, of which headache, commonly hemicranial, is the most invariable symptom. Almost as constant are aggravated nausea and vomiting, to which may be added other sensory symptoms, especially deranged vision.

Etiology.—This is obscure. It is more frequent in females—apparently three times as frequent as in males. It begins early in life, commonly at puberty, and even earlier—as early, in fact, as two years. It affects vigorous and strong as well as nervous and anæmic subjects. Exciting causes are fatigue, mental and physical, including eye-strain; digestive derangements, and menstrual disorders. As often none are discoverable.

It is usual to speak of migraine as a vaso-motor disturbance, because there are symptoms which point to involvement of the sympathetic system, but this is a matter of inference rather than demonstration. The attacks are characteristically paroxysmal. It appears to be more frequent in the winter season in this climate, and when it is not infrequently associated with a gouty or rheumatic attack. Caries of the teeth and nasal troubles are a cause in children.

Morbid Anatomy.—No lesions other than those described as causal are found. The precise seat of the pain is not known, but is believed to be in the meninges of the brain.

Symptoms.—The attack is often ushered in without any warning, at others with *prodromal* symptoms familiar to the patient. They are very various and not distinctive of the disease, but so characteristic for each case that the individual foretells the attacks on their approach. They include general discomfort, vertigo, a sense of pressure, tinnitus, spots before the eyes, chilliness, and the like. Hemianopia and scotoma may be among them. Then the pain starts in suddenly and is continuous, usually in one side of the forehead, but it may be also in the occiput, whence it extends to the half or whole head. It is extremely severe, sometimes described as blinding, at others sharp and boring or shooting. It is sometimes attended by flashes of light. Light and noise aggravate it, and a darkened room is always sought. Hemianopia is not infrequent. Along

with these symptoms are absolute want of appetite, and often intense nausea succeeded by vomiting. The vomited matter includes first the contents of the stomach (if the stomach is empty, mucous matter), and later yellow and bitter bile, whence the term "bilious headache." If the stomach happens to be full, the pain may be relieved by the vomiting.

The *vaso-motor* symptoms are conspicuous in some cases, and are assigned by some an important rôle in its causation. From this standpoint two subdivisions are made, *angio-spastic hemicrania* and *angio-paralytic hemicrania*. In the first form, described by Dubois-Reymond from observations on himself—some of the best descriptions have been by sufferers—the forehead and ear on the affected side are pale, the skin is cool, the temporal arteries contracted, the pupil often dilated, and the secretion of saliva increased—in a word, the symptoms of irritation of the sympathetic. In hemicrania paralytica, decribed by Möllendorff, also from observations on himself, the face is reddened on the affected side, it feels warm, the temporal arteries are dilated and pulsate strongly, there is sometimes unilateral sweating of the face, with the pupils contracted—symptoms suggestive of paralysis of the sympathetic. By no means all cases are capable of being thus classified, and mixed forms are met.

The frequency of the attacks varies greatly; usually they do not occur oftener than once in two weeks or once a month. They may, however, occur every ten days or even weekly.

The duration of the attack is various. Very often the patient goes to bed at night, and in the morning, or at the end of twelve hours, is relieved; or the attack may last twenty-four hours or even two or three days. The attacks continue over a period of many years, sometimes ceasing in women after the climacteric is passed, and in men after fifty.

Further speculation as to its true nature would be unprofitable here, though mention should be made that the arteries on the affected side sometimes become the seat of arterio-capillary fibrosis, a condition giving some force to the view of vaso-motor origin. A. Haig ascribes an important rôle to the accumulation of uric acid in the blood.

Diagnosis.—The symptoms of brain tumor sometimes closely simuate migraine. One case of supposed migraine under my observation turned out to be brain tumor. Ophthalmoscopic examination may discover choked disc in cases of brain tumor and thus settle the diagnosis. Such examination should always be made.

Prognosis.—This is favorable so far as life is concerned, but it is not always easy to prevent the attacks or diminish the frequency of their occurrence. It often happens that they cease after middle life.

Treatment.—Before treatment is instituted every case should be thoroughly investigated with a view to discovering causal conditions. Should such search be successful, their elimination may result in a cure. Such accessible causes are eye-strain, affections of the nose, mental and physical fatigue, and indiscretions in diet.

The attack itself is more likely to be warded off the earlier the treatment for it is instituted. The patient should be relegated to the darkened chamber. Phenacetin in ten- to 15-grain (0.66 to one gm.) doses relieves some attacks. After the first dose it may be continued in smaller

doses. Antipyrin and antifebrin are similarly successful, and I am informed by apothecaries that many women purchase these drugs regularly to relieve their attacks. Such practice should, however, be discouraged.

Sometimes a hypodermic injection of morphine, even so small a dose as ⅛ grain (0.011 gm.), acts magically, and on the whole it is the most reliable remedy, although it is one to be put off if others succeed. Caffeine is a less efficient remedy, but may be used in conjunction with morphine or immediately after it to counteract the unpleasant effect of this drug. It may be given in five-grain (0.33 gm.) doses, and is sometimes administered hypodermically. Salicylate of caffeine is also recommended in like doses. Cannabis indica is a remedy much recommended, but is unfortunately of uncertain strength. It is advised in ascending doses. Bromides may be tried. Guarana is more efficient in 30- to 60-grain (two to four gm.) doses of the powder and similar doses of the fluid extract.

If the spastic form can be distinctly recognized as present, nitrite of amyl may be expected to be serviceable, three to five drops by inhalation. In the opposite or paralytic form ergot has been advised and may be given in doses of 15 minims to one drachm (one to four gm.). Nitroglycerine in doses of $\frac{1}{100}$ to $\frac{1}{50}$ grain (0.00066 to 0.0013 gm.), and nitrite of sodium in doses of three to five grains (0.2 to 0.33 gm.), may be useful in the class of cases benefited by nitrite of amyl. Cold to the head is sometimes grateful, and where there is nausea cracked ice or cold carbonated or apollinaris water or small doses of iced champagne are sometimes efficient.

Electricity is said to have been useful in a few cases, but I have had no experience with it. It is recommended that in the spastic form the anode should be applied to the sympathetic, and in the paralytic form the cathode, the other pole being applied to the cervical cord as high as possible on the occiput.

General treatment between attacks should not be neglected. Where there is anæmia the judicious use of iron and arsenic, continued for some time, has occasionally been followed by a disappearance of the tendency to the disease.

The urine should be carefully examined, and if concentrated and tending to deposit uric acid or oxalates, diluents and the alkaline mineral waters are indicated. In a few instances in my practice the daily use of natural Vichy water, to the extent of a bottle a day, had the effect of diminishing, and in one instance of eliminating, the attacks. The conditions of a healthful life, bathing, fresh air, and simple wholesome food, should be observed. Many persons are totally free from attacks while traveling. A course at Contrexville or at Vichy, or at Carlsbad, may be of service in averting attacks.

NEURALGIA.

Definition.—Strictly speaking, the term neuralgia should be restricted to such varieties of nerve pain as are unattended with structural changes in the nerve. Formerly, many cases now regarded as cases of neuritis were called neuralgias, and it is probable that, as our knowledge grows, other so-called neuralgias will be eliminated. Finally, the border line existing between neuralgia and neuritis cannot be drawn too sharply, but as far as possible, however, the term neuralgia should be restricted to nerve pain without organic change.

Etiology.—Neuralgia is a disease of adults. It scarcely occurs before puberty and is rare in old age. It is more common in women than in men. Heredity is responsible for a tendency to neuralgia. It is frequent in the so-called neurotic families. According to Anstie, fully one-fourth of all cases are the result of heredity. Neuralgia is prone to occur in the so-called "nervous" person; *i. e.*, one who is excitable, anxious, and worrying in his disposition. In this category come, too, the hysterical neuralgias.

The debilitated, anæmic, and poorly fed are liable to neuralgia. So are they who are over-worked and worried. The most frequent exciting cause is cold. Malaria is one of the most common causes, producing, especially, hemicrania, while the malarial cachexia also predisposes to neuralgia. The pain of carious teeth is not regarded as neuralgic, but when such pain causes irritation of the peripheral branches of the 5th nerve, a neuralgia may be produced in the distal distribution.

Symptoms.—Pain is, of course, the leading symptom. "Spontaneous pain"—by which is meant pain independent of neuritis or irritation of the nerve—and the modifications to which it is subject in severity and distribution, constitute, in fact, the disease. This pain is irregularly paroxysmal, shooting, darting, or burning in character, not usually increased by motion, and if not relieved by pressure, is apt to be by gentle friction. The more the pain is increased by motion and the more there is pain over the nerve-trunks on pressure the more is it a neuritis and the less a neuralgia. Yet we cannot literally adhere to this, as evidenced by the "tender points" of Valleix—which will be further referred to under the different varieties of neuralgia. Multiple dartings and shootings, separated by seconds or minutes of freedom from pain, are characteristic.

The absence of primary tenderness is also characteristic; but after the pain has continued for some time there often succeed tenderness of the skin and even a redness and swelling, the absence of any unnatural degree of which at the beginning is considered distinctive. These phenomena, including œdematous swelling, are regarded as vaso-motor in origin. Other vaso-motor symptoms are hyperidrosis, increased secretion of saliva and tears, and elevation of temperature. Trophic effects are seen in shedding of the hair, its rapid blanching; and other symptoms to be referred to.

Muscular twitchings are also not uncommon in the seat of the pain, and sometimes even muscular spasm. The duration of an attack of

neuralgia is various, from an hour or even less to many hours. Sooner or later, if not relieved, it subsides spontaneously, though with a greater tendency to recur than when relieved by treatment.

Varieties Depending Upon Nerves Involved.

Neuralgias are variously named in accordance with the nerves affected, whence we have the following varieties :—

I. *Trifacial Neuralgia* (*Neuralgia of the 5th Pair, Tic douloureux, Prosopalgia*).—This form involves one or more of the branches of the 5th pair, rarely all. It is more common than all other varieties of neuralgia combined. Here, doubtless, we have sometimes to do with a neuritis not always easily separable. One or more numerous tender points are usually demonstrable, of which those at the supra- and infra-orbital foramen are the most conspicuous.

Of the branches of the 5th, the *ophthalmic* or the first division is that most frequently affected, giving rise to the well-known supra-orbital neuralgia. The pain radiates from the "tender point" at the supra-orbital notch over the anterior half of the head sometimes to the eye itself, the eyelid, and half of the nose. There may be injection of the eye and suffusion. There is sometimes pain in the occipital protuberance and cervical spines. This supra-orbital form has most frequently to be distinguished from catarrh of the frontal sinuses. But the latter is more likely to be symmetrical, and while the pain is severe it is more dull, less shooting, and is accompanied by coryza; it terminates suddenly with a free discharge of purulent matter, sometimes offensive.

When the distribution of the *infra-orbital* or second branch is involved, the pain occupies the area between the orbit and the mouth, over the cheek to the ala of the nose. The "tender points" are at the emergence of the nerve below the orbit, at the side of the nose, over the most prominent part of the malar bone, and along the gingival line in the upper jaw, rarely in the upper lip. When there is involvement of the third or *inferior maxillary* division, less common as an isolated form except that invading its inferior dental branch, there is a much more extensive area of pain, including the parietal eminence, the temple, the ear, the lower jaw, and the tongue. The "tender point" is in front of the ear where the auriculo-temporal crosses the zygomatic arch, where there is often burning pain. The movements of mastication and speaking may be painful, and there may be salivation. A herpetic eruption about the eye or lips is occasionally present and is then distinctive. Atrophy and induration of the skin has been included in the symptoms, but these dare only be ascribed to a neuritis.

There is a pure *ocular neuralgia* involving the eyeballs only. It may or may not be due to errors of refraction. Of these, hypermetropia, or far-sightedness, is the most common cause. Either one or both eyes may be affected. It may be accompanied by dimness of vision.

A form of trigeminal neuralgia called by Trousseau "epileptiform," consists in sudden, severe, and frequent attacks of pain, lasting from a few seconds to a few minutes, many times repeated during the day.

II. *Cervico-occipital Neuralgia.*—This affects the area of the neck supplied by the posterior branches of the first four cervical nerves, and the posterior part of the head supplied by the great occipital branch of the posterior division of the 2d cervical nerve, at the exit of which there is a tender point about half way between the mastoid process and the 1st cervical vertebra. Two other tender points are just above the parietal eminence, and between the sterno-mastoid and trapezius muscles. The pain may extend over the greater part of the neck and head, as far forward as the parietal eminence and the ear.

Exposure to cold or a draught of air is the most common cause of this form.

III. *Cervico-brachial and Brachial Neuralgia.*—This involves the area supplied by the four lower cervical and the 1st dorsal nerves, the area of sensory distribution of the brachial plexus.

The tender points are the axillary, the circumflex at the posterior part of the deltoid, the superior ulnar behind the elbow, and inferior ulnar in front of the wrist. This form is often confounded with neuritis due to rheumatic affections of the joints or injury.

IV. *Neuralgia of the Phrenic Nerve.*—This is rare, the pain in its area during pleurisy and pericarditis being rather a neuritis. The pain is at the lower part of the thorax, at the attachment of the diaphragm. Breathing is shallow, because pain is caused by the breathing movements. Coughing and even deglutition cause pain.

V. *Trunk Neuralgia.*—This naturally divides itself into two sub-varieties: dorso-intercostal and lumbo-abdominal.

(*a*) *Dorso-intercostal neuralgia* covers the area supplied by the intercostal nerves from the 3d to the 9th, and is characterized by pain along the intercostal spaces or in parts of them. It is sometimes bilateral. There is usually a constant dull pain with or without acute stabbing exacerbations, or the latter may be excited by deep breathing or motion. There may be special tenderness at the points of emergence of the three branches of the intercostal nerve, viz., near the vertebræ, anteriorly near the median line in front, and midway between these two points in the mid-axillary line.

The term *pleurodynia* has been used with a good deal of vagueness. Strictly speaking, it should be limited, as it is by Gowers, to neuralgia of the pleural nerves. Consistently with this it should not be applied to pain localized in the course or point of exit of an intercostal nerve. It is very acute in character and excited by exposure of the thorax rather than by lateral movements of the trunk. The pain of herpes zoster is not a neuralgia but a neuritis.

Another variety in this locality is the infra-mammary neuralgia of anæmic women.

(*b*) *Lumbo-abdominal neuralgia* involves the posterior branches of the lumbar nerves, especially the ilio-scrotal branch. The area of the pain is the region of the iliac crest, along the inguinal canal and the spermatic cord in the scrotum, or round ligament in the labium majus. The pain is often bi-lateral, sometimes resembling the constricting girdle pains of spinal-cord disease, from which it differs, however, by its chang-

ing place. It is especially frequent in connection with diseases of the pelvic organs, particularly in women. The testes and penis are the seat of neuralgic pains.

VI. *Neuralgia of the spinal column* is the more modern term for the "spinal tenderness" of the older authors. It is common in feeble and hysterical women, and a sequel of the modern railway accident under the name of "spinal convulsion." The pain in most cases is felt through a considerable vertical extent of the spine, but is more intense in certain spots. The dorsal region is the most common seat, next the lower cervical, and least frequently the lumbar region.

VII. *Sacral neuralgia and coccygodynia* are described by their names. These affections reside in the nerves between the bone and the skin, and are often exceedingly difficult to cure.

VIII. *Neuralgia of the feet* includes painful heel, plantar neuralgia, and erythromelalgia. In the latter, first described by S. Weir Mitchell, vascular changes, including either acute hyperæmia or cyanosis,—probably of vaso-motor origin,—are associated with severe pain in the heel or sole of the foot.

IX. By *visceral neuralgia* is meant neuralgia affecting the gastro-intestinal tract, the kidneys, ovaries, and other pelvic organs. Idiopathic nephralgia, or neuralgia of the kidney, I regard as a rare event. It and testicular neuralgia are more frequently secondary to inflammation of adjacent urinary passages, but idiopathic testicular neuralgia is less rare than nephralgia.

Neuralgias are further classified according to character and cause. Thus, in addition to the epileptiform variety alluded to, there are reflex or symptomatic neuralgias, traumatic neuralgias, herpetic neuralgias accompanying herpes, hysterical, rheumatic, gouty, diabetic, anæmic, malarial, syphilitic, and degenerative neuralgias. Many of these terms are loosely applied. The term rheumatic neuralgia is often erroneously applied to muscular rheumatism. It should not be used.

Very interesting and important is the subject of *reflex neuralgias* and *referred pains*, which have been especially studied by the late Dr. Anstie in England, and Charles L. Dana in this country. Reflex neuralgias are due to disease in organs distant from the actual seat of the neuralgia. The 5th nerve is a favorite seat of such neuralgias. Thus, an irritation of the distribution of one branch of this nerve by a carious tooth may excite a neuralgia in another distribution of the same nerve. Illustrations of referred pain is the "pain in the back" or spinal pain in ulcer of the stomach, the left scapular pain in diseases of the liver, the sacral pain in uterine disease, and the testicular pain in renal colic.

Diagnosis.—Neuralgia is chiefly to be distinguished from neuritis and the effects of pressure on nerves; also from rheumatism. From neuritis it is separated by its unilateral distribution as contrasted with the more frequent symmetrical distribution of neuritis; also by its numerous remissions and intermissions, and the shifting of the pain from one spot to another. The fixed neuralgias are more difficult of separation from neuritis, especially mild cases. The severe forms of neuritis are soon recognized by the anæsthesia which succeeds upon the hyperæsthe-

sia in the case of sensory nerves, and muscular wasting with changes in the electrical irritability in mixed nerves. In the case of compression of nerves the pain is continuous, while the symptoms and consequences of neuritis will sooner or later show themselves. Nevertheless, doubt and error must occasionally occur.

Muscular rheumatism differs in its localization in muscles or groups of muscles such as the lumbar or shoulder muscles, its continuousness and pain increased by motion.

Prognosis.—The prognosis in neuralgia is usually ultimately favorable, although some forms and cases are very stubborn. Especially true is this of neuralgia of the 5th pair. The more frequent the recurrence, and the wider the distribution, the more difficult is the cure. On the other hand, the severity of the pain is not, in my experience, a measure of obstinacy to cure, some of the severest cases being easiest relieved. Hereditary cases are the most obstinate. The same is true of cases in the decline of life. Epileptiform neuralgia is said to be incurable.

Treatment.—The treatment of neuralgia is divided into that of the paroxysm and that of the condition predisposing to it. The anæmias,—especially chlorosis,—malaria, and other predisposing causes, should be corrected by quinine, iron, and arsenic. Good nourishing food is important. Change of scene and residence are often necessary. Reflex causes should be carefully sought for and removed. Until these predisposing causes are removed, the treatment of the paroxysm affords but temporary relief.

For the paroxysm quinine is by far the most efficient remedy, and will cure many cases. Two or three grains (0.12 to 0.194 gm.) should be given hourly until the paroxysm is relieved or decided cinchonism is produced. The salicylate of cinchonidia is a valuable preparation. Some cases are relieved by phenacetin, antifebrin (acetanilid) in from ten- to 15-grain (0.66 to one gm.) doses. A combination of phenacetin and caffein, three grains (0.33 gm.) of each, in hourly doses, is often efficient. Some cases can only be relieved by sulphate of morphine. The hypodermic injection is the promptest and surest remedy, in doses of $\frac{1}{8}$ to $\frac{1}{4}$ grain (0.008 to 0.016 gm.), but morphia is a drug to be avoided in neuralgia, if possible, as the danger of acquiring the morphine habit is extremely great. The patient should never be allowed to use the hypodermic syringe himself. The use of anodynes is sometimes more than palliative, the repeated removal of the pain tending to prevent its recurrence. The combination of atropine with morphine undoubtedly modifies the unpleasant effect of the latter drug and increases its efficiency.

Belladonna and its active principle, atropine, are remedies which have long enjoyed reputation in the treatment of neuralgia, when uncombined with other drugs, but in my hands they have been feeble remedies. The doses recommended are $\frac{1}{6}$ to $\frac{1}{2}$ grain (0.011 to 0.03 gm.) of the extract and $\frac{1}{120}$ to $\frac{1}{60}$ grain (0.0005 to 0.0011 gm.) of atropine. Aconite and gelsemium have also some reputation, especially in neuralgia of the 5th nerve. Gelsemium may be given in doses of 15 minims of the tincture, frequently repeated. Gelsemia may be given hypodermically in doses of $\frac{1}{60}$ to $\frac{1}{30}$ grain (0.0011 to 0.0022 gm.) and aconite in doses of

$\frac{1}{250}$ to $\frac{1}{100}$ grain (0.00027 to 0.00066 gm.), but the latter is a remedy so dangerous that I rarely employ it. Cannabis Indica is also sometimes useful in doses of ¼ grain (0.016 gm.) three times a day, but the drug varies so much in strength that it cannot be relied upon.

Local applications are sometimes very useful. Pressure relieves many mild cases, especially when associated with gentle friction. Local anæsthetics, such as menthol, the ointments of veratria and aconitia, are similarly useful; so is the tincture of aconite painted over the involved area. The local use of opiates, at least with first removing the epiderm, is unsatisfactory. The oleate of morphine is, however, commended, and of atropine (five per cent. strength). The extract of belladonna, thinned down with glycerine so as to admit its being smeared on, is sometimes useful. Frequent renewals of all these local applications should be made in the course of the day. Counter-irritation by blisters or sinapisms, by chloroform either pure or variously diluted, and camphor may be used. The last two may be applied on lint and covered with oil silk. Both will blister if left on too long.

Cocaine might be reasonably expected to be useful, but to act through the skin surfaces the ointments and solutions containing it should be strong,—ten to 15 per cent. For mucous services this strength should be used with caution. A cocaine habit is as easily established as morphinism and is about as unpleasant in its results. The hypodermic injection of cocaine is much more efficient. The usual dose is ¼ grain (0.016 gm.) but smaller doses may be commenced with. The Paquelin cautery is often a prompt and efficient agent.

Acupuncture and aquapuncture are employed, the latter consisting of injecting water under the skin. For their local effect, also chloroform, carbolic acid, and osmic acid have been injected hypodermically. Fifteen to 20 minims (0.96 c. c.) of the first may be used, five to ten minims (0.6 c. c.) of the second, and one to two drops of a one per cent. solution of osmic acid in water and glycerine. Chloroform should be cautiously used in this manner, as it may occasion ugly sloughing. It is more especially in sciatica that these measures have been employed. Local applications of heat and cold have been found useful—cold by freezing or by the ether spray; heat by the hot-water bag, or in the case of a supra-orbital neuralgia by the nasal douche. Heat is usually more efficient than cold; indeed, the latter sometimes aggravates neuralgia.

Electricity is of uncertain value in neuralgia, but is sometimes very useful. The constant current is the form most frequently used, but faradism may also be employed. It is used in two ways: a strong current is applied at once with a view to removing the neuralgia promptly (this is scarcely to be recommended); in the second method a sedative effect is sought by a weak current, preferably of galvanism, just sufficient to produce a tingling or burning sensation. Experience goes to show that the direction of the current may be ignored, but it is commonly recommended to apply the positive pole to the painful part, the sponge being well wet with warm water, and if faradism is used, it should be with rapid interruptions.

The surgical treatment of neuralgia has been followed by brilliant

results, and has met with signal failures. The most common procedure is section of a nerve, or, better, the exsection of a portion. It has been most frequently done in the case of the 5th nerve, and is almost always followed by temporary relief. The operation is to be recommended in intractable cases and should be done at a point as near the origin of the nerve as possible, as second operations are not infrequently necessary on account of the recurrence of the pain.

Nerve stretching is also performed with a measure of relief less thorough than exsection, but in view of the fact that its disadvantages are less lasting it is the better operation to do first in the case of certain nerves. It is important to remember that relief does not always immediately follow the operation. The sciatic is the nerve most frequently stretched, but the intercostals and branches of the 5th, including the lingual, have been similarly treated with satisfactory results.

OCCUPATION NEUROSES.

SYNONYMS.—*Professional Spasm; Copodyscinesia.*

Definition.—A term applied to a group of diseases characterized by symptoms excited by an effort to perform some oft-repeated muscular act, commonly one involved in the occupation of the patient. The most usual symptom is cramp or spasm in the muscles concerned, whence this word is preceded by that of the various occupations, to indicate its special variety. Thus we have writer's cramp or scrivener's palsy, telegrapher's cramp, piano-forte player's cramp, type-writer's cramp, seamstresses' cramp, milker's cramp, etc.

WRITER'S CRAMP.

SYNONYMS.—*Graphospasmus; Cheirospasmus; Mogigraphia; Scrivener's Palsy.*

Definition.—The professional neurosis of clerks and scriveners. It is the most frequent of the occupation neuroses and may serve as the type for all.

Historical.—The first notice of writer's cramp appears to have been by Bernhart Ramazini in 1746. It was first fully described by Sir Charles Bell in 1830, and called scrivener's palsy by Samuel Solly, who published three admirable clinical lectures in The Lancet in 1864–5. Other monographs are those by G. V. Poore, in The Practitioner, in 1872–3 and 1878, also in his "Text-Book of Electricity," 1876, and Med. Chir. Trans., Vol. LXI, 1878; W. H. Erb's article in "Ziemssen's Cyclopedia," 1876, and O. Berger's article, "*Beschäftigungsneurosen*," in "Eulenberg's Real-Encyclopädie," 1st edition, 1880, 3d edition, 1894.

Etiology.—There is no predisposition in sex, the disease being more frequent in men in occupations where more men are employed, and more frequent in women where more women are employed; and it is likely that since an increasing number of women have become

telegraph operators, more cases may be expected among them, in whom, perhaps, also, the neuropathic temperament may favor it. The majority of all cases occur between twenty and fifty—154 out of 177 cases collected by Berger from Gowers, Poore, and himself. Predisposition is caused by previous injury and a neurotic disposition, while even heredity is said to predispose. An especially important factor is a faulty method of writing, while cases have occurred which were apparently independent of the usual exciting cause. Steel pens are said to be responsible for an increased number of cases since their introduction. The disease is becoming less frequent as clerical exactions grow less.

Morbid Anatomy and Pathology.—No distinctive anatomical changes have ever been discovered in writer's cramp. Its seat is doubtless somewhere in the central nervous system. Three chief theories are held regarding its nature. According to one it is essentially a local disease: weakness in certain muscles permitting over-action on the part of their antagonists, an over-action which increases to spasm. According to a second theory the spasm is reflex and due to an irritation of the sensory nerves concerned in the act of writing. The third, and usually accepted theory, makes the affection primarily and essentially central, due to deranged action in the centres concerned in the act of writing.

Symptoms.—*Spasm* is almost always the initial disturbance, commonly affecting the forefinger and the thumb; but the onset is gradual, and the first effect is an awkwardness in which the pen does not move quite as intended. It is irresistibly grasped too tightly, yet the forefinger has a tendency to slip off, the pen passing between it and the middle finger, while an attempt to mend matters by taking a new hold only increases the difficulty, and the hand labors as if tied down. It feels tired, and there is often an aching pain throughout it extending even to the arm. The writing is irregular and uneven. These symptoms may continue, with more or less impairment of the power of writing, for weeks or months, coming earlier, however, after each effort, with gradually increasing severity until the intolerable spasm sets in. This may be so violent in a combined movement of flexion and adduction in the thumb that the pen may be wrested from the grasp and thrown to a distance, or there may be a lock spasm, described by S. Weir Mitchell, in which the pen is firmly locked between the fingers. The spasm is almost always tonic in character, although it may now and then be varied by a slight start or jerk. It is sometimes associated with *tremor*. Rarely tremor occurs alone, and it may be the premonitory symptom of *atrophy*. The spasm may be limited to the act of writing, while other actions are well performed; but absolute limitation to this act is seldom met with in severe cases.

Special difficulty attends the performance of acts requiring delicate coördination of the muscles. Sometimes a patient can write with a pencil but not with a pen. *Paresis* and *paralysis* may occur with spasm, or alone. On the other hand, the strength of the hand may be quite unimpaired. Such loss of power varies greatly, being sometimes trifling, at others considerable.

Sensory symptoms are almost always present in various degrees. They may even exist alone, producing a sensory form. They are manifested at first by the distressing fatigue alluded to, or by dull pain often referred to the bones or joints, very often to the metacarpal bones or to the wrist, ceasing with cessation of writing. Sometimes there is local tenderness or a tingling sensation. Again, the pain is more severe, neuralgic in character, and distributed along the course of the nerve; induced at first by the act of writing, later by any muscular act of the part. There may be also tenderness in the course of the nerve.

Vaso-motor disturbances are seen in severe cases, manifested by hyperæsthesia, a glassy, shining skin, or a cyanosed, chilblain-like appearance; or the hand may become blue and hot on attempting to write. In the beginning the electrical reactions are normal, but in advanced cases there is a diminished faradic and sometimes increased galvanic irritability of the motor-nerve endings distributed to the muscles. It is to be remembered that the radial, ulnar, and median nerves all supply muscles employed in writing.

Diagnosis.—This is usually easy. The initial limitation of the symptoms to the act of writing sufficiently indicating the nature of the case. More frequently, other paralytic and painful affections of the arm and hand are mistaken for writer's palsy. Among these may be included hemiplegia of gradual onset, commencing insular sclerosis, early tabes affecting the arms, or pressure palsy of the musculo-spiral nerve.

In most of these cases, however, other symptoms are present or are soon superadded. More frequently, nervous persons *imagine* they have writer's palsy.

Prognosis.—A well-established case of scrivener's palsy rarely gets well. There are, however, exceptions, even under the most unfavorable conditions. The prognosis is more favorable where sensory symptoms predominate. Relapses are prone to occur when the patient returns to work.

Treatment.—Prevention, as usual, is much more effectual than curative treatment. The disease is confined almost exclusively to those who write in a cramped manner, and is said to be unknown in those who write from the shoulder. The curative treatment consists essentially in rest promptly adopted—a long rest being often sufficient to effect a cure while no other treatment can take its place. Various mechanical devices to aid in writing have not accomplished much, and the patient should learn to write with the left hand. Type-writing is, as a rule, as easily learned with the affected hand as before disability. The experience of writers has also suggested numerous simple devices by which writing is facilitated. Thus, some prefer a very thick penholder, which can be directed by the whole hand; or the pen is attached to a ring, which is slipped over the index or middle finger, and the thumb thus permitted to rest. The typewriting machine has, however, rendered all such devices of less consequence. The usual nervous tonics, such as strychnine, may be given. Hygiene of the part, including hydrotherapy, frictions, especially massage, and sometimes electricity, are useful.

The important position assigned by all neurologists to the electrical

treatment of writer's cramp demands some special consideration, especially as the methods advised are by no means uniform. The preference given to the galvanic current over the faradic is, however, almost unanimous, and, unless the latter is especially mentioned, the former is intended. Berger recommends a stabile current,—*i. e.*, a current where the electrodes are not moved about,—with the positive pole in the neck and the negative partly in the fossa supra-clavicularis, partly on the affected nerves and muscles of the arm; the length of sitting, five to ten minutes daily or every other day. Benedict recommended galvanization along the spinal column, with especial reference to sensitive vertebræ, but also localization of the galvanic current, as recommended by Berger; duration of sitting, three to four minutes, current strong enough to be easily felt. He also found subsequent faradization to the muscles most affected useful. Eulenberg also advised galvanization of the muscles affected with chronic cramp and of the involved nerve-trunks with the positive pole. In cases with tremor and rapid exhaustion the negative pole is to be applied to the spinal column and the positive on the peripheral nerve-trunks and muscles affected. Erb advised galvanization of the cervical vertebral column, with ascending stabile and labile currents combined with peripheric galvanization. In several cases it appeared to him that transverse and longitudinal currents through the head were followed by favorable results. Onimus used an ascending current through the affected arm with the negative pole in the neck and the positive pole upon the muscles of the forearm, especially the ball of the thumb, in addition to a current of moderate strength along the cervical vertebræ. M. Meyer employed a stabile galvanic current with the anode to the tender spots on the vertebral column when these were present, and the kathode on the sternum. Althouse sought to reach the cervical cord by placing the anode upon the cervical spine and the kathode on the depression between the angle of the jaw and the sterno-cleido-mastoid muscle—a position corresponding to the superior cervical ganglion of the sympathetic. The current should be mild, uniform, and uninterrupted for from three to five minutes at a time. The method should not be reversed.

Testimony is united to the effect that the galvanic treatment must be kept up for a long time, even for months continuously, with a current of moderate strength, say a maximum of four milliampères, and section electrode of about three qcm.

Faradization is recommended only in cases where there is demonstrable paresis and anæsthesia, and then in weak currents. In anæsthesia the brush may be used. Erb found that many of his patients were benefited by wearing on the arms, for several hours daily, a simple galvanic element, such as a zinc and copper plate, united by wire, and under it a moist piece of linen.

Gowers has much less confidence in electricity, especially in the spasmodic form of the disease, and is probably right when he says if the patient goes on writing it has not the slightest influence on the disease. My own experience with electricity has not been very encouraging.

The position accorded to gymnastic exercise of the arm and hand

muscles is scarcely second to that of electricity; indeed, it is preferred by some. Especially efficient appears to be that of a German writing-master, J. Wolff, of Frankfort-on-the-Main. The gymnastics are of two kinds: First, active: in which the patient moves the fingers, hands, forearms, and arms in all directions possible, each muscle being made to contract from six to twelve times with considerable force, and with a pause after each movement, the whole exercise not exceeding thirty minutes, and repeated two or three times daily. Second, passive: in which the same movements are made as in the former, except that each one is arrested by another person in a steady and regular manner. This may be repeated as often as the active exercise. Massage is practised daily for about twenty minutes, beginning at the periphery; percussion of the muscles is considered an essential part of the massage. Combined with this are peculiar lessons in pen-prehension and writing. Priority for this method is claimed by Romain Vigouroux and Th. Shott. The testimony of some of the best authorities in Europe is given in behalf of this method. Poore secured good results by combining gymnastic exercises with the use of electricity. Tenotomy and nerve stretching have been attempted and abandoned as useless.

ATHETOSIS.

SYNONYM.—*Hammond's Disease.*

Definition.—A condition of rhythmical contraction of the fingers and toes.

Historical and Nature.—It was first thoroughly studied by W. A. Hammond in 1871, also by Charcot, who considered it identical with post-hemiplegic chorea or a modified form of it. This may be regarded as correct in cases where there has been hemiplegia, and athetosis occurs in the muscles of the side which was hemiplegic. It occurs in connection with hemiplegic weakness and contraction due to tumor of the brain. Frequently it is seen as a sequel of infantile palsy. It is also found in connection with such spinal affections without brain involvement, as the spinal paralyses of children. Epileptics, idiots, and alcoholics also exhibit it in the bilateral form. Rarely, it is an independent affection, said to be the result of cold or psychical derangement, and is even held to be congenital.

Symptoms and Diagnosis.—It occurs preferably in the fingers and toes. In the fingers it is seen as a slow, though sometimes rapid, flexion and extension, adduction and abduction, with the appearance as though the fingers were attempting to grasp something. The thumbs, index, and little finger are those most frequently involved, the muscles particularly concerned being the external and internal interossei. The wrist-joint may also share in the motion, and not infrequently the opposite to that of the fingers. The patient may resist the motion but for a short time only, and it even continues during sleep, though in a less marked degree. Excitement, mental and physical, causes an increase in the motion. Subluxation of the phalangeal articulations may take place in

long-standing cases, while contractions have been known to occur in paretic extremities. Sometimes there has been hypertrophy of the front of the arm as a consequence of the constant muscular movements; in other cases the muscles are unaltered, or there may be atrophy. The electrical condition is unaltered. Similar motions occur in the toes and feet, but they are not, as a rule, so active. Even the muscles of the neck and face and tongue may share in the process. As a rule, athetosis is one-sided—hemiathetosis—but double-sided cases have been described.

TETANY.

Synonyms.—*Tetanilla; Intermittent Tetanus; Contracture des nourrices.*

Definition.—A disease consisting in continuous or intermittent tonic spasm of the extremities, usually symmetrical, but occasionally confined to one limb or rarely even becoming general.

Historical.—A knowledge of the symptom-complex of tetany was gradually acquired. The name is said to have been suggested by L. Corvisart whose book, published in 1852, was, however, preceded by one by Imbert and Gourbeyre, in 1844, and another by Drepech, in 1846. Dance in France and Steinheim in Germany described it about the same time. Trousseau (1860) first discovered the effect of pressure on the large vessels and nerves of the arm in producing the contraction in the hand-muscles. F. Chvostek (1877) described the markedly increased motor-excitability of nerves known as Erb's symptom; Chvostek and N. Weiss (1880) discovered the phenomena of facial and lip muscle contraction, elicited by tapping the facial nerve. Hoffmann (1887) showed that in tetany even sensory nerves are motorily over-excitable, and L. von Frankl-Hochwart (1887) that only galvanic excitability is increased and faradic not.

Etiology.—Tetany occurs chiefly in children, and even when present in adults it has usually made its appearance in comparatively early life. It has been supposed to be rare in North America, but J. P. C. Griffith, in a noteworthy paper read before the Association of American Physicians in 1894, collected 72 cases occurring in this country. Of these, five happened in his own experience. It is rather more common in girls than boys.

Among possible causes may be mentioned digestive derangement, including dilatation of the stomach and diarrhœa; rheumatism; exposure to cold and wet, whence it is sometimes called rheumatic tetany; rickets; open wounds; laceration; dentition; removal of the thyroid gland (13 cases followed 37 operations in Billroth's clinic, and removal of the thyroid in dogs has been followed by tetany). Pregnancy, acute fevers, and diphtheria are also alleged causes. Trousseau suggested the name *contracture des nourrices.*

Symptoms.—These are usually limited to the hands and feet. In the former the thumbs are flexed into the palms, the fingers firmly bent at the metacarpo-phalangeal articulation, but straight elsewhere. The fingers are adducted, the ring and middle fingers sometimes overlapping.

The wrists are flexed, the elbows bent, and the arms folded over the chest. In the lower limbs the knees and hips are stiff and extended, the feet extended and toes adducted. Sometimes there is dorsal flexion of the foot and flexion at the knee. *Contractions* may last for from a few hours to several days. The term *continuous* may be applied to those cases in which the contractions have lasted uninterruptedly for over two days, and *intermittent* when they do not last longer than two days without permanent or temporary disappearance. Following this standard, Griffith found 38 cases intermittent and 25 continuous. The spasm is always associated with *tenderness* or *pain*, the latter being often extreme. At other times these symptoms are present only in the beginning of the attack or when the members are handled. Rarely the muscles of the back, neck, and face are involved; and there may be *trismus*, the angles of the mouth being drawn out.

Associated symptoms are *stridulous respiration*, regarded by some as an essential part of the disease or as a manifestation of the disease in the larynx. Further interesting phenomena, especially studied and called cardinal symptoms, are *contraction caused by tapping the muscles;* as, for example, the pectoralis or the facial muscles, known as Chvostek's symptom. Another is Trousseau's symptom—the production of *spasm by pressure* upon a *large artery* or *nerve*, especially in the arm; and still another is Erb's symptom—*increased electrical excitability.* Inability to urinate may be present, and anæsthesia has been recorded among symptoms. There may be slight elevation of temperature and frequent pulse.

Diagnosis.—The rarity of this disease sometimes causes it to be overlooked, while differences of view as to what constitute its essential symptoms also cause a different diagnosis. Thus, some would exclude the carpo-pedal spasm of children; while Gowers, Dana, and Griffith include these cases under tetany. Many cases of mild spasm succeeding gastro-intestinal irritation and the like would be regarded by some as tetany and by others not. It possesses nothing in common with tetanus, whose name it so closely resembles, but whose symptoms are totally different.

Prognosis.—This is usually favorable, recovery taking place in from a few days to months. The fatal cases are those associated with dilated stomach and thyroidectomy.

Treatment.—The cause of the condition should be sought and, if possible, eliminated. After this, remedies calculated to diminish nervous excitement should be administered; also wholesome hygienic measures availed of, including massage, passive motion, and electricity. Warm baths are especially to be recommended. The cases attended with severe pain may require the hypodermic use of morphine, and delayed response to the latter may even demand chloroform inhalation.

HYSTERIA.

Definition.—Hysteria is a morbid state of the nervous system, in which may be manifested every variety of nervous symptom due to deranged function of the cerebral, basal, and spinal centres; associated with lowered will-power and exaggerated emotional tendencies.

Etiology.—Hysteria is a disease of civilization and of certain races. It is unknown in the barbarian, and is rare in Northern races, while the volatile Southern temperament favors its development. Thus the French and Italians of the Latin race furnish many subjects, while it is rarer among Germans, English, and Americans. The disease is also frequent among Hebrews.

Fully 20 women have hysteria to one man. The sexual organs of women have been held responsible for this, and the name hysteria is derived from ὑστέρα, a womb; but recent studies tend to show that this conception is erroneous. In males the disease takes more the form of hypochondriasis, but in them also convulsions, contractures, and paralysis occur. It is found in boys rather than adult males because of the greater impressibility of the nervous system in the young. About half of all cases occur in the second decade after puberty, though it may also occur earlier; one-third between twenty and thirty, while boy subjects are commonly under the age of puberty. Masturbation and adherent prepuce are held responsible for many cases in boys. Heredity plays a certain part, while the neurotic constitution especially favors hysteria.

Among the exciting causes are especially included diseases of the generative organs in women, but their influence as compared with other illnesses may simply grow out of their frequency. Ovarian disease has especially been held responsible and ovarian tenderness is undoubtedly a frequently associated symptom. Association with others similarly affected is an undoubted factor, and it is not unusual for the disease to spread itself from one to a number of girls living under the same roof. Various diseases other than those mentioned also predispose to hysteria. Even local affections, including injuries, may thus operate, and hysterical joint-affections may follow trauma of a joint. Strümpell relates an instance of a girl, who from having inhaled smoke acquired hysterical paralysis of the vocal cords. General diseases of an exhausting kind, such as fevers, nervous diseases, functional and organic, predispose to hysteria. Hysteria is common in prostitutes. Cerebral tumors, tubercular meningitis, multiple neuritis, chorea, infantile hemiplegia, often cause conspicuous hysterical phenomena. Diphtheritic paralysis may pass into hysterical palsy, while Gowers has known hysterical convulsions to attend the onset of embolic hemiplegia as shown by autopsy. Among psychical nervous causes are fright, such as attends a runaway or a fire; an angry scene; the constant operation of trifling mental causes, including worry and anxiety.

Symptoms.—An idea of the number and variety of the symptoms of hysteria has probably been obtained from the definition given—a variety which belongs to no other disease, and which may include almost all symptoms excited by any of the numerous nervous diseases. There are,

however, a certain number which are characteristic of hysteria and possess diagnostic significance, which have been called by Charcot "hysterical stigmata." They include, especially, modifications of sensibility. In addition to these are also derangements of motion, the maximum expression of which is the hysterical convulsion; also vaso-motor symptoms. The hysterical patient, however, is characterized by certain general, corporeal, and mental peculiarities which should be first considered. Such persons are emotional, irritable, capricious, sensitive, often wilful, sometimes because of indifferent early home training and overindulgence. They exaggerate every illness, and demand an inordinate amount of sympathy. If women, they are at times disagreeable and petulant or doggedly silent, while at others they are charming and fascinating. They are usually intellectually bright, almost never stupid. Other hysterical cases present no mental peculiarities. As to physical development, the hysterical patient is by no means always delicate; indeed, some of the most stubborn cases are those which appear in blooming health, rosy, well-nourished, and handsome.

(1) Derangements of Sensation.—The symptoms in this category are, as a rule, only elicited by the special examination of the physician; being rarely discovered by the patient. They include, especially, alterations of cutaneous sensibility, manifested by anæsthesia or hyperæsthesia. Most striking is insensibility to painful impressions, known as analgesia. It is usually tested by thrusting a pin deeply into the flesh—an act which is often totally unfelt. Less invariably is there failure to appreciate the sharp irritation of the electric current. Such analgesia may be confined to definite parts of the body, half the body, or may be general. It may extend to the mucous surfaces as well, and even to the deeper tissues, as those of the muscles and joints. While analgesia is the most common manifestation of deranged sensibility, there may be absence of the sense of temperature, of pressure, and even of the muscular sense.

Hyperæsthesia is almost equally characteristic. The areas involved may be exquisitely sensitive or they may be but slightly so, requiring, sometimes, considerable pressure to develop the tenderness, while at others it is elicited by the slightest touch. The hyperæsthesia is especially noticeable when the attention of the patient is directed to it by such remarks as, "This will hurt you very much when I touch you." The sensitive areas may also be limited or extended and anywhere—on the head, thorax, limbs. Ovarian tenderness is especially frequent on the left side. Even more characteristic is the hyperæsthesia of the spinal column,—the so-called "hysterical spinal irritability,"—which affects the column as a whole or in segments, not infrequently a single vertebra. The sensitiveness may be so extreme that the slightest contact may cause the patient to cry out, while strong pressure may be necessary to cause it. Of special interest also are the hysterical zones to be again referred to.

The special senses are variously involved. There may be simple dimness of vision or narrowing of the field due to anæsthesia of the peripheral part of the retina. There is often total amblyopia, but never hemianopia. Hysterical acromatopsia is not infrequent. According to

Charcot, the loss of the appreciation of violet is the most common, then of green, and, lastly, of blue and yellow. Loss of hearing is not infrequent, and still more frequent is anæsthesia of taste and smell, even bitter substances, like quinine, or pungent ones like vinegar, producing no impression or but a trifling one. The same is true of the sense of smell.

(2) Derangements of Motion.—The most striking of these is paralysis. It commonly comes on suddenly, apparently as a result of fright or other suddenly acting cause. It may, however, be gradual, and take weeks for its development. It is most frequently hemiplegic, but may be monoplegic, rarely diplegic, while every form of organic paralysis may be simulated. Hemiplegia is more usual on the left side; according to Weir Mitchell four times as frequent as on the right. The face is not affected, the neck and arms rarely, the legs oftenest. The patient can sometimes move these in bed or even when sitting up, while all attempts at walking are unsuccessful. Or she may be able to move the arms when the eyes are open but not when they are shut. It is a paralysis of the will. Sometimes one leg only is paralyzed, giving rise to a peculiar gait: the free leg making long strides while the paralyzed one is dragged along with a shuffling noise, not swung outwardly in a circle as in true hemiplegia. Sometimes there is ataxia with paresis. Paralysis may be both flaccid and spastic. Though far more frequently a symptom of hysteria in women, it may be as striking in man. Paralysis of the vocal cords is one of the most frequent symptoms of hysteria, giving rise to aphonia. The paralysis is easily demonstrable by laryngoscopic examination, because of anæsthesia of the pharynx. It may be so marked that the vocal cords actually open with attempt at phonation. Anæsthesia and motor paralysis are commonly associated.

Contractures and spasms are a form of motor derangement; they may occur alone or with anæsthesia and paralysis. They exhibit every variety, and may attack any group of muscles; they may be tonic or clonic, sudden or gradual in development. The tonic contracture is most usual in the arm, which is flexed at the elbow and wrist, while the fingers tightly grasp the thumb in the palm. In the feet, also, flexures predominate, the feet being inverted and the toes flexed. In the larger joints, on the other hand, the extensors are involved, as a rule. Rarely extensor contractures occur in the small joints; all disappear with chloroform narcosis. The reflexes may be very much exaggerated and the condition closely resemble lateral sclerosis. Even hysterical trismus may occur, and a very striking result of abdominal contracture is the phantom tumor, which is found usually just below and in the neighborhood of the umbilicus, often simulating a firm and solid growth. The mechanism of its production, according to Gowers, is a relaxation of the recti and spasmodic contraction of the diaphragm, together with inflation of the intestines and an arching forward of the vertebral column. Women have even been prepared for surgical operation on such tumors when the delusion was dissipated by the anæsthetic. Such tumor is not infrequently associated with symptoms of spurious pregnancy—pseudo-cyesis. Visible tremor may be present, rarely hysterical athetosis.

(3) Vaso-Motor Derangements.—A striking pallor is often present, at other times hyperæmia, and even a hot skin. Hemorrhage from internal organs, especially the stomach and lungs, often alleged, is apocryphal. Commonly, the blood is derived from the gums and its amount is never considerable. Yet such symptoms have been the basis of a diagnosis of pulmonary disease or gastric ulcer. Hysterical stigmata or hemorrhages in the skin are also alleged, but are invariably fraudulent.

Hysterical fever belongs also to vaso-motor symptoms. A temperature of 106° F. (41.1° C.), and even more, has been reported. Such temperatures are characterized by their irregular occurrence. Actually, they are extremely rare, being, in most instances, traceable to deception.

Anomalies of secretion include profuse and scanty perspiration, the latter resulting in a peculiar dryness of the skin; the salivary secretion is similarly influenced, and modifications in the urinary secretion are some of the most characteristic phenomena of hysteria. They include ischuria, but especially polyuria, the patient passing a large amount of very light-colored urine of low specific gravity. Excessive thirst is also frequent, further augmenting the polyuria. The chemical composition of the urine is altered in many severe cases; thus, the phosphates and urates have been found diminished, while the ratio of earthy to alkaline phosphates may be changed to one to two or one to one, instead of one to three. Such changes are held by Charcot's school to be diagnostic of convulsive hysteria as contrasted with epilepsy.

(4) Visceral Derangements.—The *digestive system* is especially disturbed by simple indigestion, depraved appetite, flatulence, and gastric pain. Not infrequently there is spasm of the œsophagus, causing difficulty in swallowing, in some instances expulsion of food before it reaches the stomach. Hysterical vomiting is very common, and alleged vomiting of impossible substances is one of the most characteristic symptoms. An antagonism to food is sometimes present, so extreme that death by starvation has been barely averted; indeed, is said to have occurred. Constipation is a frequent and troublesome symptom. Much more rare is the opposite condition of diarrhœa.

Cardio-vascular and *pulmonary* symptoms exhibit every variety, including irregularity of the heart's action,—tachycardia and bradycardia,—præcordial oppression and sense of suffocation, with extreme frequency of breathing and deranged rhythm. Laryngeal spasm, hysterical cough, and hysterical hiccough are frequent symptoms. Hysterical cries with inspiration or expiration, and imitation of the sounds produced by various animals are described by the French neurologists. Joint affections, purely hysterical, were early studied by Sir B. Brodie, and later by Sir James Paget. They involve the knee and hip and consist of fixation, tenderness, and even swelling.

The *mental symptoms* are a prominent feature of hysteria, and vary greatly in their manifestations. Irritability and capriciousness of temper, maniacal excitement, hallucinations, and even insanity may occur. The hysterical trance is a well-known condition. It may come on spontaneously, but more frequently it follows one of the forms of hysteroid

attacks to be later described. The cataleptic state may be associated with this symptom.

(5) Convulsive Seizures.—Hysterical convulsions are a recognized symptom, while in some they are the only manifestation of the disease. Their severity varies greatly; but two degrees are described, a milder or minor, and a severer or major.

(*a*) *Minor Form.*—This may come on suddenly or be preceded by a prodrome, including hysterical behavior, such as laughing and crying; a sense of constriction about the throat, or that of a ball rising in it (the so-called globus hystericus); a feeling of anxiety with shortness of breath; or pain and discomfort in the chest or abdomen.

In the actual seizure the patient falls, with this striking feature that she rarely fails to find a soft spot, such as a sofa or bed, to receive her. The convulsion consists in clonic contractions of a disordered and irregular kind, in which all four extremities and even the trunk may take part. Though seemingly unconscious, the patient still gives, to the careful observer, the impression of a certain method in her madness. The convulsion lasts usually a few minutes, when it passes off spontaneously, or the patient may be aroused by some powerful impression, such as the dashing of cold water in the face, or a sharp galvanic shock. She may remain emotional for a time, but the period of torpidity, so characteristic of the epileptic fit, is rare.

(*b*) *Major Form* (*Hysterical Epilepsy*).—This has become widely known, more particularly from the graphic descriptions and vivid pictures furnished by the French school of neurology. It is much less common in this country; indeed, rare outside of hospital walls, where prostitutes are the usual subjects. The attack may be preceded by prodromata similar to those that precede the milder attacks. The convulsion is described by French writers as having four distinct stages:—

(1) The epileptoid state, closely simulating a true epileptic attack, with apparent unconsciousness, tonic spasm, even opisthotonos, grinding the teeth, livid face, succeeded by clonic convulsions, relaxation, and coma; lasting rather longer than the true epileptic attack.

(2) The period of "contortions and grand movements," called by Charcot "clownism," characterized by emotional display, striking contortions, or cataleptic poses.

(3) The period of plastic positions and passionate attitudes, including those of ecstasy, fright, beatitude, or erotism.

(4) The return to consciousness and a stage characterized especially by manifestations of delirium, with extraordinary hallucinations and by hypnotic "suggestibility." In it visions are seen, voices heard, and conversations carried on with imaginary persons. Imaginary events are related as actually true. These hallucinations sometimes even persist after recovery.

Suggestion and Hypnosis.—At this point it is suitable to say something of these conditions so closely associated with the hysterical state and which have attracted much attention of late. By suggestibility is meant the susceptibility of a person to the production of a definite psychical or physical state dependent upon the arousing of corre-

sponding ideas in the mind. It is really a further development of the hysterical mental constitution already referred to, in which the patient permits himself to be dominated by his imagination. Suggestion is merely the artificial fostering of the psychical peculiarity. It is most easy during the part of the hysterical attack, when the patients speak, hear, and answer. At such times a definite direction may be given to the patient's ideas. If he is told in an emphatic, convincing manner that he is in a certain situation, be it one of a pleasurable kind or a state of suffering or danger, he believes it, and at once, by behavior or expression, shows that he believes it, and is actually experiencing the conditions named. Physical states may be similarly suggested, such as paralysis, contractures, and anæsthesias, while severe pain may be inflicted without exciting sensibility. After the attack is over the subject is totally ignorant of what has transpired, but during another attack may remember the events of the previous one, or, what is still more strange, supposed events, furnishing thus an instance of double consciousness.

Hypnosis is closely allied to suggestion. It is, in fact, nothing more nor less than the intentional production of a hysterical attack, or a hysterical psychosis by suggestion. As Strümpell graphically puts it, "Hypnosis is an artificial hysteria." The French school make four principal forms of the hypnotic state with many transitions :—

(1) The cataleptic state, in which the limbs retain all the positions artificially given them.

(2) The state of suggestion or artificial hallucination, in which patients are induced to eat tasteless and unnatural food with a gusto.

(3) The lethargic state : a state of apparent unconsciousness with the eyes closed, the muscles relaxed, yet with a markedly increased excitability in the muscles and nerves in which a light tap on a nerve like the facial is sufficient to put all the muscles supplied by it into a tetanic contraction far outlasting the irritation.

(4) A state of hysterical somnambulism, in which the patient, while remaining half unconscious, still answers automatically questions put to her, obeying orders or giving them, and sometimes exhibiting certain sensory hyperæsthesias. It will be seen that each of these correspond with one or another of the different manifestations of the hysterical attack.

Hysterogenous Zones.—In this connection some further reference should be made to the so-called hysterogenous zones already alluded to. These are hyperæsthetic areas especially studied by Richet, on which persistent pressure will sometimes excite a hysterical attack. While the ovaries are favorite hysterogenous zones, the zones may be in any part of the body ; as, for example, the sides of the trunk. Such pressure may also cause an existing attack to subside. Hysterical spasm may also be localized or limited to groups of muscles.

Diagnosis.—This is not usually difficult. There is something indescribable in the bearing and appearance of a hysterical patient which enables the experienced physician often to recognize the disease at a glance. While, as stated, every phenomenon of any organic nervous disease may be present, yet the essential symptoms of organic lesion are still wanting, and there are symptoms which are peculiar to hysteria alone. The an-

æsthesias are peculiar in their area of distribution, and hysterogenous zones are nowhere else found. The hysterical convulsion is quite *sui generis*, the throat and pharyngeal symptoms are not found elsewhere, and the emotional symptoms are tell-tale.

Prognosis.—This is very rarely serious, though the course and duration of the disease varies greatly. The milder cases may be of very short duration, while the more serious may last for weeks and months—always, however, with intermissions and changes. Only in the rare instances of hysterical starvation alluded to do we have a fatal result.

Treatment.—A proper prophylactic treatment, so commonly overlooked, would prevent almost every case of hysteria. The counteracting of all that is mentioned under the head of predisposition constitutes such treatment. Wholesome discipline and training in youth, the inculcation of self-denial as contrasted with over-indulgence and the gratification of fancy, and careful exclusion from the companionship of hysterical persons make up the sum of these.

The successful curative treatment of hysteria also more frequently depends upon the individuality of the physician than on the remedies employed. Indispensable, however, is the removal of the causes which predispose to the disease, whether they be of the nature of moral influences or bodily ailment. Among the most difficult to eliminate of the former are those which arise from the fondness and sympathy of relatives who have from long habit become almost slaves to the fancies of the hysterical subject, and with whom, in consequence, firmness has become impossible. It is in consequence of such difficulties that the isolation plan of treatment, which has become inseparably associated with the name of Weir Mitchell, has been so successful. Originated for neurasthenic cases, it is as applicable to the hysterical in whom neurasthenia is also often present, while hysteria also often forms a large factor in the neurasthenic state. Whenever possible, the patient must be removed from her previous surroundings, her family, and even friends. This accomplished, the details of management largely depend upon the peculiarities of the case, but in a general way the Weir Mitchell plan may be said to consist as follows:—

First, and indispensable, is the care of an intelligent and sensible nurse. Under her charge the patient is put to bed and kept in a condition of absolute rest, even reading being prohibited, and also at first self-feeding. Massage is used daily, at first for short periods, which are gradually lengthened, until an hour is thus consumed. With massage is associated electricity, the faradic current with slow interruptions being usually preferred. Thus, with a small electrode, the motor nerve-points can be picked out, and the contraction of individual muscles produced. Massage and electricity both have for their purpose the substitution of exercise, to which end the former is by far the more useful and important. Both are discontinued during menstruation. The food at first is milk, which has been usually skimmed, but, in my own experience, good milk unskimmed and diluted with one-fourth its bulk of water, or aërated water answers the purpose better. The proportion of casein is less, and the oil, which is so valuable for the nutrition of the patient, is retained in more nearly its nor-

mal quantity. At first from four to six ounces of milk are given every two hours. After a week or ten days a chop or a few raw oysters are added at luncheon, with a cup of coffee or tea, and later an egg at breakfast and bread and butter or biscuit with the milk, the latter being continued at two-hour intervals. The patient should have a thorough sponge bath daily at the hands of the nurse. It is convenient to make out a schedule including the hours for nourishment, massage, and electricity, of which the last should be separated by several hours. Massage should be followed by a full hour's rest. Under this forced feeding the patient gradually fattens, and concurrently with this the excitability of the nervous system usually grows less. In a month or six weeks the patient is allowed to sit up, at first for a few minutes only, but each day a little longer, until the whole day is thus spent, interrupted by periods of rest. Later she is taken out to drive, and then to walk for gradually increasing distances, until, in the vast majority of instances, she is enabled to perform enormous amounts of physical exercise without fatigue. I have known cases bedridden for months and even years, women whose relatives had been worn out with nursing, who, after a few weeks of this treatment, acquired the most vigorous health, walking many miles a day and presenting an appearance of health and strength which would be considered absolutely impossible by one unfamiliar with the results of this mode of treatment. As a rule, three months should be asked for its fulfilment. As has already been said, the individuality of the physician has much to do with the success of the method. A firm, earnest, and yet gentle manner will do more with such cases than one who is vacillating and disposed to yield to the caprice of the patient, who soon measures such a person and effectually antagonizes the treatment. An element of "suggestion" must perhaps be acknowledged in the power of the physician thus constituted, yet the full application of this principle of treatment by hypnotic suggestion is to be deprecated. The nurse in charge must be similarly constituted, and it not infrequently happens that a nurse otherwise excellent is totally unadapted for the management of a case of this kind.

As to medicines, the number which is useful is few. Iron and arsenic, in very moderate doses, are the only ones which are actually curative. The various nervous sedatives, including valerian, asafœtida, the bromides, the milder hypnotics such as phenacetin, rarely chloral, may be used as occasion requires; morphia never, or almost never.

The paralysis and contractures generally require some time to overcome, but their true nature being determined the physician may confidently promise an ultimate cure. This is accomplished mainly by manipulation aided by electricity, under the use of which the symptoms are gradually overcome and the patient, induced at first to walk for a few steps, will slowly acquire full power of locomotion. Anæsthesia is best treated by faradization and the electrical brush. Paralysis of the vocal cords is also best treated by electricity, suitable electrodes having been devised for that purpose.

Allusion should be made to metallotherapy, a treatment instituted by a French physician named Burq, who ascertained years ago that by lay-

ing plates of metal upon a cutaneous surface affected by hysterical anæsthesia, sensation is sometimes at once restored not only in the immediate region but also sometimes in a much larger area. The cases so treated were, for the most part, hysterical hemianæsthesias. Iron is the metal most frequently efficient, but sometimes copper, zinc, or gold are employed. The process of determining the metal essential to each individual case was called *metalloscopy*, and Burq held that this method would also have the same effect if given internally. A committee of the Paris Society of Biology tested these statements in 1876, and confirmed them, except as to the internal administration of the metal. A similar discovery was that of Charcot, also substantiated, that the return of sensation to an anæsthetic area as the result of applying a metal plate is accompanied by a simultaneous development of anæsthesia upon the opposite side previously normal and in an exactly corresponding place. This is known as transfer. Other hysterical symptoms than anæsthesia have been found to exhibit analogous phenomena. Thus transfer can sometimes be observed in hysterical amblyopia, acromatopsia, deafness, loss of smell and taste, contractures, and paralysis, while such transfers may be induced by other means than metal plates, known as æsthesiogenous remedies. They include large magnets, feeble galvanic currents, static electricity, vibrating tuning forks, and sinapisms. It must be plain to any one who thinks that these phenomena are merely the result of suggestion produced by ideas similar to those already described. Their career will doubtless end like that of Perkins' tractors. Hypnotism has also been employed of late for the treatment of hysteria, and has acquired some popularity in France, where it has been especially practised by the school at Nancy. Wonderful cures have doubtless thus been accomplished. But based as it is upon mysticism and imagery, and being already much abused by charlatans, it is to be hoped that its fate will also be that of metallotherapy and Perkins' tractors.

NEURASTHENIA.

Synonyms.—*Nervous Exhaustion; Nervous Weakness; Encephalasthenia; the American Disease.*

Definition.—A term originally suggested by George N. Beard, in 1879, for a complexus of symptoms without anatomical basis, in which muscular weakness, nervous irritability, and pain are variously manifested. Beard defined nervousness as "Deficiency of nerve force, manifested chiefly by undue sensitiveness to external impressions," and neurasthenia as "A sign and type of functional nervous disease" evolved out of this general nervous sensitiveness. The line of demarcation between neurasthenia and hysteria is not always definite. Not only do the two conditions sometimes merge, but certain cases of neurasthenia are in no way distinguishable from the minor forms of hysteria. The condition is called *spinal*, *cerebral*, *cardiac*, or *gastric*, according as the symptoms dependent on one or the other of these systems predominate, but the line of demarcation is not sharp.

Etiology.—The same class of persons who are predisposed to hysteria are predisposed to neurasthenia, and such predisposition may be inherited or acquired. So, too, many of the exciting causes of the former become the exciting causes of the latter. Among these are over-strain of mind and body, over-work, especially over-work associated with care and anxiety. It is distinctive of neurasthenia as contrasted with hysteria that it is more frequent among men, on whom business care and financial worry fall more severely. It is well known that men differ greatly in their power to bear the mental strain incident to the struggle for existence or business success. From the special prevalence of this disease in America, it has been called the "American Disease," and is reasonably ascribable to the fact that mental and physical strength in this country is more taxed than in any other.

Morbid Anatomy.—Although Beard took great pains to prove that neurasthenia is a physical and not a mental state, and that these phenomena do not come from emotional causes or excitability but from nervous debility and irritability, there has been found no distinctive morbid change associated with its complexus of symptoms any more than with hysteria. It is barely possible that the recent investigations of C. F. Hodge, demonstrating changes in nerve-cells during functional activity, may result in some further knowledge in this direction.

Symptoms.—It has already been said that the symptoms of the minor forms of hysteria are the symptoms of many cases of neurasthenia. The appearance of the patient may be that of perfect health; less frequently he looks worn and worried. In the *spinal* form motor phenomena are the most conspicuous. Of this, and, indeed, of all forms, the most constant symptom is muscular weakness, as a result of which the patient complains of being tired and weary, even too weak at times to keep out of bed. Such weakness may affect the gait, making it uncertain and trembling, and the acts performed by the upper extremities may be similarly embarrassed. There may be hyperæsthesia and paræsthesia, and even the special senses may be affected, especially vision and hearing. The latter is more frequently over-sensitive, and vision may be obscured by the presence of scotomata or muscæ volitantes. Especially characteristic is a low-spiritedness or despondency, often painful to witness, and which may alternate with irritability or moodiness. Another symptom is sleeplessness, though many patients sleep well; indeed, there is occasionally an irresistible disposition to sleep. A disposition to seek solitude is characteristic, while at other times the patient fears to be alone. Again, he is restless, unsettled, and impelled to move about from place to place, while there is sometimes a pronounced disposition to suicide. Confusion of mind, and especially a difficulty of dealing with figures, is a very common symptom, sometimes an initial symptom, the simplest arithmetical problems being quite impossible with one so affected.

The *cardiac* form is characterized by palpitation and frequent irregular action of the heart and præcordial pain, which give rise to the belief in the patient's mind that he has cardiac disease, arterio-capillary fibrosis or "hardening of the blood-vessels" as it is called by the laity. Of vaso-motor phenomena there may be flashes of heat, sudden sweats,

even night sweats, and a relaxed state of the peripheral blood-vessels to an extent which may cause the "water-hammer" and even capillary pulse similar to that of aortic regurgitation. Epigastric pulsation is often an annoying symptom in women. Among visceral symptoms are, especially, gastric pain, the distinctive symptom of "nervous dyspepsia," but there is also distention and discomfort after eating, or a constant noisy motion of gases—borborygmus. Polyuria is a conspicuous symptom. A slight degree of glycosuria and even intermittent albuminuria have been reported. The opposite condition of urine—a dark hue and high specific gravity—is more rarely present. Hoarseness and aphonia are laryngeal symptoms, while very frequent breathing is a distinctive hystero-neurasthenic symptom.

Diagnosis.—This is generally easy, and is arrived at by the exclusion of the objective symptoms of organic disease and by the etiology. For it will be observed that all of the symptoms which have been narrated are subjective in character. The so-called spinal irritation is a condition which resembles neurasthenia, and probably some of the cases so named which are not hysteria are cases of nervous exhaustion. Sensitiveness of the vertebræ is not apt to be present in neurasthenia; whereas it is the most distinctive symptom of spinal irritation.

Prognosis.—Recovery from neurasthenia may be confidently promised to almost every case which is in a position to meet the indications of a successful treatment, which, unfortunately, are apt to be expensive, though the modern hospital affords to even the poorer classes an asylum where the treatment may be successfully carried out. On the other hand, it is mostly the rich who suffer from neurasthenia, and they are in a position to meet these requirements.

Treatment.—The first essential condition of a successful treatment is removal of the causes which are responsible for the illness. To this in the case of women, and sometimes of men, the most successful adjuvant is the rest treatment of Weir Mitchell, the technique of which has been already described under the treatment of hysteria. After this and, in the case of men often even before this, removal from the scene and surroundings which attended the development of the disease is most useful. Travel away from home, especially in foreign countries, a sojourn at a sanitarium or health resort, the seaside, the woods, the mountains, for prolonged periods, have always, in my experience, sooner or later been followed by recovery. For the poor, the rest-cure as carried out in hospitals may be substituted for the more expensive methods of home treatment.

The treatment of the *insomnia* of neurasthenia calls for brief special consideration, and what is said here may apply to the treatment of any form of simple insomnia, by which I mean insomnia not the result of pain. Modern therapeutics have added to our resources a number of harmless drugs which are more or less efficient to this end. The best of these, considered from all standpoints, is sulphonal. Not less than ten to 15 grains (0.66 to one gm.) should be given to an adult, while twice as much may be given if needed. I prefer to give this dose and repeat it in an hour if no effect follows. It is bulky, soluble with difficulty in cold

water, but readily so in any hot menstruum, and especially suitable is hot milk. It should be given an hour or two before sleep is desired, but associated quietude is necessary to secure its effect. Nearly the same may be said of phenacetin in the same doses. It is rather more conveniently administered, and may be placed dry on the tongue and washed down with a mouthful of water. Paraldehyde is an excellent remedy but very disagreeable, and is more prompt in its action than sulphonal or phenacetin and should be given in drachm (four gm.) doses. Chloralamid is also a good hypnotic; its dose is 30 grains (two gm.). It should be dissolved in some alcoholic menstruum diluted to ½ ounce (13.5 c. c.). Trional may be used in doses of 15 to 20 grains (one to 1.3 gm.). Chloral,—as a simple hypnotic is better, perhaps, than any of those named,—has yielded its former high place to those just mentioned because of their harmlessness. It has the further disadvantage of sometimes causing drowsiness the next day, and occasionally it is exciting. From ten to 30 grains (0.66 to two gm.) should be given at a dose. It is especially useful when combined with morphine, making a much smaller dose of this drug efficient, but morphine should not be used if it is possible to get along without it. Hydrobromate of hyoscin may be used in doses of $\frac{1}{100}$ grain (0.00064 gm.) if the drugs named fail. Sometimes it acts like a charm, at others it produces the opposite effect—exciting the patient. One trial suffices to settle the question.

It is true of all the drugs named that their effect is apt to wear off, and increasing doses must be used. It is, therefore, desirable to obviate the necessity of their use as early as possible, and, if possible, substitute other measures. Often the patient simply needs a start to put him in the way of sleeping, while sometimes the simple feeling that there is something at hand, which he can use if he wishes, gives him the needed confidence and he goes to sleep at once. A warm bath before retiring, or even at times a cool bath or cool sponging, and again a hot bath, promotes sleep. To persons residing in cities, sleep is often favored by a sojourn at the seaside, many being able to sleep there when they cannot do so at home. The same is true of the country or the mountains.

It is important, too, in our efforts to secure sleep for our patients to investigate the various functions, derangement of any of which may keep a neurasthenic patient awake. Irregularities of digestion and circulation should receive attention. An undigested meal or a loaded bowel often keeps one awake, while an excited heart by its ceaseless beating repels the restful sleep without which life is wretched. Often a light meal or a single glass of wine seems to furnish the brain-cells the right amount of stimulus to enable them

> "To shut the banging doors and windows wide
> Of restless sense, and let the soul abide
> Darkly and stilly for a little space,
> Gathering its strength to pursue the race."

TRAUMATIC NEUROSES.

SYNONYMS.—*"Railway Brain;" "Railway Spine;" Traumatic Hysteria; Erichsen's Disease.*

Definition.—A neurasthenic or hysterical state, the result of shock from railroad accident, or accident of similar alarming character.

Historical.—The condition was first studied by Erichsen in 1868 to 1875, under the designation of "Railway Spine." Erichsen regarded it as an inflammation of the spinal meninges. Leyden, in 1875, made important contributions to the literature of the subject in treating of spinal concussion. Spitzka, in a review of "Spinal Injuries as a Basis of Litigation," in 1883, referred the symptoms to the category of spinal irritations. J. J. Putnam and G. L. Walton, in 1883, first pointed out the hysterical nature of the affection, a view which is commonly accepted to-day. H. W. Page, an English railway surgeon, published in 1885 a considerable volume directed against Erichsen's view. There still remain a few, and among them S. V. Clevenger, in a work on "Spinal Concussion," published in 1889, and Gowers, in his most recent edition (1892), who hold that railway spine is something more than a purely mental condition. W. A. Hammond suggested the anæmic origin.

Etiology.—Profound nervous shock, however induced, by railroad accidents, shipwreck, boiler explosions, and the like, even where the sufferer himself is not a victim, but is profoundly impressed by it, are capable of producing this nervous state.

Symptoms.—These are not essentially different from those of neurasthenia from other causes. The most remarkable fact with regard to them is that they do not necessarily immediately follow the accident, and there may be some interval of time between the two events—the accident and its result. In some cases the symptoms appear suddenly, in others they are gradual in their invasion. All the symptoms detailed under neurasthenia may be present, especially spinal tenderness and pain in various parts of the body, principally the back and head; there may be numbness, and tingling in the extremities, increased muscular irritability, and increased knee-jerk. The latter varies from day to day, and may be exhausted by repeated stimulation. Extreme depression of spirits is another symptom. Other patients exhibit active hysterical symptoms, including modifications of sensation and motion, hemianæsthesia, anæsthesia, paresis, and even paralysis.

In the more severe cases where there is actual concussion the symptoms suggest organic changes, which are, indeed, actually found at times in the shape of pachymeningitis. Such cases exhibit diminished superficial reflexes, with exaggeration of the deep ones. There may be severe pain, variously distributed. Other symptoms are alterations in the temperature sense, and in the muscular sense, both of which may be bilaterally distributed. There may also be modification of the special senses, including smell, taste, and vision, with inequality of pupils. There may be monoplegia with or without contracture. Symptoms which imply true organic change are optic atrophy, bladder symptoms, paresis,

and exaggerated reflexes. Such cases are sometimes, though rarely, fatal.

Morbid Anatomy.—In the vast majority of cases anatomical changes are not discoverable; in fact, as most cases recover, there is little opportunity to seek them. In a few, however, morbid alterations have been found in the brain and spinal cord, including degeneration of the pyramidal tracts of the cord, demonstrated by Edes in four cases; multiple sclerosis in the white matter, and arterio-sclerosis in the vessels of the brain with scattered areas of degeneration.

Prognosis.—Most cases get well. The effect of litigation is often to delay recovery, while successful litigation does not always relieve the symptoms, and when it does it is by no means always speedily—months and even years elapsing before the cases recover. A few cases, where there is true organic disease, perish.

Treatment.—Rest, mental and physical, is the first essential condition of recovery. It may be aided by the measures useful in other forms of neurasthenia, such as massage, electricity, and proper feeding. Medicines avail little, except for their moral effect. Narcotics should be avoided.

OTHER FORMS OF FUNCTIONAL PARALYSIS.

Abasia-Astasia.

Definition.—Abasia (*α* privative; *βασις*, a step) is a term given by P. Blocq, in 1888, to a difficulty in starting the act of walking from a state of previous rest. Astasia (*α*, and *στασις*, a standing), an inability to stand, contrasted with integrity of sensation, muscular strength, and coördination of other movements of the legs.

Nature.—The phenomena are thus far inexplicable in the absence of discoverable lesions, and the difficulty in their comprehension is increased by the fact that we know nothing certain as to localization of the centres of walking and standing. It is a disease of adults, equally frequent in men and women—as determined by Knapp's study of 50 cases, of which half were in either sex.

Symptoms.—These occur in connection with a variety of morbid states, and a large majority of them are doubtless hysterical.

In the "unconscious" variety the patient is without any idea that he cannot walk or stand when he finds suddenly that he cannot do either. Another variety of abasia-astasia is the "hypochondriacal," in which the patient acts under "conscious" erroneous impression that he cannot walk or cannot stand. It is sometimes associated in the hypochondriacal paranoiac with paræsthesia, and in the neurasthenic with abnormally increased sense of fatigue. A third form is associated with some suddenly acting "shock," as fright, which acts inhibitorily on the motions of the patient. Finally there is the "coercion" variety of abasia-astasia, in which the patient while in the act of walking or standing is suddenly seized with the idea that he shall walk or cannot walk. This differs from the hypochondriacal form in that the patient is conscious

of the erroneousness and absurdity of the idea, but is nevertheless coerced by it.

These different forms are not always sharply defined. Suddenness is especially characteristic in the "unconscious" form. In other cases the patient may walk a few steps and then suddenly break down. Sometimes he stands rooted to the ground, as it were. At others the development is slow, requiring even a couple of years to reach its acme. Sometimes it is preceded by trembling or staggering, as associated symptoms, the result of the effort of the patient to stand or move forward.

Closing the eyes usually increases the difficulty. On the other hand, sometimes, with the eyes closed the patient can walk in the normal manner, when it is impossible to do so with the eyes open. The latter is especially true of the hypochondriacal variety. In these cases, too, the natural gait is sometimes restored after attempting an unusual method of walking, as walking backward or with the legs crossed or by leaping or in military step. So, also, abasics can walk on all fours. The morbid state is also influenced by certain surroundings, as broad open surfaces or long narrow corridors or standing without special support. Th. Ziehen refers to a case in which it came on when the patient walked under a tree, the moving leaves of which produced moving shadows. There is sometimes associated tachycardia; at others evident hysterical symptoms, such as tender spots, hemianæsthesia, and the like. In other cases there are epilepsy, paralysis agitans, or chorea.

Diagnosis.—This is based upon the retention of absolute integrity of sensation and of muscular strength and of coördination of the legs, demonstrable in the recumbent position. By these features it is distinguished from tabes and Friedreich's ataxia. The symptoms resemble those of disease of the tubercula quadrigemina and cerebellum, but in the former there is oculo-motor paralysis, and in the latter vomiting, choked disc, and headache. From hysterical paraplegia it differs in that the power of motion is intact in the recumbent position

Prognosis.—This is regarded as favorable, though relapses occur.

Treatment.—The evident hysterical nature of the affection, in the majority of cases, suggests the treatment for such cases. The rest-cure, massage, gymnastics, electricity, gradually increasing practice in walking, are measures which are likely to be useful. Th. Ziehen, to whose article in "Eulenberg's Real-Encyclopädie" I am indebted for much of the information in this section, recommends "suggestion without hypnosis," especially in the hypochondriacal and hysterical forms, as a reliable means of rapid cure; and in the cases where fear or terror are conspicuous, small doses of opium.

Periodical Paralysis.

Definition.—A form of hereditary or family paralysis involving the general muscular system, recurring at intervals of from four to six weeks and confined mostly to children.

Etiology.—The disease is hereditary and transmitted through the mother. As many as twelve members of a single family have been affected. Goldflam suggests that the paralysis is due to autointoxication,

the poison acting upon the nerve endings in the muscles; while he also found that the urine secreted during the attacks was more toxic than at other times.

Symptoms.—The disease occurs in youth and in the midst of health, even during sleep. Beginning as a weakness or weariness in the arms and legs, it is usually complete in twenty-four hours. It is rarely confined to the legs and may also involve the muscles of the neck and even those of the tongue and pharynx.

Sensation for the most part is unaffected as are also the special senses. The deep reflexes are diminished, sometimes abolished, while the superficial reflexes are feeble. Faradic sensibility of nerves and muscles is greatly lessened, sometimes absent. There is no fever and sometimes the temperature is below normal, while the pulse is slow.

The attack recurs at intervals of from one to two or more weeks, in some instances, daily. It begins to abate usually in a few hours or after a day or two and ultimately disappears completely, and the patient remains well until another attack sets in.

VASO-MOTOR AND TROPHIC DERANGEMENTS.

Preliminary Remarks.—Two kinds of vaso-motor nerves are described, the vaso-constrictors and the vaso-dilators, which are influenced in opposite ways by irritation and paralysis. Although Stricker and others have been at much pains to demonstrate the existence of vaso-dilators, the number hitherto recognized is but few, limited as far as I know to the chorda tympani nerve, the nervi erigentes, and in some fibres the sciatic, while thus far the proof of their existence has been confined to the domain of experimental physiology. Pathological phenomena are, therefore, almost wholly referred to effects on the vaso-motor constrictors, abnormal constriction being ascribed to irritation, and abnormal dilatation to paralysis. There are many seats of vaso-motor irritation and paralysis. One of these is doubtless the cortex cerebri, as shown by the effect of emotion in producing both blushing and pallor, and by the fact that actual lowering of temperature has been produced on the opposite side by irritating motor areas, and an increase of temperature by excising them. A vaso-motor centre undoubtedly resides in the medulla oblongata in the region of the upper olivary body in rabbits, the irritation of which causes vascular constriction, and destruction causes vascular dilatation. In the cord the vaso-motor fibres run mostly in the anterior columns whence they pass out by the anterior roots, but some probably also traverse the posterior roots. From what has been said it seems likely, also, that these nerves decussate, though exactly where is not known. Outside the spinal cord the sympathetic is the chief carrier of the vaso-motor filaments, which go thence to form networks about the blood-vessels. Some vaso-motor filaments are probably contained in all peripheral nerves.

Symptoms which point to *vaso-motor paralysis* are an abnormal hyperæmia, redness and warmth of the skin, objective as well as subjec-

tive. These occur in connection with the various forms of cerebral and spinal paralysis as well as with injuries of the sympathetic system, and in such purely functional affections as hysteria and neurasthenia. Sweating is another symptom of vaso-motor paralysis; more rarely swelling and pain, as in the condition described by S. Weir Mitchell as erythromelalgia.

Symptoms of *vaso-motor spasm* are the opposite conditions; pallor and coolness, with occasional stiffness, formication, and even pain, such as will be described under Raynaud's disease, and scleroderma.

It is still undetermined whether there are trophic nerves separate from the sensory and vaso-motor systems. We do, however, know that sensory and motor derangements are followed by nutritional changes of a very decided character, while certain other phenomena are probably of a mixed character; *i. e.*, partly vaso-motor and partly trophic. Such, for example, are urticaria, erythema exudativum, and herpes along the course of nerves.

Acute Angeioneurotic Œdema.

Synonym.—*Giant Urticaria.*

Definition.—Œdematous swelling occurring suddenly in various parts of the body, disappearing in a few hours, perhaps to recur again.

Historical.—Its history has been studied by Quincke and Strübing.

Etiology.—Heredity is sometimes observed but other cause is unknown.

Pathology.—The condition is regarded by Quincke as a vaso-motor neurosis producing sudden dilatations and permeability of the vessels. It is, however, one of the derangements which may be said to be of mixed vaso-motor and trophic origin.

Symptoms.—The face, especially the eyelids and nose, are the most usual sites, but the swelling may affect any part of the body, as the hands, face, or genitalia. Even the mucous membranes may be invaded, especially the lips, mouth, and pharynx, while a fatal œdema of the larynx has occurred. The onset is sudden and the patient's previous health may have been excellent. Gastro-intestinal disturbances, are, however, sometimes associated, manifested by vomiting, colic, diarrhœa, and gastralgia. There is also at times heat, redness, and itching.

Treatment.—Remedies calculated to increase muscular and nervous tone, such as strychnine, quinine, and iron, are indicated. In other respects the treatment is symptomatic, and directed to whatever symptoms demand attention.

Raynaud's Disease.

Synonyms.—*Local Asphyxia, and Symmetrical Gangrene of the Extremities.*

Definition.—A vaso-contractile disease, characterized by three stages, more or less complete, viz.:—

(1) Local syncope.
(2) Local asphyxia.
(3) Local gangrene.

Historical.—The disease was first described by Maurice Raynaud in 1862. Raynaud's thesis, translated by Barlow, with additional cases and bibliography, is published in Vol. CXXI, 1888, New Sydenham Society's publications.

Symptoms.—The disease is more frequent in women—Raynaud's cases including 20 women and five men. It is also a disease of early life; the majority of Raynaud's cases were between the ages of eighteen and thirty, while five were between three and nine. The first phenomenon noted is an unusual pallor or anæmia of the part, resulting in marble-like whiteness and loss of sensation. This is the *local syncope*. Affecting, as it often does, the fingers and toes, these have been called dead fingers and toes. It follows exposure to cold, and in those predisposed, to comparatively slight degrees of cold. The condition disappears under warmth, and then only does pain manifest itself—when the parts are being thawed out, as the saying is. The anæmia is then followed by engorgement, and the parts previously pale become purple and livid, constituting *local asphyxia*. The change is not simultaneous in all the fingers, some being still white while the others are livid.

The local asphyxia may succeed the local syncope, or it may come on independently of it. The tip of the nose and helices of the ears are parts prone to cyanosis, but in addition to the fingers and toes, the hands, feet, and arms and legs may be involved. A peculiar and striking mottling is the result on these large surfaces, produced by an alternation of various shades of purple with intervening lighter-hued spaces. In the darkest areas the capillary circulation is quite stagnant. There are also swelling, resulting stiffness, and pain, the latter often extreme and associated with an intense itching. But in Raynaud's disease there is perhaps more frequently anæsthesia than pain. These are the phenomena, too, of chilblains, with which so many suffer in this climate with the first approach of cold weather. In Raynaud's disease, as in chilblains, these symptoms may pass away in time under the influence of warmth; in fact, for a long time they occur only during the colder weather. A reaction takes place, and the parts assume a bright red color in which the circulation is very active, and the anæmia produced by pressure is rapidly replaced by an active hyperæmia. These attacks may keep recurring for years without further effect, though in extreme cases there may be slight loss of substance in the ear-tips and finger-ends, which in time may become indurated, uneven, and scarred from this cause.

The third stage of *local* or symmetrical *gangrene* is reached in a few cases only. In these the parts affected remain asphyxiated and the phenomena of dry gangrene make their appearance. The fingers or toes, one or more, become black, dry, and cold, while gangrenous blebs appear in the parts adjacent to the sound tissue, a line of demarcation occurs, and the dead part sloughs away less extensively than at first seemed likely to be the case. Rarely, and only in cases of young children, does a fatal termination occur.

These symptoms which have been described may be said to be essential, but others also may be added of great clinical interest. One of these is *hæmoglobinuria*, which is, of course, associated with a corre-

sponding albuminuria. There are at times a few blood discs in the urine. Hæmoglobinuria, when present, generally occurs at the same time with the cyanosis, and the attack has a number of times been preceded by a chill. Other associate symptoms, less common, are scleroderma and œdema, probably angeio-neurotic.

At other times cerebral symptoms, including torpor and partial loss of consciousness, are present; at others, epilepsy, mania, delusions, and even temporary hemiplegia. Dimness of vision is a symptom easily explained, on the supposition that there is a spasm of blood-vessels producing local retinal syncope. Other associated symptoms are peripheral neuritis with tingling and formication—neuritis being regarded as one of the causes of the disease; arthritic swelling; urticaria; erythema; also colicky pains, nausea, vomiting, and diarrhœa.

Pathology.—Three chief theories have been brought forward to explain Raynaud's disease:—

(1) That it is due to endarteritis obliterans.

(2) That it is caused by peripheral spasm; and

(3) That it is the result of vascular spasm.

The intermittent nature of the disease is quite incompatible with its causation by endarteritis, which is progressively increasing in its effects. It is true that some of the results of peripheral neuritis are similar to those of Raynaud's disease, but the frequency of the former affection as contrasted with the rarity of the latter, militates, also, against this view.

The theory of arteriole spasm, suggested by Raynaud himself, best explains the symptoms. Its frequency among women and children, whose vaso-motor system is so impressible; its occurrence under the influence of cold, which is one of the most powerful excitors of vaso-motor spasm; the frequent dimness of vision, which has been shown by ophthalmoscopic examination to be associated with contraction of the central artery of the retina; the occasional precedence of a chill; and the phenomena of hæmoglobinuria, all go to show the probability of vaso-motor spasm. Since the hæmoglobinuria is apt to be associated with hæmoglobinæmia,—which probably arises from the solution of hæmoglobin liberated in the asphyxiated parts,—such an origin for the hæmoglobinuria must be admitted. It will be noted that the conditions in Raynaud's disease are in some respects similar to those of intermittent hæmoglobinuria, and such similarity affords a basis which may ultimately help to explain more satisfactorily the phenomena of both. The relation of Raynaud's disease to chilblains also affords an interesting field of investigation—in fact, has already been studied by Legroux.

Prognosis.—This is not altogether unfavorable. Only delicate and feeble children, as a rule, perish, while it is quite possible, under favorable circumstances, to outgrow the tendency.

Treatment.—Persons subject to local syncope and local asphyxia should be protected from cold, and when the attack comes on they should be kept warm, if necessary in bed, the parts being wrapped in wool, and subjected to artificial heat. Friction may be associated with advantage. Galvanism and faradism are recommended.

B. B. Cates, of Knoxville, Tenn., has recently reported the successful treatment of Raynaud's disease by nitroglycerine in doses of $\frac{1}{100}$ grain (0.00065 gm.) increased to $\frac{1}{50}$ grain (0.0013 gm.) three times a day.

Progressive Facial Hemiatrophy.

Synonym.—*Unilateral Progressive Facial Atrophy.*

Definition.—A gradual progressive wasting of the bony, muscular, integumental, and adipose tissues of half the face.

Etiology.—That it is a trophic neurosis can scarcely be doubted. In one case, that of Mendel, which came to autopsy, there was the terminal stage of a neuritis in all the branches of the trifacial.. In Homen's case, an acute one and perhaps not strictly to be regarded as an instance of true facial hemiatrophy, a tumor was found pressing on the Gasserian ganglion and trigeminal nerve. These facts would go to show that it is a neurosis of the latter nerve rather than the sympathetic.

The disease usually begins in youth, but in a few cases it did not make its appearance until middle age. It is rather more common in the female sex.

Symptoms.—The atrophy is much more frequent on the left side than on the right. It may begin as a circumscribed spot on the cheek or chin, or diffusely, involving first the subcutaneous tissues, the muscles, chiefly those of mastication, and finally the bones, especially of the upper jaw. In the cases which begin in early youth, the muscles remain intact. The tissues of the orbit take part, and the eye appears sunken. The corresponding half of the tongue and soft palate are sometimes involved. The hair on the same side may fall out and appears thin. The line of demarcation is sharp in the median line. In a few rare instances the disease is bilateral, and in a few cases also the atrophy involves the corresponding shoulder and arm. Sensibility is intact.

Diagnosis.—The disease though very rare can scarcely be confounded with anything else. The facial asymmetry associated with congenital wry neck alone resembles it. Strümpell mentions a case of facial *hemihypertrophy* in a boy of ten under his observation.

Treatment.—A suitable treatment is electricity to the atrophic side alternated with massage.

Acromegaly.

Definition.—A disease characterized by enlargement of the bones, especially of the hands, feet, and face.

Historical.—It was first described by Marie, of Paris, in *Revue de Medicine*, 1886. It had, however, been previously described under other names as "hyperostosis of the entire skeleton" by Friedreich, as general hypertrophy or "Makrosomie" by Lombroso, as "giant growth" by Fritsche and Klebs. Since then numerous cases have been reported, and the disease was exhaustively described by Arnold, of Heidelberg, in Ziegler's *Beiträge*, in 1891.

Etiology.—It is a disease of the early adult life, usually under thirty, and is, perhaps, slightly more frequent in women. Heredity,

syphilis, and the specific fevers have preceded the disease, but no necessary relation has been shown.

Morbid Anatomy and Pathology.—This consists in a true hypertrophic enlargement of the bones, except the superior maxillary which contributes to the enlargement of the face by a dilatation of the antrum, while the lower jaw is simply enlarged. As stated, the enlargement is uniform and symmetrical instead of involving only the shaft as in the osteitis deformans, or the ends as in arthritis deformans, and is quite independent of rheumatism. In certain cases also enlargement or atrophy of the thyroid gland, enlargement of the thymus and of the pituitary body have been noted. The changes in the latter Marie sought to make responsible as disease of the thyroid is for myxœdema.

Symptoms.—The most striking features are the enlarged hands and feet, the appearance of the former being well characterized as spade-like, while the fingers and nails are broad. The legs and arms, on the other hand, are not elongated, but late in the disease the forearms and legs may increase in circumference; while the extremities of long bones, like the femurs, are often prominent. The scapulæ, clavicles, sternum, and the ends of the ribs are also sometimes involved. The proper use of the hands is not interfered with. The head and face are enlarged, the latter elongated, while the neck looks short, and the inferior maxilla may project beyond the upper, and lower lip protrude in consequence. The ears are unduly prominent, while the cartilages of the nose, eyelids, and larynx are enlarged and thickened, as is also sometimes the tongue. The spinal column may be involved and there may be kyphosis. The muscles, on the other hand, are sometimes atrophied and the genitalia unusually developed. The skin though coarse and exhibiting a tendency to perspire, is not thickened as in myxœdema.

Among other symptoms are mental dulness, a sense of fatigue, and quite severe pain in the head and extremities; alteration of voice due to changes in the tongue and larynx and possibly to paresis of the vocal cords; impairment of the senses of taste, smell, and hearing; blindness due to optic atrophy; thirst, shortness of breath, asthmatic attacks, palpitation, and even hypertrophy of the heart. There is menstrual derangement and early cessation of the menses in women. The alterations in the thyroid have been alluded to and an area of dulness over the manubrium is ascribed by Erb to persistence of the thymus.

Diagnosis.—This is easy. The difference between acromegaly and osteitis deformans and arthritis deformans has been mentioned. In the former, too, as pointed out by Marie, the face is triangular with the base upward, while in acromegaly it is ovoid with the large end downward. Acromegaly has been mistaken for congenital progressive atrophy or "giant growth," but in the latter only one limb is usually involved and the shaft of the bone is affected.

Prognosis.—The duration of the disease is long and usually ultimately fatal, although it is sometimes arrested.

Treatment.—None has been found to be of any value.

Scleroderma.

Synonyms.—*Cutis tensa chronica; Sclerema; Dermato-sclerosis; Glossy Skin.*

Definition.—A chronic, somewhat diffuse, indurated, hide-bound, and pigmented condition of the skin, tropho-neurotic in origin.

Historical.—The disease was first described by Alibert in 1817. The most recent publication on the subject is "*Die Sklerodermie*," by Lewin and Heller, 1895, in which 508 cases were collected.

Etiology.—This is obscure. It is more common in women than men, and most frequent in early adult and middle age. In one case under my observation, that of a hack-driver, long exposure to wet and cold seemed a likely cause, and others report similar experience. Rheumatism, especially of the joints, and strong impressions on the nervous system have been regarded as causes.

Pathology.—The identity of scleroderma and morphœa is claimed by some. I follow Louis A. Duhring in separating them, because both are capable of assuming a variety of forms which present entirely different clinical features at various stages. Scleroderma is much rarer than morphœa. In the matured forms, while the epidermis is unaltered, there is increase of pigment in the lower layers of the rete, with a distinct overgrowth of connective tissue in the corium and subcutaneous connective tissue. Contrary to what would be expected, the sweat and sebaceous glands appear to be normal.

Symptoms.—The disease appears first in the neck, shoulders, back, chest, arms, and face. It begins usually as a stiffening of the skin which passes over into a hard, tense, unyielding tissue, resisting motion, and causing fixation and flexion. The patient is literally "hide-bound." The hand with its smooth, glossy surface, utterly without wrinkles, is striking and distinctive. The change may involve the greater part of the body and even the whole of it. Where less general, it is symmetrical. The condition passes insensibly into that of the surrounding healthy tissue. Pigmentation is usually a later symptom, but may be an early one.

There is generally no constitutional disturbance or other local symptom such as pain, burning, and tingling, but more rarely these are present. The evolution of the condition is generally slow, requiring weeks and months, and when completed it is apt to remain unchanged for months or years, or slowly passes away leaving the skin normal. Rarely, however, an atrophic state may succeed, producing such a shrinking or contraction that the integument is apparently bound to the bones, while over the joints the skin may become so fixed and immobile that ulcers and excoriations easily form.

Diagnosis.—The diagnosis rarely furnishes difficulty. In some stages it resembles morphœa, from which it will be distinguished when that subject is considered.

Prognosis.—This should always be guarded, as the disease is often intractable, though recovery sometimes occurs.

Treatment.—Treatment of a curative kind is unknown. The patient should be thoroughly protected against cold, as he is exceedingly sensitive. Frictions with oil are a rational means for softening the skin,

and may give comfort, but do not check the spread of the disease. Cod-liver oil, iron, and arsenic are indicated. The constant electrical current has been recommended in the local forms.

Morphœa.

Synonym.—*Keloid of Addison.*

Definition.—A trophic, asymmetrical neurosis of the skin characterized by patches of skin firm in texture, white, pale pink, light yellow or waxy hued, sometimes elevated, at others depressed.

Etiology.—More common than scleroderma, it is also found more often in women, and at all ages. Its etiology is unknown, but its trophoneurotic origin is more than likely.

Symptoms.—The patches occur more frequently about the breasts and neck and sometimes in the course of nerves, such as the intercostal or lumbar, or on the face along the branches of the 5th pair. They range from 0.4 inch (one centimeter) to four inches (ten centimeters) in diameter. There may be a preliminary hyperæmia with itching of the skin and increased pigment deposit or a milk-white leucoderma from the beginning. The spots are dry, without perspiration, sometimes scaly. Ultimately there may be anæsthesia, in pinkish or purplish hyperæmic spots or in small linear cicatricial-like areas, which grow rapidly. In fact, the rapidity of spread of the spots is one of the most interesting clinical features. In the later stages there is often distinct atrophy and cicatrization with pigmentation. The spots may persist for months or disappear in a few weeks, and though more frequently persistent for a long time, they ultimately disappear spontaneously. The spots seem to be the direct result of a cutting off of the circulation by a narrowing of the blood-vessels. This may be by compression by an inflammatory exudate, but is more likely to be a vaso-motor constriction, probably due to irritation of the vaso-constrictor nerves.

Histologically there is a condensation of the connective tissue of the corium with a shrinkage of the papillary layer.

Diagnosis.—Morphœa differs from scleroderma in that its lesions are more circumscribed, and in an absence of sclerodermic hardness. Pigmentation and cicatrization usually appear only in the later stages of morphœa, while they are seen in the early stages of scleroderma before there is change in structure. Scleroderma is symmetrical in distribution, morphœa not. The atrophic striæ seen in one form of morphœa closely resemble the lineæ albicantes of pregnancy or other cause of distention.

Treatment.—That recommended in scleroderma may be expected to be useful in morphœa, especially arsenic which is commended by Duhring. Here, too, the constant galvanic current is held to be of service, an extended trial being, however, necessary.

Ainhum.

Synonyms.—*Ainham; Quigila; Suhka Pakla, or Dry Suppuration; Pityriasis ethiopius; Scleroderma annulare.*

Definition.—A trophic disease resulting ultimately in the amputation of one or more toes, especially the little toe, confined almost exclusively to male negroes.

Historical.—It was first described, in 1866, by da Silva Lima, of Brazil, to which country it was at first thought to be limited, but since then cases have been reported from almost every quarter of the tropical and semi-tropical globe, including the extreme southern United States.

Etiology.—It would seem that a moist, sandy soil and warm climate must have some influence in its etiology, but nothing definite is known. Its practical limitation to the colored race has been referred to. The operation of a pathogenic organism has been suggested and the disease as an amputating leprosy. Traumatism has undoubtedly been associated with it.

Symptoms.—Ainhum begins as a furrow or crack at the digito-plantar fold, seen first on the inner side. In a few days the toe will swell and become the seat of a burning, shooting pain, which may extend into the foot and leg, though pain is not constant. The furrow increases laterally and in depth until finally the toe is constricted and the distal end becomes ovoid. The swelling subsiding, spontaneous amputation ultimately takes place, a dry scab forms at the furrow and the case ends. It is not confined to one toe, though it is as a rule. Sensation is not usually destroyed, though it may be, and the nail remains unchanged. There are no constitutional symptoms.

The histology of the process has been studied by C. H. Eyles, who concludes that there is an ingrowth of epithelium with corresponding depression of surface, due to a hyperplasia that strangles the papillæ and cuts off the nourishment of the epithelium and causes it to undergo horny change. The bone-changes are those of a rarefying osteitis, proceeding from the periosteum inward. According to Collas, it is an amputating leprosy.

The **diagnosis** is easy. There is no disease which resembles it.

The **prognosis** is favorable as to any danger to life. The duration of the disease is from two to four years.

Treatment is unnecessary.

RELATION OF INFECTIOUS PROCESSES TO DISEASE OF THE NERVOUS SYSTEM.

The responsibility of micro-organisms in the causation of disease as acknowledged at the present day has been extended to certain nervous affections which are known to succeed many infectious processes. No study of nervous diseases is therefore complete without presenting, at least in summary, the conclusions which have thus far been reached. As in the case of many of the infectious diseases themselves, it has come to be acknowledged that nervous affections are due more to

soluble toxines developed in the course of bacterial action than to the micro-organisms themselves.

Our knowledge is scarcely definite enough at this time to justify a lengthy consideration of the subject, and I limit myself to a summary of the action of the various infective agents upon the nervous system from a recent paper by James J. Putnam,* which he has arranged under the following headings :—

(*a*) As to the acute (nervous) symptoms of onset of the infectious diseases.

(*b*) As to the symptoms which occur during its course.

(*c*) As to the affections met with during convalescence.

(*d*) As to the remote effects.

Summary of Facts and Conclusions.

Variola.—Toxic symptoms of *onset* often severe ; no characteristic symptoms during *course.* During *convalescence* various forms of spinal disease occur with relative frequency, of which disseminated myelitis and disseminated sclerosis are the most common. Acute poliomyelitis, encephalitis, and neuritis occur, though rarely. Acute ascending paralysis of the type of Landry's disease has been noted. Meningitis due to the primary infection does not occur. There are no characteristic remote symptoms, unless disseminated sclerosis be so considered.

Scarlatina.—Toxic symptoms of *onset* rarely severe ; no characteristic symptoms during *course*, though in some epidemics meningitis may perhaps occur (Bendel) ; nervous affections of *convalescence* rare but important. Optic neuritis may occur apparently without meningitis, and the same may be true of epilepsy (Gowers). Meningitis (except from secondary infection) is rare, but does perhaps occur, even in early stages (Bendel)†. Focal hemorrhagic encephalitis is reported (Fürbringer). Spinal affections and multiple neuritis are rare.

Measles.—Toxic symptoms of *onset* and *course* not severe nor characteristic, though paralysis during this stage may occasionally occur (Lob).‡ During *convalescence*, spinal affections occur with greater frequency than after scarlatina. Optic neuritis sometimes occurs (Wadsworth),§ but it is probably dependent upon meningitis. This meningitis is sometimes slight and temporary. Apart from the secondary form, due to purulent infection, a primary form occurs, though rarely, the pathology of which is uncertain. (See Meningitis Serosa ; Quincke.) Multiple neuritis and protracted psychoses are rare.

---

* American Journal of the Medical Sciences, March, 1895.

† *Wiener Med. Presse*, 1894, No. 23, p. 889.

‡ *Cbl. für Klin. Med.*, 1893, 50.

§ Boston Med. and Surg. Jour., 1880, Vol. CIII, p. 636.

Typhoid.—Symptoms of *onset* rarely severe; those of *course* and occasionally of *onset* often severe and somewhat characteristic. The diseases of *convalescence* consist in psychoses and neuroses (typhoid spine), multiple neuritis, neuritis of single nerves (palatine, optic, and other cranial nerves), transverse myelitis, and poliomyelitis; meningitis, both primary and secondary; disseminated sclerosis. Widespread peripheral nerve degeneration has been found by Pitres and Vaillard, even when no paralysis had been noticed.

Fevers of the Malarial Type.—Toxic symptoms of *onset* and *course* severe and somewhat characteristic. Symptoms of *convalescence*, multiple neuritis; disseminated myelitis and sclerosis; hemiplegia, generally due to pigment thrombosis, and other nervous affections of various types (Browning). All of these are rare.

Diphtheria.—Toxic symptoms of *onset* not severe; paralyses of *course* and *convalescence* characteristic, especially by their distribution. Lesions sometimes not found;* sometimes widespread, and involving the cord and medulla as well as the nerves. Bikeles has found changes in the posterior root zones in an ataxic case. Disseminated sclerosis has been observed as a sequel, though rarely.

Tetanus.—Typical complex of symptoms, which, however, do not come on at once after inoculation. This interval of latency is not occupied by bacterial growth, since it occurs after injection of sterilized cultures. Brunner thinks there is a gradual increase in the irritability of the spinal centres. Lesions sometimes occur in the spinal cord, but are not necessarily co-extensive with the symptoms. There are no characteristic affections of *convalescence.*

Rabies.—Typical complex of symptoms, which are probably toxic in the strict sense, though associated (whether regularly or not is uncertain) with slight, widespread lesions (Golgi) involving the cord, medulla, cerebellum, and brain hemispheres. There are no characteristic affections of *convalescence.*

Influenza.—Toxic symptoms of *onset* often severe. It is rare to see characteristic nervous symptoms during *course,* though optic neuritis and other nervous affections occasionally occur very early. Affections of *convalescence* numerous and important, the chief being multiple neuritis; myelitis, especially of the cervical region; meningitis and encephalitis (focal, hemorrhagic); protracted psychoses; severe neuralgias; papillitis and retro-bulbar neuritis (rare); locomotor ataxia, perhaps usually preceded by multiple neuritis. It is doubtful if, in the case of locomotor ataxia, influenza is more than a contributive cause.

Erysipelas.—No characteristic toxic symptoms of *onset* and *course.* During *convalescence* (though rarely), meningitis, myelitis, multiple neuritis. A few cases of optic neuritis and atrophy (Gowers) have

* See, among recent pathologists, Bikeles in Heft II. of Obersteiner's "*Arbeiten,*" Wien, 1894.

been observed even quite early, and a case of paralysis of the soft palate.

Pneumonia.—Toxic chill at *onset.* During *convalescence,* meningitis, multiple neuritis, optic neuritis (Gowers).

Acute Rheumatism.—Symptoms of *onset* not severe. During *course,* hyperpyrexia, meningitis (rare from primary infection), delirium, coma, convulsions. During *convalescence,* chorea, neuritis.

Syphilis.—No characteristic acute toxic symptoms, strictly speaking, unless neuralgia be so considered. Eventually, typical cerebral, spinal, and peripheral affections due to lesions originating in new growths. Remote effects, of which the pathogenesis is undetermined, consisting in diffuse dorsal myelitis approaching the type of the non-syphilitic form; also locomotor ataxia, lateral and amyotrophic-lateral sclerosis; perhaps nuclear degenerations; general paresis.

Gonorrhœa.—No characteristic toxic symptoms of *onset.* During *convalescence,* myelitis (rare) of unknown pathogenesis, multiple neuritis.

Lepra.—No characteristic toxic symptoms in a strict sense, though the blood and urine are said to contain toxines. Nerve-degeneration excited by presence of bacillus in lymph spaces of nerve and in the nerve-fibres (Babes).

Tuberculosis.—No strictly toxic symptoms, unless the paralyses and cerebral disorders occurring during tubercular meningitis are of that character, which is probable. In the course of the disease, local affections of the brain, cord, and nerves occur, of which meningitis and neuritis are the chief.

Ulcerative endocarditis (due to various bacteria) is associated not infrequently with meningitis.

Pyæmia and septicæmia, including the puerperal form. Violent toxic symptoms of *onset* and *course.* During *convalescence,* multiple neuritis (rare).

Beri-beri and *kindred affections; Landry's disease; Poliomyelitis;* some forms of *multiple neuritis* occurring independently of antecedent disease; *chorea; herpes zoster; amputation neuritis* may be classed as possibly of infectious origin. The evidence for the infectious origin of *epilepsy* and *paralysis agitans* is at present of little weight. (J. J. Putnam "The Relation of Infectious Diseases to Diseases of the Nervous System," Am. Jour. Med. Sci., March, 1895.)

SYPHILIS OF THE NERVOUS SYSTEM.

SYNONYM.—*Syphilis of the Brain and Spinal Cord.*

Definition.—A term applied to the condition and symptoms resulting from invasion of the nervous system by syphilitic disease.

Etiology.—Any one of the various lesions which arise in the tertiary stage of acquired syphilis, or as the result of inherited syphilis, may invade the nervous system. More frequently they are the result of acquired syphilis, but a gummy tumor may develop in the brain of the fœtus in utero. The phenomena of syphilis of the nervous system present themselves, usually some time after the primary infection. Exceptionally only do they present themselves before the end of the first year. More frequently ten to twenty years elapse before the first symptoms appear.

Age and sex have a bearing only as they influence the distribution of the disease. The same conditions which predispose to the development of other brain affections predispose also to the implantation of the syphilitic poison. Such is the inherited nervous disposition, or vulnerability acquired in any way, say through traumatic or septic agencies. Such predisposition favors the earlier operation of the syphilitic poison as well as of other causes of nervous disease. For evident reasons also nervous syphilis is more frequent in the male than the female sex.

Morbid Anatomy.—As stated, any of the tertiary syphilitic lesions may be the cause of nervous syphilis. While the fundamental process in each instance is the same, it may be quite definitely subdivided into two,

(1) Syphilitic new formations.

(2) Syphilitic disease of the blood-vessels.

(1) Includes—

(*a*) Specialized new formations, the syphiloma or gumma.

(*b*) Simple inflammatory products.

I. (*a*) The *syphilitic gumma* or syphiloma is a circumscribed yellow or greyish-yellow mass often caseated in the centre. It may occur as a single focus or in multiple foci. Its most frequent seat is in the dura mater, or the subarachnoid space, whence it invades the brain substance and adjacent vessels and nerves. More rarely it starts *ab initio* in the substance of the brain, when it sometimes closely resembles the tyroma or solitary tubercle. The tendency to degeneration, which results in the caseation referred to, is the characteristic feature of the gummy tumor, but it is associated with another property of extreme importance, a fibroid change at the periphery, which produces the appearance of a capsule about the tumor, although no distinct capsule exists. Such fibroid change may also interpenetrate the tumor itself.

(*b*) Of even greater clinical importance is the *simple inflammatory luetic neoplasm*, which starts in the meninges as a flat tabular swelling—meningitis gummatosa—most frequently at the base of the brain, especially in the neighborhood of the optic chiasm, but also in the fissure of Sylvius and on the convexity. In these more diffuse areas one often finds, alongside of each other, the different stages of young granulation tissue, cheesy foci, and contracting connective or scar tissue. Either

of the two membranes, the dura or pia, may be involved, but in the pia the inflammation is usually circumscribed. Specific inflammation of the substance of the brain or cord is rare.

II. The *intra-vascular syphilitic growth*, starting from the endothelial cells of the intima, produces a firm connective tissue which differs from atheroma in being thicker and more translucent, but which, after internal administration of iodide of potassium, becomes thinner and more opaque, in fact, more like the simple atheroma, for which it is, then, often mistaken. Thickening of the adventitia is also sometimes superadded. It is to be remembered that this intra- and peri-vascular formation presents no distinctive histological features any more than does syphilitic meningitis. The vessels affected are those of the base of the brain, especially the middle cerebral and its branches. The possible results of such intra-vascular growth are *occlusion*, with resulting necrotic softening or induration of the cortex, especially in children; or *intracranial aneurisms* of the larger arteries. Of these Gowers says, "We know two other causes of such intracranial aneurisms. One, very rare, is traumatic arteritis. The other is embolism 'imperfectly occluding the vessels,' probably the cause of two-thirds of such aneurisms before the degenerative period of life. In the remainder, in which there is no history of injury and no evidence of embolism, there is a history of syphilis in so many cases as to justify the opinion that most of them are due to this influence. Often the history is imperfect, because the aneurism has been unsuspected until the final rupture, and syphilis has not been inquired for. When we consider how great is the amount of new growth in the walls of a diseased artery, and how prone are the new elements to change into extensible fibroid tissue, the wonder is that aneurism is not a more frequent occurrence. Probably the explanation is to be found in the common persistence of the elastic layer, which affords the chief safeguard against permanent dilatation."

A third but rare effect of syphilitic vascular disease is *hemorrhage* within the substance of the brain, which is limited by the rarety of syphilitic disease of the arteries within the substance of the brain.

In the *spinal cord* syphilis most frequently produces chronic inflammation of the dura mater, spinal pachymeningitis. Its effect is mainly seen on the spinal and bulbar nerve roots, although the cord also may be invaded by sclerotic fibrous tissue. The pia mater is rarely invaded alone, but may be in association with the dura mater, especially in diffuse inflammation.

Of direct invasion of the *cranial nerves* by syphilitic inflammatory disease as contrasted with the results of compression by tumor or chronic meningeal inflammation, we know very little. It is possible they may be invaded by an inflammation involving the sheath and interstitial tissue.

Symptoms.—Of fundamental importance in studying the symptomatology of nervous syphilis is the fact that the symptoms due to syphilitic disease are in no way distinctive. They are essentially the same as those from tumor or meningitis or occlusion of blood-vessels from other cause. This being admitted, it might seem scarcely necessary to make a separate subject of syphilis of the nervous system. It, however, affords oppor-

tunity for a somewhat more systematic classification of the lesions and a review of the symptoms caused by them.

These symptoms vary with the seat of the lesion, of which sometimes quite a close diagnosis can be made. The specific nature of the lesion may be inferred to a degree from the symptoms, but most largely from the history. From the grouping of the symptoms may be inferred the most important of the local processes as originally suggested by Huebner. They are :—

I. *Basal Brain Syphilis or Basal Gummy Meningitis.*—The symptoms of the flat tabular swelling at the base of the brain are to a certain extent those of a tumor in this section, viz., general cerebral and focal symptoms, due to the local effect of the growth. To these are added features which have a certain suggestive diagnostic value, no more. Such are intense headache, often worse at night, apathy, loss of memory, somnolence, and tendency to stupor, all of which characterize more or less the typical tumor of the brain. Maniacal excitement, ultimate imbecility, and physical weakness may supervene. The compression symptoms due to basal syphilis are also the same as those due to tumor. The nerves concerned are especially the optic and oculo-motor and to a less degree other nerves of the eyeball. The symptoms include narrowing of the field, hemianopia, and even total blindness, alterations in the pupil and defects in the movement of the ball and lids. In syphilis these effects are irregular and seldom bilateral. There may also be optic neuritis, and even choked disc, but the latter is less frequent than in other varieties of brain tumor. Syphilomata always cause a rapid and severe form of optic neuritis, so that chronicity in a neuritis is presumptive evidence against its syphilitic origin. Especially characteristic of syphilis is variation in the severity of the symptoms, due to variation in intra-cranial pressure the result of contraction of the new formation ; and especially characteristic is amelioration, the result of properly timed specific treatment. Other nerves which suffer are the auditory, facial, olfactory, and trigeminal. Gummata on the nerve trunks furnish no symptoms distinct from those of inflammation.

The course of syphilitic lesions is subacute or subchronic, never acute or chronic. Marked improvement, arrest, and even cure may result from proper treatment. On the other hand, involvement of the blood-vessels by syphilitic disease may lead to hemiplegia, epilepsy, and bulbar symptoms of which there can only be one termination—and that a fatal one.

II. *Gummy Meningitis and Syphiloma of the Convexity and Neighborhood of the Fissure of Sylvius.*—In this more rare seat of localization the same prodromata may precede the severer symptoms, sometimes for a considerable time. The latter include, first, general or local epileptiform convulsions which are sudden and succeed each other at longer or shorter intervals. They are of great diagnostic value, it being true of them, as originally said by Fournier, that occurring in subjects over thirty, if not due to uræmia or alcoholism, in nine out of ten cases they are caused by syphilis. They are rarely preceded by an aura. Other cortical symptoms are hemiplegic or monoplegic paresis, cortical derangements of speech, such as motor aphasia and the like, and imbecility. Choked disc,

on the other hand, does not occur in lesions of the convexity. These cases are often fatal. The convulsions continue, consciousness becomes deranged, coma supervenes, and death ensues. Even here, however, a judicious treatment is often of great service.

Syphilitic meningitis is more common at the base of the brain than at the convexity, though gummy tumors are more common in the latter situation. In either place the symptoms of the local meningitis are such as indicate a surface lesion, at the base by implication of the cranial nerves, at the convexity by causing motor phenomena. These signs always point to a superficiality of situations and become thus of value in a topical diagnosis.

III. *Syphilitic Disease of the Walls of the Arteries.*—The characteristic symptoms are those of a sudden focal lesion, and are due to the sudden closure of a vessel, by thrombosis. The most frequent symptom is hemiplegia. Convulsions are exceedingly rare. Hemianopia may sometimes be present indicating disease in the posterior cerebral artery, while lesions may occur in parts of the brain where there can be no focal symptoms, while disease of the basilar and vertebral arteries, sufficient to cause definite symptoms, is seldom survived. In 19 cases out of 20, according to Gowers, the Sylvian artery and its branches are the vessels invaded.

Cerebral embolism causes the same symptoms, but it seldom occurs before forty-five or the age of tendency to degeneration, unless there is valvular heart disease or endocarditis. Under these circumstances, therefore, we can predict syphilis with considerable certainty. Except embolism and injury, sudden hemiplegia occurring between twenty-five and forty-five is very seldom due to any other cause than syphilis. The course of the disease and extent of the paralysis vary as greatly as when caused by embolism. It may be slight and transient, or severe and lasting. All of the symptoms may not occur instantaneously, some hours or days being required to develop them. On the other hand the phenomena of embolism are more apt to be suddenly complete. In thrombosis one stroke may succeed another at short intervals, and this is rather characteristic of syphilis. So is the fact that *consciousness is seldom totally lost.* Giddiness and vomiting sometimes precede it, more often does severe headache, in fact, in more than half the cases. The headache may be general or chiefly on the side of subsequent lesion. It may precede the threatened lesion by only a few days, or a week, often for several weeks, rarely for two or three months. There may be tingling in the side about to be paralyzed. In severe cases death takes place promptly, ordinarily with high temperature.

IV. *Cases of Combined Cerebral and Spinal Syphilis.*—Under these are included cases illlustrating any one of the three forms considered, associated with symptoms of a more widespread syphilitic disease. Thus there may be a combination of the cerebral symptoms described with spinal symptoms, the latter especially often as the result of meningitis of the cervical cord producing paraplegia, hemiplegia, or arm paresis alone, and pain in the course of the spinal nerves. Or the cerebral symptoms may be associated with those of tabes or progressive paraly-

sis, and if so, there is scarcely a limit to the complex of nervous symptoms which may thus arise.

Diagnosis.—A gumma of the cortex and glioma in the same region may produce identical symptoms. So, too, the symptoms due to occlusion of an artery by syphilitic disease may be identical with those due to embolism, in fact there are no symptoms or combinations of symptoms that are not produced by other causes. How, then, shall we know the syphilitic origin? Having first made the topical diagnosis we note any modification of the usual symptoms of meningitis or brain tumor or arterial occlusion, or such additional symptoms as may be due to syphilis. What are these? We have seen that the very sudden onset of symptoms is not usual in syphilis except from occlusion of a blood-vessel. At least a week is usually necessary for their development. On the other hand, they are very seldom chronic. We have seen that choked disc is rare in syphilitic tumors, and that when present it is the result of an acute optic neuritis. We have seen that there is great variation in the severity of the symptoms. We have seen that convulsions, occurring after thirty and not due to uræmia or alcohol, are in nine cases out of ten due to syphilis; that localized spasms, unilateral or more limited, ocular palsies, morbid somnolence, point to syphilis; that hemiplegia in young persons or prior to forty-five years of age not due to embolism from cardiac disease is likely to be due to syphilis. Persistent headache, worse at night, either alone or associated or preceding the palsy, is characteristic of syphilis. Of the utmost value in diagnosis is the therapeutic test,—the effect of treatment. The symptoms are almost invariably relieved by specific treatment. Above all, though not conclusive, is the knowledge of the presence of syphilis learned from the anamnesis, from ocular discovery of lesions, from miscarriages in women, and the presence of syphilides.

Prognosis.—There is scarcely a limit to the possibilities of a timely and appropriate treatment of syphilis of the nervous system and especially of the brain, and though many perish of it, it is generally because treatment has been too long deferred. It is to be remembered, however, where there is actual destruction of nervous tissue as in those forms of degenerative disease due to syphilis, such as tabes, no medicines can restore it. It is only the syphilitic lesion itself which is amenable to treatment, and with its removal comes relief to pressure and irritation on which so many of the symptoms of nervous syphilis depend.

Treatment.—It is evident from what has been said that treatment, to be efficient, must be prompt and early; whence the importance of an early diagnosis. Mercury and the iodide of potassium are almost the sole drugs needed, and at first they should be united. The most effectual way to bring about the mercurial effect is by inunction. The method of inunction, using 30 to 90 grains (two to six gm.) daily, and beginning with the axilla, has been described on p. 180. Simultaneously the iodide of potassium should be administered, beginning with ten grains (0.66 gm.) three times a day, rapidly increasing until some effect is produced. As soon as this effect is noted the dose may be held at a sufficient amount, usually a drachm (four gm.) a day. It is well known that very large doses of the iodide are borne in syphilis without producing iodism.

The mercurial inunction should be kept up for a couple of weeks after the symptoms have commenced to yield, or it may be substituted by the bichloride of mercury in $\frac{1}{12}$ grain (0.0055 gm.) doses three times a day, which may be further reduced and finally omitted. The iodide should, however, be kept up in such doses as are well borne for an indefinite time. Such remedies should be given as are indicated to relieve special symptoms, as phenacetine, antipyrin, and opium to relieve pain. A prompt bleeding is undoubtedly of service at times in apoplectic cases, and is harmless in any case. It should, therefore, be used tentatively in all cases where not contra-indicated by debility, the quantity of blood drawn being regulated by the effect on the symptoms and on the pulse.

Should it happen that the symptoms are totally relieved by treatment, what should be our course thereafter? Certainly not to allow the patient to believe he is permanently cured. For almost inevitably the symptoms will return if such a course is pursued. Doubtless the advice given by Gowers in his admirable Lettsomian lecture is good, that every syphilitic subject, for at least five years after the date of his last symptoms, should have a three weeks' course of treatment twice every year, taking for the time 20 or 30 grains of iodide a day. But it is questionable whether the period should be limited to five years. Better is it to continue the intermittent treatment for the remainder of his days. Especially likely are the symptoms to return in the second half century when the natural tendency to tissue-degeneration sets in.

SECTION XI.

DISEASES OF THE MUSCULAR SYSTEM.

MYOSITIS.

Rheumatic Myositis, Acute and Chronic.

These have been treated in connection with the subject of rheumatism.

Infectious Myositis.

Definition.—A rare form of acute or subacute inflammation of striated muscle, due to unknown infectious agencies.

Morbid Anatomy.—Several cases have come to necropsy. The conditions found have been firmness and fragility of the muscle substance, fatty degeneration, serous infiltration and hyperplasia of inter-fascicular connective tissues. In another case there was hyaline degeneration in varying degree, without involvement of the intermuscular tissue.

Symptoms.—The parts usually involved are the extremities, but the disease may also invade the trunk muscles and heart. There are swelling, with slight œdema, hardness, and stiffness, making motion painful and difficult. Instead of pain, there is rarely paræsthesia. The symptoms resemble those of trichiniasis, insomuch that it has been called pseudo-trichiniasis. In addition to the symptoms named, an erythematous rash, irregularly scattered over the trunk and extremities, is regarded by Löwenfeld as characteristic. It is sometimes followed by slight pigmentation. There sometimes succeeds an atrophy of groups of affected muscles, and Wagner suggested that some of the cases may be examples of acute progressive muscular atrophy. Such cases are hardly fair examples of infectious myositis. The duration of the disease is from three months to three years.

Another form of infectious myositis is acute purulent myositis, sometimes associated with pyæmia.

Progressive Ossifying Myositis.

This is a rare form of myositis, in which the muscles undergo progressive calcification, localized or extending over widespread areas. The disease is more common in males, and usually begins about puberty. It occupies many years in development, and consists in a preliminary inflammatory process, followed by more or less extensive deposits

of bony plates throughout the muscular system, and at times in ossification of entire muscles, with fixation of joints and vertebræ.

Treatment.—No treatment has availed in any of these forms of acute inflammation.

IDIOPATHIC MUSCULAR ATROPHIES. PRIMARY MYOPATHIC FORMS OF MUSCULAR ATROPHY.

In addition to the spinal or myelopathic forms of muscular atrophy described under nervous diseases, there are several varieties of muscular wasting, which seem to reside in the muscles themselves, and which are therefore strictly idiopathic. These forms occur in the young, and follow upon decidedly hereditary disposition. They are all probably the result of a congenital tendency to defective development.

There are several clinical types of primary muscular atrophy, of which the principal are :—

(1) Pseudo-hypertrophy.

(2) Erb's form of juvenile or hereditary muscular atrophy.

(3) The facio-scapulo-humeral type of Landouzy and Dèjèrine.

(4) A peroneal type of progressive muscular atrophy, beginning in the peroneal muscles or intrinsic muscles of the foot.

I. Pseudo-Hypertrophy of Muscles.

Synonyms.—*Pseudo-hypertrophic Muscular Paralysis; Lipomatosis luxurians muscularis* (Heller); *Atrophia musculorum lipomatosa* (Seidel).

Definition.—A condition of muscular paresis associated with atrophy of muscle which is obscured by interstitial fatty overgrowth.

Etiology.—This is especially an affection of childhood, and heredity is an important causal factor, many members of the same family being sometimes affected through several generations. Males are more frequent subjects than females, though the disease is more apt to be transmitted through the mother even though she may not herself be a subject. Heredity is not invariable. The disease usually begins before puberty, though sometimes as late as the twentieth or twenty-fifth year or later. Hysteria, epilepsy, feeble-mindedness, with an occasional anomaly of the skull, have been observed in the same families.

Morbid Anatomy.—No lesion of the nervous system has been found, except accidental and insignificant complications. Minutely examined the muscles exhibit marked differences in the size of the muscular fasciculi, some being wider, many narrower than normal, while there is considerable increase in the adipose and connective tissue between the fasciculi. The fibrillæ themselves are not fatty, and the transverse striation of the muscular fasciculus is everywhere maintained, in which respect this form differs from others in which there is an absolute obliteration of the fasciculi.

Symptoms.—The disease begins gradually with paretic symptoms, without the hypertrophic appearances which are later so pronounced.

A child previously healthy exhibits clumsiness in its movements and insecurity on its legs, being especially awkward in jumping and running up-stairs. Then close examination discovers that certain muscles or groups of muscles are enlarged, the calves of the legs being especially conspicuous. The extensors of the leg, the glutei, the lumbar muscles, the deltoid, triceps, and infraspinales next become enlarged, while the hands, arms, and neck are rarely involved, in strong contrast to the spinal atrophies. Walking becomes more and more difficult, until finally a diagnosis may be made from the gait alone, which becomes waddling, while the shoulders are thrown back, the belly forward, the vertebral column being also arched forward in the lumbar region. The buttocks stand out, and the legs are far apart. In walking the legs are raised slowly, the toes dropping from paresis of the dorsal flexors. Especially characteristic is the child's method of rising up from the floor. He first gets on all fours and raises his trunk by the arms on the floor. The arms are then drawn toward the legs until the knees can be reached, when, with one hand on the knee, he pushes himself up, then grasps the other knee and completes the act of raising himself to the erect position. Late in the disease the same paretic condition may extend to the upper extremities, making it impossible to rise.

The enlargement of the muscles is due to an interstitial deposit of fat, and as a consequence they are soft and flabby, instead of hard and firm as in true hypertrophy. Thus the hypertrophy is truly a pseudo-hypertrophy, the condition being really one of atrophy of muscular substance. Along with this may be associated a genuine atrophy with loss of substance in other muscles, especially in the upper extremities. Very rarely there is a true hypertrophy.

Fibrillar twitchings are rarely present. Electrical excitability is diminished in proportion to the destruction of muscular tissue, but there is never a reaction of degeneration. Sensibility remains normal, and the sphincters are intact. The patellar reflex is sometimes absent. The skin, especially of the legs, sometimes presents a peculiar bluish mottling. As a rule the intelligence of the child is preserved, though sometimes there is mental and moral obliquity.

II. Erb's Form of Juvenile Hereditary Atrophy.

This type is also commonly found before the age of twenty, usually between fifteen and twenty, but its subjects are not as young, as a rule, as those of the pseudo-hypertrophic form. It is also hereditary in families of which female members are affected, while the boys may have pseudo-hypertrophic paralysis. It starts rather more frequently in the upper extremities, the upper arms and shoulders, but may begin also in the back and legs. The following are the muscles involved, according to Erb: In the upper extremities the pectoralis major, latissimus dorsi, and later the triceps; while there remain normal the sterno-mastoid, the levator anguli scapulæ, the coraco-brachialis, the teres major and teres minor, the deltoid, the supra-spinatus and infra-spinatus, and the small muscles of the head, which, it will be remembered, are remarkably wasted in myelopathic

atrophy. The muscles of the forearm, except the supinator longus, remain exempt for a long time, if not altogether. In the lower extremities the glutei, the quadriceps, the peronei, and the tibialis anticus are affected, while the sartorius and calf muscles are spared for a long time.

Very characteristic is the marked projection of the scapula, due to paralysis of the serratus. The gait in this form becomes waddling, and walking ultimately impossible, although, like its congeners, its progress is slow, twenty-three to thirty-eight years being the range of duration of cases described by Erb. Bulbar symptoms are rare, but the diaphragm may atrophy and death be due to respiratory deficiency.

The muscular changes are essentially atrophic, though in the beginning a few of the muscular fibres may be hypertrophied. The interstitial connective tissue is increased, its nuclei proliferated, and there is no interstitial fat. The number of muscle nuclei is also increased and vacuoles may be seen in the individual fasciculi.

III. The Facio-Scapulo-Humeral Type of Juvenile Palsy.

This is also a family form. Duchenne called attention to the fact that in certain children's palsies the muscles of the face are involved in atrophy, but the fact was overlooked until Landauzy and Déjérine opened the subject anew, and showed that this event is not infrequent, indeed, may be the first symptom. It is found still a little later in life, say the twentieth to thirtieth year. In these cases the eyes can no longer be completely closed, and whistling, laughing, and talking become difficult. An appearance characteristic, even diagnostic, known as the *facies myopathique* results, to which the half-closed eyes, the sunken cheeks, and the drooping of the upper lip contribute. The muscles of mastication, the internal ocular, and those of the forearm and hand remain normal. Fibrillary contractions are absent and there is no reaction of degeneration. In other respects it resembles the juvenile form of Erb with which it is closely allied. From what has been said it is very probable that the three forms just described are modifications of one variety, a probability strengthened by the fact that two or more of the types may be present in the same family.

IV. The Peroneal Type of Progressive Atrophy.

Synonym.—*Progressive Neural Muscular Atrophy.*

This form, described by Charcot and Marie, and independently by Tooth, is met with in the second half of childhood, seldom after twenty. It occurs also in families, more frequently in males. It begins in the peroneal muscles, involving also the intrinsic muscles of the foot, and may lead to club foot of the variety pes equinus or pes-equino varus. The upper extremities may be affected after many years, and rarely it begins in the hands. It differs from the other forms of juvenile atrophy in the presence of fibrillary contraction and the occasional presence of the reaction of degeneration, while vaso-motor and sensory disturbances may also be present.

Degeneration of the peripheral nerves has been found with ascending degeneration of the columns of Goll. Both the symptomatology and morbid anatomy of the last form, so far as known from a limited number of autopsies, go to show that it is really a result of neuritis.

Diagnosis.—The diagnosis of the first three forms of juvenile atrophy from myelopathic atrophy has been sufficiently considered when treating of progressive muscular atrophy. The last has distinctive features.

Prognosis and Treatment.—These are also essentially identical with those of progressive muscular atrophy.

MYOTONIA CONGENITA.

SYNONYM.—*Thomsen's Disease.*

Definition.—A hereditary affection, characterized by over-development of muscles and by tonic cramp on attempt at voluntary motion.

Historical.—The disease was first described, in 1876, by Thomsen, a Schleswig physician, in whose family it had been present for five generations. Since then numerous cases have been described in Scandinavia, in Germany, France, and Italy. It is rare in this country and England. In 1889 Hale White made a thorough study of the subject and published his results in Guy's Hospital Reports for that year.

Etiology.—The disease is always congenital, the symptoms making their appearance in early childhood, and in family groups, more frequently in men. A few isolated cases presenting the same symptoms have been described. It is to be regarded as a congenital anomaly of the muscular system.

Morbid Anatomy.—In addition to an obvious macroscopic enlargement, there is also found histologically an evident increase in the volume of the muscular fasciculi, recognized by Erb and confirmed by Hale White, together with intermuscular proliferation of the muscle nuclei, and moderate increase of the connective tissue itself. The heart is exempt, but the diaphragm may be involved. There is no lesion of the spinal cord.

Symptoms.—The disease manifests itself at first in childhood by a stiffness or "mild tetanus," in which the relaxation which necessarily precedes each muscular act is delayed. Voluntary contraction takes place slowly and with difficulty. The arm muscles are also involved, and thus the child's play is interfered with. There is, however, no paralysis, and after motion is started it proceeds with facility. Prompt, rapid, and precise muscular movements are, however, difficult, and military service, for example, becomes impossible. The condition is aggravated by cold and emotion. The muscles are characterized, especially in the extremities, by a voluminousness of development which is in strong contrast to their power. Rarely the facial, ocular, and laryngeal muscles are affected.

Sensation and the reflexes are normal. Rarely there is mental weakness. A peculiar reaction of muscle and nerve to *both* currents is

developed, called the *myotonic reaction* of Erb. The motor nerves show quantitatively a normal faradic and galvanic excitability, and all briefly acting stimuli give short contractions; but with continuous irritation the contractions attain their maximum slowly and relax slowly, while vermicular wave-like contractions pass from the cathode to the anode. The muscles are also faradically easily excited, responding to a fairly strong current always with the above-described prolonged contraction. To galvanic irritation of muscle there is a slight increase of excitability, and to somewhat strong currents the contractions are sluggish, tonic, and continued. They occur only with current closure and not opening. The mechanical irritability of the muscles to strokes from the percussion hammer is also increased.

Diagnosis.—If more is needed than the peculiarity of the muscular phenomena, the electrical and mechanical muscular reactions described are characteristic.

Prognosis.—The disease is incurable, but patients become accustomed to the defect and conceal it as much as possible.

Treatment.—Nothing specific is known. Friction and massage, with muscular gymnastics, are rational measures to be recommended.

SECTION XII.

THE INTOXICATIONS.

ALCOHOLISM.

Definition.—The effect on the human economy of the intemperate use of alcohol in some one of the forms in which it is used as a beverage. Such effect is either acute or chronic.

Acute Alcoholism.

This is the condition known as inebriety or drunkenness. Varying amounts of alcohol are required to produce it, very small quantities sufficing to intoxicate those unaccustomed to its use, while the habitual drinker may consume large amounts without effect.

Symptoms.—The order of symptoms is not always the same. More frequently the primary effect is one of excitement, associated with flushed face and bright eye and loose tongue. To this succeeds the well-known staggering gait of drunkenness, which increases until its subject is unable to walk and finally falls to the ground. The ready speech, at first coherent, now wanders at random, and finally ceases altogether. The stage of narcosis is reached, and the drunken man breathes stertorously in his sleep, his face being congested and breath alcoholic. He may, perhaps, be aroused, and may respond vaguely and incoherently to a question, but soon drops off into sleep again.

In another subject the first stage is much more violent, and he may cry out boisterously, and either spontaneously or upon the slightest provocation inflict injury or even commit murder. In other subjects, again, there is no stage of excitement, and they are morose, or pass gradually and directly into stupor. The stage of incoördination and ultimate stupor is, however, invariable if the quantity of alcohol drunk is enough to bring it about. The effect is upon the cortical nerve cells of the brain.

Other less conspicuous features are a lowered temperature,—96° F. (35.6° C.) to 90° F. (32.2° C.), or even lower,—involuntary evacuations of the bowels and bladder, dilated pupils, and muscular twitchings.

Diagnosis.—The diagnosis of drunkenness is usually easy, yet mistakes are not infrequent; it has been mistaken for apoplexy or apoplexy with fracture of the skull. In the latter case stupor is usually deeper

and the patient cannot be aroused, while the breathing is more stertorous. The subject should always have the benefit of the doubt, and resident physicians in hospitals will often save themselves and the institution they serve much opprobrium if they will remember this. Uræmic coma developing with convulsions also simulates drunkenness and, where the existence of Bright's disease is unsuspected, may cause error. In such the odor of alcohol in the breath is wanting, while that of urine is sometimes present. The pupil is variable in uræmia, being sometimes dilated and sometimes contracted. In all doubtful cases the urine should be drawn by the catheter and tested for albumin.

Chronic Alcoholism.

This is a condition which supervenes sooner or later in individuals who habitually use alcohol intemperately. Intemperance does not always imply the consumption of the same amount of alcohol, smaller quantities producing harmful effects in some persons while larger amounts are apparently harmless in others. The predisposition in some persons to be easily affected organically by alcohol is due to some as yet uncomprehended constitutional weakness. There is reason to believe that the children of alcoholics are not only more susceptible to the degenerative effects of alcohol, but also to other diseases such as gout, rheumatism, syphilis, and diseases of the nervous system. Among the latter may be mentioned especially epilepsy and melancholia, dementia and insanity.

Morbid Anatomy.—If we include under this the numerous morbid states which are directly or indirectly ascribed to the long-continued use of alcohol, such as cirrhosis of the liver, gastritis, low grades of meningitis, and the arterial changes so frequently ascribed to it, a large section of space would be consumed. Fortunately these conditions have already been described as separate entities and their relation to alcohol as a cause discussed.

A few words may, however, be devoted to the consideration of the effect of alcohol on elementary parts, since it is through such effect that its consequences are produced. Some time ago Lionel S. Beale called attention to the destructive effect of alcohol on protoplasm. Quite recently, in 1894, Obersohn, working in the laboratory of Gaule, in Zürich, demonstrated not only that alcohol, ether, and chloroform destroy cellular protoplasm, but also that the cells which are the most complicated, so far as function is concerned, such as nerve cells, are the most vulnerable. These conclusions were confirmed by other experimenters, among them Wilkins, in this country, in 1895, and the whole tendency of experiment and observation at the present day is to show the destructive effect of alcohol on elementary histological units.

Symptoms.—The symptoms of chronic alcoholism are very numerous, and by no means always the same. Unlike those of acute alcoholism they are not confined to any one tissue, as that of the nervous system, but may extend to any. So, too, chronic alcoholism predisposes to other diseases. Its direct effect is, as already stated, mainly on the protoplasm of cells, modifying or impairing its normal metabolism, at times

destroying cells and substituting them by fibroid material, at others inciting to inflammatory action; at others still simply delaying oxidation, as in the case of the adipose vesicle, whose fat remains unoxidized, because its congener, alcohol, is more easily oxidized. In some instances, as in the case of the liver-cells, fat is deposited in new situations because it cannot be sufficiently burnt up. Different kinds of alcoholic beverages also seem to act differently, some, as gin, producing destruction of liver-cells and cirrhosis, and others, as malt liquors, producing fatty livers. It is also true that persons addicted to intermittent debauch are less liable to inflammatory lesions than constant consumers.

The symptoms may be classified according to the systems they invade. Thus we have the effects of alcohol on the—

Nervous System.—The most constant of these is the well-known unsteadiness,—especially of the hands in the performance of muscular actions. It is also apparent in an attempt to protrude the tongue. Gradual mental deterioration is an inevitable consequence, sooner or later, of chronic alcoholism. It is manifested in sluggishness of intellect, in weakness of resolution, a loss of moral character, in irritability, restlessness, and occasionally dementia and insanity. Yet it is surprising how some enormous consumers of alcohol maintain their mental acumen and ability to manage large financial interests, while their vascular and digestive apparatus is evidently the seat of advanced degeneration. When dementia and insanity are present, they are probably due to vascular degeneration and consequent secondary changes in the brain structure. The tendency of such insanity is toward delusions, including suspicion, distrust, fear of impending evil, and more rarely delusions of grandeur, as in general paralysis of the insane.

Multiple and simple neuritis is a well-recognized and almost characteristic symptom of chronic alcoholism, and has already been considered

Pachymeningitis hemorrhagica is sometimes met with. More frequent is slight thickening and turbidity of the pia-arachnoid membrane. But this is not peculiar to alcoholism, being the same as that found in the neuroses of insanity.

Digestive Apparatus.—This is a favorite point of attack in alcoholism. Chronic gastric catarrh is one of its most frequent consequences, producing loss of appetite, nausea, constipation, coated tongue, and foul breath, symptoms which are always worse in the morning, and are temporarily relieved by the dram which the habitual drinker is apt to seek at this time of day. Autopsy in such cases may be negative as to the stomach, or reveal the changes described under chronic gastric catarrh.

Lungs.—Thirty years ago Lancereaux announced that alcoholic excesses are one of the principal causes of tuberculosis, affecting by preference the back of the right lung, while disease of the left in front is the result of insufficient aëration or alimentation; also that such disease is characterized by improvement and general arrest if the patient leave off his habit, and by recurrence if he relapses. So recently as 1895* this

* Effets comparés des boissons alcooliques chez les l'homme et leur influence prédespasante sur la tuberculose, France Med., 1895, xliii., et al.

clinician retracted this view, while the observations of Lagneau developed the remarkable fact that tuberculosis only became prevalent in France after the phylloxera had ruined the vines and curtailed the supply of wine. Then tuberculosis, previously only one-half as common in men as in women, reversed its election, twice as many men being affected as women.

The Liver.—The liver exhibits more easily demonstrable changes, more frequently cirrhosis and contraction, but sometimes also fatty infiltration and enlargement. The interstitial overgrowth so characteristic of cirrhosis is probably secondary to a primary poisonous and destructive effect of the alcohol on the cells as confirmed by the experiments of Weigert, and more recently by those of Obersohn and Wilkins above referred to. In other cases the liver is fatty and enlarged. The compression of the cirrhotic liver on the portal vessels produces secondary effects in hyperæmia of the stomach causing gastric catarrh; of the rectum, producing hemorrhoids, and of the œsophagus, pharynx, and nasal mucous membrane resulting in hemorrhage in any one of the localities; in dilatation of the venulæ of the face and nose, and eruptions on the latter, constituting the acne rosacea or "blossom," by which the toper is so often marked. In many cases, on the other hand, the livers of hard drinkers have been found normal.

Vascular Changes.—These consist in endarteritis, sclerosis, and thickening, followed by atheroma and fragility.

Kidney Changes.—The effect of alcoholism on the kidney is also twofold in the direction of contraction and enlargement, the former due to gradual destruction of renal cells and tubules with substitution of interstitial tissue, the latter to fatty infiltration and hypertrophy. I have often expressed the belief that alcohol is a less frequent factor in the production of interstitial nephritis than was formerly supposed, because of the facilities for its elimination in its long journey from the stomach through the liver and lungs before it reaches the kidney. In this I am sustained by W. Howship Dickinson and the enormous experience of the late Henry F. Formad as Coroner's physician in Philadelphia.* The enlarged kidney of alcoholics was also studied by Formad, who called it the "pig-back" kidney, and found it a true hypertrophy rather than a degenerative change.

Delirium Tremens, or Mania a Potu.

This is a special manifestation of chronic alcoholism, ascribed to the long-continued action of alcohol on the brain, though its occurrence coincides rather with the sudden withdrawal of the alcohol. On the other hand, a debauch by a person previously temperate, however prolonged, is never followed by mania a potu, so that the relation of the illness to the withdrawal of alcohol may be more apparent than real. Purely accidental circumstances may determine the cessation from drinking. It is very frequently an attack of acute illness, especially pneumonia,

* "Heart and Kidney in Bright's Disease," Trans. of the Assoc. of Amer. Physicians, Vol. IV, 1889. Dr. Formad's experience covered as many as 1172 autopsies in a single year.

to which drunkards are especially predisposed. The first symptom is usually sleeplessness associated with intense depression, or there may be intense restlessness, during which the patient, unless restrained, will go out of the house on some imaginary business. To these succeed hallucinations of vision, as the result of which he imagines he is pursued by monsters, serpents, rats, mice, and other vermin. The intense shivering terror of the victim under these circumstances is pitiable, and the "horrors"—a term applied to the disease—is but a feeble expression of the terrors of the patient. Frequently, in his attempts to escape these objects, he is unmanageable and must be confined. Suicide is not infrequent with such patients. At other times the eager though misguided intelligence displayed in watching the imaginary objects is amusing. Auditory hallucinations may be present, and unusual noises complained of. At the same time, even though the patient is violent, the pulse will be found frequent, feeble, and often irregular. There is great muscular weakness, as evidenced by the tremor which accompanies all muscular acts. There is slight fever, 102° to 103° F. (38.9° to 39.4° C.), which is increased if there is intercurrent inflammatory disease.

Prognosis of Delirium Tremens.—If there be pneumonia recovery is a rare event, but if delirium is uncomplicated, recovery generally takes place, certainly from the first attack, and generally even after one or more attacks and a duration of from three or four days to a week. If recovery does not take place the adynamia increases, the pulse grows increasingly feeble, the tongue dry, the delirium becomes muttering, and the patient dies with the usual symptoms of the typhoid state. This event is, of course, more common in hospital practice.

Diagnosis.—This is never difficult. The symptoms certainly resemble those of meningitis, and meningitis sometimes also is present, but with the history of the case and the general appearance of the patient a mistake is not likely to be made. It is most important, however, to examine each case physically, as pneumonia is so frequently associated with delirium tremens and constitutes its most serious danger. Again, a pneumonia of the apex is sometimes accompanied by delirium similar to that of delirium tremens.

Treatment of Alcoholism.

Acute alcoholism rarely requires any treatment except restraint from the further use of alcohol and opportunity to sleep off the debauch. A full dose of chloral—15 to 30 grains (one to two gm.)—may be necessary where there is extreme excitement. Morphine is indicated, but as alcoholics sometimes have contracted kidney, caution should be exercised in its use. In cases where the subject is not too drunk to swallow, half a drachm (two to four c. c.) of aromatic spirit of ammonia often acts happily; and where there is reason to believe that alcohol or undigested food is in the stomach, an emetic of warm water and mustard—a heaped dessertspoonful to half a pint (250 c. c.)—may be given and the stomach washed out. Should it be deemed desirable that an emetic be given to one unconscious, apomorphine hypodermically administered is the best,—$\frac{1}{10}$ to $\frac{1}{6}$ grain (0.0066 to 0.011 gm.).

The first step in the treatment of *chronic* alcoholism is the withdrawal of the poison. Except where mania a potu is present this may be total. Nothing is to be gained by gradual withdrawal, while it only prolongs struggle. No aids like morphine or chloral or cocaine should be used, as to do so is simply to substitute one evil for another and to weaken the resolution of the victim. The bromides may, however, be availed of, and trional, chloralamide, and sulphonal may be employed to procure sleep. Not less than 15 grains (one gm.) of any of these drugs should be administered for an adult, while twice the dose may be necessary. Hydrobromate of hyoscine is often an admirable remedy to quiet excitement. It may be given in doses of $\frac{1}{96}$ grain (0.0007 gm.). Attempts at reformation are rarely successful, but success is not impossible. Some means of restraint is usually indispensable, and as a rule can only be secured in an institution. Unfortunately, relaxation of this is apt to be followed by a relapse. The difficulties increase in the presence of hereditary tendency. An abundance of nutritious food should be insisted upon, as it is found to be the best substitute for alcohol, while tea and coffee may be allowed freely, having the advantage of being stimulating without intoxicating. Tonics, such as strychnine $\frac{1}{30}$ grain (0.0022 gm.) three or four times a day, or quinine, should be administered.

As to the remainder of treatment it must be mainly symptomatic, directed to the symptoms as they arise. Neuritis, one of the most important of these, has been elsewhere considered.

Still another drawback is the intense depression which succeeds the exciting effect of alcohol and often impels to a return to its use. To cure this longing which he ascribes to gastritis and a peculiar irritation of the gastric nervous supply, Zedekauer recommends :—

℞. Chlorated Water, ... 2 drachms (8 gm.)
Decoction of Althæa, ... 5¼ ounces (165 ")
Cane Sugar, ... 2 drachms (8 ").
M. et S.—A tablespoonful every two or three hours.

This, he says, relieves the unpleasant sensation and longing for drink, and restores the appetite.

Various means have been at different times resorted to with the object of disgusting the victim with alcohol. C. Carter strongly commends atropine, because of its physiological antagonism to alcohol. He says if small doses—less than $\frac{1}{100}$ grain (0.00065 gm.) of atropine be administered hypodermically three or four times a day to a victim of the alcoholic habit, it will produce a great distaste for alcoholic liquor in from one to five days. Whiskey will become repellant both to sight and odor, and will have a most intolerable taste, resembling that of turpentine or benzine. If under these circumstances drinking is still attempted, nausea and vomiting follow without the addition of apomorphine or other emetic to the liquor.

Treatment of Mania a Potu.

The first indication after withdrawal of the alcohol is to secure sleep. For this purpose the soporifics named above scarcely suffice,

though they may be tried in the full doses specified. Especially may we hope to obtain some result from the hyoscine in doses of $\frac{1}{96}$ grain (0.0007 gm.) given hypodermically. In many cases of delirium tremens it is scarcely possible to do without morphine, which may be given in ¼ grain (0.0165 gm.) doses, caution being observed not to repeat too often. Chloralose may be given in ten-grain (0.65 gm.) doses, dissolved in warm water; it has the advantage of small doses, equal in effect to the largest of chloral, while it also diminishes tremor, and has no harmful secondary effects. R. Bellamy gives 20 grains (1.3 gm.) of trional, mixed in water, with ten minims (0.65 c. c.) of tincture of capsicum, after a calomel purge. A very hot bath is given, of which the temperature is gradually lessened. If in thirty minutes the delirium shows no sign of abatement, ten grains (0.65 gm.) are again given. In all cases forced feeding in small quantities often repeated is practised, the diet consisting of milk, eggs, and soups. The fluid extract of ipecac has recently been recommended by W. F. Waugh, of Chicago, *to produce sleep*, in 20 to 30 minim (2.6 to two c. c.) doses in water, followed by dorsal decubitus for at least five minutes to avert nausea. A cold bath sometimes has a tranquilizing effect, especially if there is fever, or even sponging the body throughout. Many things must be done to keep the patient occupied, because, after all, the treatment amounts for the most part to a conflict between the patience of faithful attendants and the irrepressible and, at times, almost maniacal desire of the patient to get away. In preventing this it may sometimes be necessary to confine him to the bed, but all gentleness should be exercised in carrying out this measure. It is much better to use a folded sheet than the unsightly straps which are sometimes used in hospitals.

I have said that alcohol may be withdrawn at once. Some object to this because of fear of resulting adynamia, but should it happen that there is great weakness, as indicated by frequent and feeble pulse, it is much better to stimulate with the aromatic spirit of ammonium, digitalis, and strychnine, ½ drachm (two gm.) doses of the first, ten minims (0.65 c. c.) of the second, and $\frac{1}{30}$ grain (0.0022 gm.) of strychnine being given every three hours to overcome such weakness. Even larger doses may be demanded by emergencies. A nourishing food, in easily assimilable shape, repeated at short intervals, should be insisted upon as the best substitute for alcohol. With the first sound sleep comes, usually, relief, and the patient awakes convalescent, unless, as already said, the mania is accompanied by acute disease, like pneumonia, when death is apt to be the termination, whatever our efforts.

THE MORPHINE HABIT.

SYNONYM.—*Morphinism.*

Definition.—An irresistible craving for morphine, which is commonly used in gradually increasing daily doses to meet the demand. Periodic attacks or "morphine sprees," comparable to alcohol sprees, during which large quantities are used for the time being, also occur.

Etiology.—The morphine habit is most frequently acquired as the

result of long-continued administration of morphine, by a physician's order or otherwise, to relieve some suffering caused by a painful or incurable malady, or for insomnia. The influence of heredity in favoring the formation of the habit is acknowledged. Neurotic persons are more apt to become its victims. The victim of alcohol often becomes a morphine fiend, being deluded by early experience with the drug to believe that he can thus overcome the previous more disgusting, if not more terrible, habit. The same is true of cocaine.

Symptoms.—The chief symptom is, of course, the *craving for morphine*, but it brings with it others which are more or less temporarily relieved by a dose of the drug. Among these are *irresolution* and *loss of self-control, and a moral obliquity*, similar to that induced by alcohol, especially in women, who are the most frequent subjects. *Untruthfulness*, especially with regard to the drug and the quantities used, is habitual. Epigastric *pain* or *nausea*, or both, are frequently complained of toward the time when another dose is due, though whether this is actual or feigned is not always easily determined. *Mental depression* is a more constant and characteristic symptom, associated with *intense anxiety, restlessness*, and a *sense of impending evil*, both relieved for a time by the dose. All of these symptoms are increased by a more prolonged withdrawal of the drug, when the mental depression becomes intense, sometimes impelling to suicide. So far from the usual constipating effect of morphine being produced by the drug thus used, diarrhœa is not infrequent.

As the habit is prolonged, *tremor*, *paresis*, and more rarely *ataxia* are superadded, while diffuse and neuralgic *pain* is complained of. *Sleep* is irregular, digestion is bad, and appetite and nutrition fail, the pulse becomes feeble and rapid, vaso-motor derangements appear, as shown by a tendency to sweating and by dilatation of the pupils. Except when under the direct influence of the drug, the patient grows weak and becomes a ready victim to acute disease.

On the other hand, the opium eater sometimes attains an old age, presenting a wizened, sallow appearance quite characteristic. The quantities consumed are often enormous, as much as 400 grains (25.65 gm.) daily dose being reported. The pleasurable effect so often ascribed to opium is rarely realized, though it is not unlikely that a certain amplification and distortion of actual facts which may arise in the dreamy state may form the basis of such weird and beautiful fancies as are pictured by De Quincy.

Diagnosis and Prognosis.—The diagnosis is easy, but the prognosis exceedingly uncertain because of the difficulty in carrying out treatment.

Treatment.—Successful treatment is scarcely possible outside of an institution, and even within one, serious difficulties beset the way, chief of which is the deception practised by the patient. Patients should be divested of their own clothing and put to bed in hospital garb, because in this way alone can we be sure that morphine is not concealed about the person. In the case of women, whenever possible, a separate nurse should be assigned to each case. The latest testimony favors complete and sudden withdrawal of the drug as furnishing a short struggle, though

a severe one. Such treatment is usually followed by diarrhœa, vomiting, and insomnia. Some counsel even that no adjuncts should be employed, but certainly there can be no harm in the employment of general tonic treatment and remedies directed to the irritability of the stomach and torpor of the liver. A calomel purge is useful at the start. It is a well-established fact that, as in alcoholism, the patient should be well nourished, given such food as milk, cream, beef juice, or beef peptonoids, rich broths, and beef teas. Where there is great asthenia, aromatic spirit of ammonium, strychnine, and digitalis may be given as directed under alcoholism. If possible, an occupation of an absorbing kind should be furnished. In most cases it is impossible to secure the consent of the patient to sudden and complete withdrawal, when the gradual plan must be adopted. The success of either plan depends on securing effectual control of the patient, and if this cannot be obtained all efforts fail.

To promote sleep one of the numerous hypnotics of which the present day is rich should be given. Chloralamide is probably the best of these. It is not easy of administration, because of pungent taste and difficult solubility. Ten grains (0.66 gm.) or 20 grains (1.33 gm.) are a moderate dose, and are easily soluble in a fluid drachm (3.7 c. c.) of a mixture of two parts alcohol and one part glycerine. Of such solution two teaspoonfuls should be given in a glass of sherry wine or four tablespoonfuls of milk at the ordinary temperature. Trional and sulphonal or somnal may be given in 15 to 20 grain (one to 1.3 gm.) doses dissolved in hot water. Hyoscine in doses of $\frac{1}{96}$ grain (0.0007 gm.) may also be tried. Chloral itself may be used in doses of from ten to 30 grains (0.66 to two gm.). If there is cardiac weakness the dose should not exceed ten grains (0.66 gm.). Chloralose may be given in ten-grain (0.66 gm.) doses in wafers or in hot milk. Too much carelessness is practised by physicians in placing morphine in the hands of patients to be used at their pleasure. The hypodermic syringe has wrought untold mischief and should never be placed in the hands of patients. On the other hand, when morphine is judiciously ordered for patients suffering extreme pain only, it is very rarely the case that a habit is established.

CHLORALISM.

Definition.—The habitual use of chloral or the chloral habit.

This habit is sometimes acquired when the drug is used to obtain sleep or prescribed by the physician for any purpose.

Symptoms.—For symptoms and treatment of *acute chloral poisoning* see page 1163.

The presence of the *chloral habit* is characterized by nervousness, mental weakness, and depression of spirits, even to a degree of melancholia. There may also be general weakness characterized by muscular tremor and cardiac palpitation. These symptoms are aggravated by sudden withdrawal of the drug. There is sometimes dyspnœa aggravated at meals or after exertion. Mania and dementia are reported.

Various skin eruptions or a tendency toward them are a symptom.

Though there may be no eruption, the slightest exertion or a glass of wine will produce an intense erythematous redness on the face and elsewhere on the body. This erythema, which may also extend to the mucous membranes, is ascribed to vaso-motor weakness. There may be diarrhœa from the same cause.

Treatment.—Treatment requires the gradual withdrawal of the drug and cardiac stimulation by ammonia and digitalis, the use of good food, tonics, massage, and electricity. For insomnia, if needed, sulphonal, trional and somnal administered as above directed are more suitable than chloralamide. In extreme cases morphine may be used. It is not usually difficult to master the habit.

COCAINISM.

Cocainism has become a comparatively frequent modern habit. It is especially common among physicians, some of whom acquire the habit in tentative local applications to their own mucous membranes in the treatment of patients. I have known three successive chiefs of clinic in throat and nose dispensary service to acquire the habit. It is also taken as a substitute for some other drug habit, and its subjects are very apt to be those with neuropathic tendencies.

Symptoms.—The effect is a total demoralization of the individual, who loses all moral responsibility, delaying and neglecting appointments in the most remarkable manner. There is volubility of tongue, suggesting alcoholism, and the presence of hallucinations, which also resemble those of the alcoholic effect. The eyes are bright and pupils dilated. The subject becomes suspicious, charging his wife with infidelity, and his best friend with persecuting him. Hallucinations of hearing, sight, and smell are sometimes present, including tinnitus aurium. Mild epileptoid seizures, with partial loss of consciousness, may occur, limited to muscle groups, as about the eyes. Nystagmus is also a symptom. The pulse becomes weak and feeble. The symptoms are often associated with those of alcoholism and opium.

Treatment.—If uncomplicated, treatment is promising. It mainly requires withdrawal of the drug, which should be total. The assistance of a trusty nurse or friend may be needed, but it is not often necessary to remove the patient to a sanitarium. I am speaking of uncomplicated cases. Cases complicated with alcoholism or the opium habit are more difficult to handle, and an institution becomes necessary.

Tonics of the usual kind, strychnine, in full doses, and quinine, should be ordered. Non-intoxicating stimulants, like ammonia and coffee, should be given to counteract the depressing effect, while good, nourishing, easily assimilable food is necessary.

LEAD POISONING.

SYNONYMS.—*Colica pictonum; Plumbism; Saturnism; Devonshire Colic.*

Definition.—A disease of manifold symptoms resulting from the toxic effect of lead on the system, having its subjects mainly among workers in lead works, painters, glaziers, and plumbers.

Etiology.—The lead enters the system by inhalation, the digestive tract, or by the skin. Almost without exception the cases I have had in hospital were from the lead works in the neighborhood of Philadelphia. The Philadelphia Hospital is almost never without one or more such cases. Even animals in the neighborhood of lead works are said to have had the disease, and birds which have been fed on berries grown in the vicinity. Water which has been kept in lead tanks, or even painted tanks, or water passed through lead pipes, has produced the disease. It must, however, be very pure water, such as rain-water, and it is the very impurities of our drinking waters which protect us. Almost all drinking waters contain sulphate of lime, the sulphuric acid of which combines with the superficial layer of lead and forms an insoluble coating of sulphate of lead which prevents further solution.

Accidental contamination has been caused by the use of cosmetics and hair dyes. To the use of baking powder containing chrome-yellow was traced a number of cases occurring lately in Philadelphia in a very interesting study by Dr. D. D. Stewart.* Even the use of vegetables canned in tin vessels is held to have produced lead poisoning.

All grades of what is known as tin, which is really iron coated with a layer of tin, contain a small quantity of lead; and the more inferior the tin the larger the quantity of lead. Certain conditions favor the solution of this lead. Thus, if any of the vegetable acids, as acetic, tartaric, or citric, be present, they may dissolve the lead and form soluble salts, which are readily absorbed. Of course, such solution is favored by prolonged action of the acids; hence old canned vegetables are more dangerous than those recently canned, and it would be a wise measure to insist that canned fruits and vegetables should be stamped with the date of the canning. The solder used in closing the cans may also be a cause of poisoning from this source. When it is considered how enormous is the consumption of canned fruits and vegetables, and how few the cases of lead poisoning traceable to it, it is evident that even moderate precautions may suffice to remove the danger altogether.

Among the more rare causes of lead poisoning may be named materials used in making rag carpets,† cooking in badly-glazed crockery-ware, beer drawn through lead pipes, or beer, cider, and wine from bottles which have been washed with shot of which some have been left behind, the use of snuff packed in spurious tin-foil containing lead, and from sleeping on mattresses the hair in which was dyed black by some

* Philadelphia Med. News, June 18 and December 21, 1887.

† A very interesting case thus caused is reported by Drs. J. Milton Miller and G. Oram Ring, in the Amer. Jour. of the Med. Sciences for February, 1896, p. 193.

lead-containing substance; and one, a most incredible case, mentioned by Naunyn, is that of a proof-reader who was poisoned after many years' reading of printed proof. Notwithstanding the solubility of the acetate of lead so much used in medicine, it is very rare that poisoning has resulted from its administration, and there need be no fear of using it for the purposes to which it is indicated until at least a couple of drachms have been given.

It has always been an interesting question, how lead in the system operates to produce its peculiar effects. That the lead itself lodges in the tissues is easy of demonstration, and analysts have gone so far as to determine the exact quantity in the different tissues of animals poisoned by lead; which, by the way, is surprisingly small, the largest amount found being ¼ of one per cent. in the bones, while that in the muscles was but 0.002 to 0.003 of one per cent. On the other hand, it would seem that lead is contained in the tissues of many persons who are healthy; according to J. J. Putnam, in 25 per cent. It was formerly the custom to ascribe the symptoms of lead poisoning in part to the direct action of lead in the tissues; the cramps and the palsy to the presence of lead in the muscular fasciculi; the colic to the lead in the unstriped muscular fibre-cells, and the nervous symptoms to the lead in the nerve-centres. In part, too, these phenomena were ascribed to anæmia of the tissues, due to the contraction of the arterioles, induced by the presence of lead in the muscular coat. More recently, however, the soundness of these views has been shaken by some experiments of Heubel, and the tendency appears to be to ascribe the phenomena of lead poisoning to neuroses, which are undoubtedly central in the case of the encephalopathic phenomena, and which are probably central in that of the peripheral symptoms. On this supposition, the phenomena of lead poisoning have been compared to those of chronic alcoholism, which are ascribed to effects upon the nerve-centres, due to the long-continued circulation through them of blood charged with alcohol.

Most cases occur among adults, usually between thirty and forty, but in children occasionally. Women are said to be more predisposed than men, as four to one, and to be more readily brought under its influence.

The period of exposure necessary to produce lead poisoning varies greatly, from a month to many years.

Morbid Anatomy.—This is not striking. Tissue may contain a considerable amount of lead without exhibiting changes. Fatty degeneration and fibrosis are, however, characteristic. Thus, the muscles become fatty and fibroid. The kidneys gradually lose their parenchymal cells and become fibroid, while nerves exhibit fatty degeneration. In the spinal cord are found in chronic lead poisoning the changes characteristic of anterior poliomyelitis, *i. e.*, sclerosis of the anterior cornua, with atrophy of the cells and nerve-fibres, but the remainder of the cord and nerve-roots are not altered. Changes in the central nervous system, even when there are symptoms of lead encephalopathy, are not numerous. In 32 out of 71 cases, Tanquerel found none. Von Monkalow discovered a high degree of atrophy, especially marked over the frontal regions, at the

vertex, and the crurà cerebri. Small hemorrhages in various parts of the brain, and atheroma of the arteries, have been noted; also overgrowth of connective tissue. Severe entero-colitis has been found in acute cases.

Symptoms.—While the symptoms which make known the presence of lead poisoning are often rapid in their development, and sudden in their occurrence, there seems scarcely on this account sufficient reason for dividing them into two classes of acute and chronic.

The most striking of the symptoms, and often the first to which attention is called, is *colic*. Indeed it, with the next to be considered, is often the sole manifestation, of the disease, and from these alone a diagnosis may be made, if a patient has been exposed. The term lead colic has long been a recognized term in medical terminology. It is most frequent in the region of the umbilicus, and is often relieved by pressure. It varies greatly in degree, being sometimes a simple grumbling pain, at others of frightful severity, the patient writhing in the paroxysm, which, as a rule, does not last long, but is soon followed by another; but it may continue for hours or until relief is afforded by treatment. It is probably due to powerful contractions of the muscular wall of the intestine, by which the nerve-filaments distributed through it are compressed. Unlike flatulent colic the abdomen is not distended but flat, and may even be contracted, sometimes so much so that it is said that the vertebræ may be discerned through the abdominal walls. Still, distention of the abdomen is occasionally present. The pulse during the attacks of colic is often strikingly slowed, having been noted as slow as 30 per minute.

Groups of muscles anywhere, and especially the flexor muscles, as of the arms and legs, become involved in cramp, the latter more frequently. There may also be cramps in the fingers and toes. In addition to these painful cramps, which, like the colic, are intermittent, there is pain in the neighborhood of the joints. The sum of these painful joints and muscles has received the name *arthralgia saturnina*. They are quite frequent, occurring, according to statistics of Tanquerel, in 755 out of 2151 cases.

Constipation is very common, even more constantly present than the colic, and yet it is not invariable, and may even be substituted by diarrhœa.

A *blue line* on the patient's gums is a very characteristic symptom, and appears at the border of contact of the gums with the teeth, or just above it. As a rule it is easily recognized when present. It is caused by the presence of sulphuret of lead, produced by the action of sulphuretted hydrogen upon the lead in the tissues of the gums. Hence the line is more common and distinct on the gums of those who take no care of the mouth, and in whom sulphuretted hydrogen is generated in the decomposition of the food. This line often remains after all other symptoms have subsided, and although it is not invariably present, its disappearance may be considered a pretty sure sign that the lead has been practically eradicated.

Anæmia is a very constant symptom in lead poisoning, and its higher degrees are attended by a sallowness which early gave rise to the term *icterus saturninus*, but which is in no way due to a deposit of bile pigment. In more serious cases, too, the impaired nutrition results in an

purgatives and diuretics may aid the elimination. The iodide of potassium is the remedy most relied upon to eliminate the lead from the system. It is believed that after its absorption into the system the lead becomes intimately united with the albumin of the tissues, forming an insoluble compound; that the iodide of potassium, after its absorption, combines with the lead, and forms a soluble iodide of lead, which is dissolved out, reënters the circulation, and is passed out with the urine and fæces. It is evident that elimination by these channels will be encouraged by purgatives and diuretics. It is even suggested that acute lead poisoning may be produced by the liberation of the soluble lead-salt into the blood in this way. Hence, caution is suggested in the use of the iodide. Practically, I can scarcely conceive this to occur with such doses as are ordinarily given, ten grains (0.66 gm.) three times daily, after a while reduced to five (0.33 gm.), but this dose should be kept up indefinitely. Iodide of potassium is more efficient when given fasting and freely diluted.

For the paralyzed muscles faradic electricity is indicated and should be daily applied, both to resist the tendency to atrophy and to overcome it.

That restorative and blood-making remedies, in the shape of nutritious, easily assimilable food, together with iron, should also be given to antagonize the cachexia which is always a part of plumbism, is evident. In view of the nervous and muscular symptoms which enter so largely into the disease strychnine may be expected to be a useful adjunct to our treatment, and it is generally so considered. It should be given in full doses, $\frac{1}{30}$ grain (0.0022 gm.) three times a day, and increased to $\frac{1}{20}$ (0.0033 gm.), which should be kept up. Ergot is said to have been useful in restoring the power of muscles involved in the palsy.

ARSENICAL POISONING.

Acute Arsenical Poisoning.—Acute arsenical poisoning is usually the result of an accident or intentional ingestion of Paris green or "Rough on Rats," prepared and sold for the destruction of rats, mice, vermin, and insects.

Symptoms.—These are intense *abdominal pain*, at first gastric, with *vomiting*; later intestinal, with *diarrhœa* and *tenesmus*, which may be followed by collapse and death. The symptoms are not unlike those of cholera, including rice-water stools, cardiac weakness, and cyanosis. Sometimes a skin eruption makes its appearance, sometimes blood and albumin in the urine. Fatal cases terminate in one to two days.

Recovery from these acute effects may be followed by paralysis.

Treatment.—The victim of acute arsenic poisoning rarely needs an emetic, as the arsenic itself induces vomiting. Should free emesis not occur, mustard or sulphate of zinc, ten to 30 grains (0.0648 to 0.1944 gm.) should be administered, and the stomach well washed out by draughts of warm water. With the emetic or before it the antidote should be administered. The best antidote is freshly precipitated sesqui-

oxide of iron, which forms, with arsenic, an insoluble compound. It must be freshly prepared, taking any of the sesqui solutions of iron, preferably the chloride, and neutralizing it with sodium carbonate or magnesia. The precipitate, being hastily washed by emptying on a muslin or filter, pouring water on it and allowing it to drain, should be freely administered. Dialyzed iron may be used, but it is best also precipitated before use with ammonia or other alkali. In extreme cases the tincture of the chloride of iron, Monsel's solution, or any of the sesqui preparations, may be substituted for the precipitated sesquioxide.

After the emetic has acted, and while the antidote is being given, castor oil should be administered to carry off the poison from the bowels.

Chronic Arsenical Poisoning.—This is ascribed to wall papers covering occupied apartments, sometimes to arsenic long administered as a medicine, to artificial flowers, and clothing fabrics. The glazed green and red papers are those especially dangerous. Occasionally, arsenic medicinally administered may produce the symptoms of slow arsenical poisoning.

Symptoms.—Chronic arsenical poisoning may be suspected in the presence of unexplained anæmia and debility, irritation of the conjunctiva, mouth, pharynx, and lower digestive tract, numbness, tingling, and gastralgia; also nervous symptoms and altered nutrition to special parts. All these symptoms may, however, be produced by other causes. Paralysis may also ensue, resembling that of lead palsy, but affecting rather the lower extremities, especially the extensors and peroneal group, whence may arise the characteristic *steppage gait* of peripheral neuritis. Deranged electrical reaction may be present before any loss of power, but on differential examination a weakened power of wrist extension and feeble power to spread the fingers may be detected.

Treatment.—The patient should be removed from the exposure and symptoms treated as they arise. The iodide of potassium may be used.

PTOMAÏNE POISONING.

Ptomaïne, from the Greek πτῶμα, a cadaver, is a word suggested by the Italian toxicologist, F. Selini, for substances generated in the decomposition of organic matter, which more recent studies have shown to be the result of bacterial action. Ptomaïnes are basic, uniting with acids to form salts. Leucomaïnes are similar basic substances formed in the living body. Ptomaïnes differ greatly in their character and properties, certain ones being intensely poisonous, others harmless. For the former L. Brieger suggested the name toxins, retaining that of ptomaïnes for the non-poisonous basic products; but, as Victor C. Vaughan suggests, there are difficulties in the way of such classification because a ptomaïne may be poisonous under certain conditions and harmless under others.

Leucomaïnes are more usually harmless, though they also under certain conditions may produce disease.

Among ptomaïne poisons are the agencies which are responsible for

purgatives and diuretics may aid the elimination. The iodide of potassium is the remedy most relied upon to eliminate the lead from the system. It is believed that after its absorption into the system the lead becomes intimately united with the albumin of the tissues, forming an insoluble compound; that the iodide of potassium, after its absorption, combines with the lead, and forms a soluble iodide of lead, which is dissolved out, reënters the circulation, and is passed out with the urine and fæces. It is evident that elimination by these channels will be encouraged by purgatives and diuretics. It is even suggested that acute lead poisoning may be produced by the liberation of the soluble lead-salt into the blood in this way. Hence, caution is suggested in the use of the iodide. Practically, I can scarcely conceive this to occur with such doses as are ordinarily given, ten grains (0.66 gm.) three times daily, after a while reduced to five (0.33 gm.), but this dose should be kept up indefinitely. Iodide of potassium is more efficient when given fasting and freely diluted.

For the paralyzed muscles faradic electricity is indicated and should be daily applied, both to resist the tendency to atrophy and to overcome it.

That restorative and blood-making remedies, in the shape of nutritious, easily assimilable food, together with iron, should also be given to antagonize the cachexia which is always a part of plumbism, is evident. In view of the nervous and muscular symptoms which enter so largely into the disease strychnine may be expected to be a useful adjunct to our treatment, and it is generally so considered. It should be given in full doses, $\frac{1}{30}$ grain (0.0022 gm.) three times a day, and increased to $\frac{1}{20}$ (0.0033 gm.), which should be kept up. Ergot is said to have been useful in restoring the power of muscles involved in the palsy.

ARSENICAL POISONING.

Acute Arsenical Poisoning.—Acute arsenical poisoning is usually the result of an accident or intentional ingestion of Paris green or "Rough on Rats," prepared and sold for the destruction of rats, mice, vermin, and insects.

Symptoms.—These are intense *abdominal pain*, at first gastric, with *vomiting;* later intestinal, with *diarrhœa* and *tenesmus*, which may be followed by collapse and death. The symptoms are not unlike those of cholera, including rice-water stools, cardiac weakness, and cyanosis. Sometimes a skin eruption makes its appearance, sometimes blood and albumin in the urine. Fatal cases terminate in one to two days.

Recovery from these acute effects may be followed by paralysis.

Treatment.—The victim of acute arsenic poisoning rarely needs an emetic, as the arsenic itself induces vomiting. Should free emesis not occur, mustard or sulphate of zinc, ten to 30 grains (0.0648 to 0.1944 gm.) should be administered, and the stomach well washed out by draughts of warm water. With the emetic or before it the antidote should be administered. The best antidote is freshly precipitated sesqui-

oxide of iron, which forms, with arsenic, an insoluble compound. It must be freshly prepared, taking any of the sesqui solutions of iron, preferably the chloride, and neutralizing it with sodium carbonate or magnesia. The precipitate, being hastily washed by emptying on a muslin or filter, pouring water on it and allowing it to drain, should be freely administered. Dialyzed iron may be used, but it is best also precipitated before use with ammonia or other alkali. In extreme cases the tincture of the chloride of iron, Monsel's solution, or any of the sesqui preparations, may be substituted for the precipitated sesquioxide.

After the emetic has acted, and while the antidote is being given, castor oil should be administered to carry off the poison from the bowels.

Chronic Arsenical Poisoning.—This is ascribed to wall papers covering occupied apartments, sometimes to arsenic long administered as a medicine, to artificial flowers, and clothing fabrics. The glazed green and red papers are those especially dangerous. Occasionally, arsenic medicinally administered may produce the symptoms of slow arsenical poisoning.

Symptoms.—Chronic arsenical poisoning may be suspected in the presence of unexplained anæmia and debility, irritation of the conjunctiva, mouth, pharynx, and lower digestive tract, numbness, tingling, and gastralgia; also nervous symptoms and altered nutrition to special parts. All these symptoms may, however, be produced by other causes. Paralysis may also ensue, resembling that of lead palsy, but affecting rather the lower extremities, especially the extensors and peroneal group, whence may arise the characteristic *steppage gait* of peripheral neuritis. Deranged electrical reaction may be present before any loss of power, but on differential examination a weakened power of wrist extension and feeble power to spread the fingers may be detected.

Treatment.—The patient should be removed from the exposure and symptoms treated as they arise. The iodide of potassium may be used.

PTOMAÏNE POISONING.

Ptomaïne, from the Greek πτῶμα, a cadaver, is a word suggested by the Italian toxicologist, F. Selini, for substances generated in the decomposition of organic matter, which more recent studies have shown to be the result of bacterial action. Ptomaïnes are basic, uniting with acids to form salts. Leucomaïnes are similar basic substances formed in the living body. Ptomaïnes differ greatly in their character and properties, certain ones being intensely poisonous, others harmless. For the former L. Brieger suggested the name toxins, retaining that of ptomaïnes for the non-poisonous basic products; but, as Victor C. Vaughan suggests, there are difficulties in the way of such classification because a ptomaïne may be poisonous under certain conditions and harmless under others.

Leucomaïnes are more usually harmless, though they also under certain conditions may produce disease.

Among ptomaïne poisons are the agencies which are responsible for

various forms of meat poisoning, poisoning by milk products, by shell-fish, and fish.

Meat Poisoning.—This succeeds the eating of various forms of meat which has been the seat of a decomposition in the whole or some one of the constituents of the mass.

Sausage poisoning, also called botulismus and allantiasis, follows the eating of sausage. Numerous outbreaks have occurred in Germany, more particularly in Wurtemberg and adjacent Baden. Already in 1820, Kerner had collected reports of 76 cases, of which 37 were fatal, and in 1822 he had increased the number to 155, with 84 fatal. The poisonous qualities are referred to defective methods of preparation, which permit decomposition.

Ham poisoning, not due to trichina, has occurred in England, Germany, Switzerland, while poisoning has also been traced to beef, mutton, veal, turkey, and goose-grease, and in America to canned meats. Some of these must be ascribed to muriate of zinc and tin, but others are doubtless due to the meats. Poultry, especially if kept too long, and game-birds also prove poisonous at times.

Symptoms.—The symptoms of various epidemics vary somewhat, but the following are most constant, after a period of incubation of from one to forty-eight hours : *nausea*, *vomiting*, *cramps*, and *diarrhœa*,—in a word, acute gastro-intestinal irritation. To these may be added *dryness of the mouth*, *constriction of the throat*, and *difficulty in swallowing*, *vertigo*, *indistinctness of vision*, *dilatation of pupils*, while sometimes *constipation* substitutes diarrhœa. *Thirst*, *headache*, and *muscular weakness* may also be present.

The symptoms may begin at once without incubation in a feeling of languor and general malaise, loss of appetite, nausea, and griping pain in the belly.

In fatal cases the symptoms of cholera are simulated, such as cramps in the legs or arms, or both, muscular twitchings, stiffness of the joints, drowsiness, coldness of surface, pinched features, blueness of fingers and toes and around the sunken eyes,—in a word, the symptoms of collapse. On the other hand, the temperature sometimes rises to 101° to 103° F. (38.3° to 39.4° C.), with a pulse from 100 to 128.

Poisoning by Milk and its Products.—The causes of poisoning by cheese claimed attention as far back as 1827, when analyses of poisonous cheeses were made by Hunnefeld. The older view that the poisons are fatty acids has been refuted, and Vaughan isolated in 1884 a ptomaïne, which he has called tyrotoxicon (τυρος, cheese, and τοξικον, poison). Tyrotoxicon was not, however, always found by Vaughan in cheeses of acknowledged poisonous properties. In 1885 he found tyrotoxicon in milk which had stood in well-stoppered bottles for about six months, and in 1886 Newton and Wallace obtained it from milk which had poisoned a number of persons in a hotel at Long Branch, N. J. Since then tyrotoxicon has been isolated many times from poisonous milk. Finally, in 1886, Vaughan obtained tyrotoxicon from ice-cream which had proved poisonous, and since then it has been frequently found in such cream. A number of cases of poisoning after eating "cream puffs" have been re-

ported in Philadelphia and elsewhere, in which doubtless the same ptomaïne is responsible. Within a few days of this writing a family under my observation were poisoned with *blanc mange*, of which all had eaten freely and which had been made for several days.

Symptoms.—The symptoms of milk and cheese poisoning are like those of gastro-intestinal irritation.

Poisoning by Shell-Fish and Fish (*Ichthysmus*).—The mussel furnishes the most frequent source of poisoning from this cause, instances of which were reported as early as 1827 by Combe. A ptomaïne was isolated by L. Brieger, in 1885, from poisonous mussels at Wilhelmshaven, where numerous instances occur. Brieger has called it mytilotoxine; from *mytilis*, a mussel. It is found chiefly in the liver of the mussel, but whether in a special poisonous mussel or a mussel which becomes poisonous under certain circumstances is not settled, though the latter would seem to be true since Schmidtmann found that non-poisonous mussels placed in the waters of Wilhelmshaven Bay became poisonous, and poisonous mussels from the latter became harmless after being placed in the open sea.

Symptoms.—Both cooked and raw mussels may produce the poisonous symptoms. Three sets are described:—

First, those of gastro-intestinal irritation, similar to those described as due to meat poisoning and which may terminate fatally within two days, the autopsy revealing inflamed stomach and intestines.

In a second set of symptoms the nervous system seems to bear the brunt of the poison, and these cases are said to be the most frequent. The symptoms include a sense of heat and itching, usually beginning in the eyelids, but soon extending over the whole face and sometimes over a large portion of the body. An eruption, vesicular and papular, makes its appearance and intensifies the itching. The eruption is often followed by asthmatic breathing. Sometimes the dyspnœa precedes the eruption, the face becomes livid, the patient unconscious, and there are convulsive movements of the extremities. In other cases there are delirium, convulsions, coma, and death within three days. In other nervous cases there are numbness and coldness, frequent pulse but no fever, the pupils are dilated, and death takes place in a couple of hours with symptoms of collapse.

In a third set of cases a condition like intoxication by alcohol is present, followed by paralysis, coma, and death.

Treatment of Ptomaïne Poisoning.

This is mainly symptomatic—the purgative and emetic effect of the poison generally gets promptly rid of any residue which may be in the stomach or intestinal canal. But if there is any reason to believe that these are not emptied purgatives should be administered, and of these calomel is probably the best because it is less apt to be rejected.

In addition counter-irritation by mustard, hypodermic injection of ¼ grain (0.0165 gm.) morphine, repeated if necessary, to relieve pain, digitalis ten to 30 minims (0.66 to two gm.), and strychnine $\frac{1}{30}$ grain (0.0022 gm.) administered in the same manner to counteract collapse may be given. Stimulants by the mouth should be given if retained, and to

this end champagne becomes very suitable, or milk mixed with carbonated water may be given in small quantities.

GRAIN POISONING.

For a century or more certain districts have been subject to ailments which have been traced to the use of certain grains as food, some of which have been found to be spoiled or the seat of disease. People in certain parts of France, Germany, Switzerland, Italy, Spain, and India have been thus affected.

(1) Ergotism.—Ergotism is one of these. It is a disease found to succeed upon the use of meal contaminated with the *sclerotium*, an intermediate stage of development of the *claviceps purpura*, a fungus which infests the rye-grain. *An* ergot is this sclerotium, which appears at the base of the grain as a hard, dark-hued "spur," which, as it grows, lifts up the diseased and withered mass of the original grain. Wheat, barley, and rice may also become spurred. The growth of the fungus is favored by wet seasons. The disease prevailed in France, Switzerland, and Germany much more commonly from the tenth to the eighteenth century than at present. The cause of ergotism was discovered in 1830 by Thuillier.

Two forms of chronic ergotism are recognized, one convulsive or spasmodic, the other gangrenous.

Spasmodic Ergotism.—After a prodromal period of ten to fourteen days, during which there is a peculiar sense of weariness and anxiety, a tingling and sense of formication in the skin, especially of the fingers and toes, gastro-intestinal irritation manifested by vomiting, purging, and colicky pains, accompanied sometimes with slight fever, *spasmodic symptoms set in.* These consist at first in involuntary twitchings, which soon pass into painful continuous contractions, the arms being flexed and the legs and toes extended. The cramp lasts for an hour or more, followed by a period of exhaustion, which may be succeeded by another painful convulsion. There may be delirium, melancholia, or dementia. The urine may be suppressed or violent dysuria may be present from spasm of the bladder. Pustules, boils, whitlows, and other evidence of deranged nutrition may appear. Cardiac contractions are slow and feeble, and the arteries constricted, containing little blood. Death may occur from cardiac paralysis, and is often preceded by convulsions or paralytic symptoms. The duration of the illness is four to eight weeks or longer.

Posterior sclerosis was found in some of the cases which came to necropsy. Thus, Tuczek and Siemens found it four times in nine autopsies, which represented, also, the deaths in a group of 29 cases.

Gangrenous Ergotism.—This form is ushered in by the same prodrome as that described for the spasmodic. On this succeeds, from the third day to the fourth week, an erysipelatous redness in some peripheral locality, as the toes and fingers, ears and nose. This is followed usually by dry gangrene, but the moist form, which may be confined to a finger or toe or may involve the whole hand or foot, may also appear. The disease may not go beyond the erysipelatous redness.

For acute ergot poisoning, see concluding section.

(2) Pellagra.—This is a disease thought to be due to a fungus which infests mouldy maize or Indian corn. Lombroso and others have isolated a ptomaïne from the meal made of such corn. The disease occurs in Lombardy, the south of France, and in Spain, especially among the poorer classes in the country districts, where the meal of maize is largely used. It begins almost invariably in the spring of the year in an erythema, which is followed by a scaly and wrinkled condition of the skin, especially in the parts exposed to the air. Occasionally crusts form, and beneath these pus is found. Along with these skin troubles there are digestive derangements, salivation, dyspepsia, and even dysentery. The disease lasts a few months, when improvement sets in. In the more severe and chronic forms there may be headache and backache, the strength and mental faculties are affected, sensation is obtunded, and cramps with convulsions supervene, much as in ergotism.

The morbid anatomy is vague. There may be fatty degeneration and a pigmentation of the viscera.

(3) Lathyrism, or Lupinosis.—This is a condition resulting from the use of meal made from the chick-pea, or grain of a variety of vetches, more particularly the lathyrus salivus and lathyrus cicera. It is used in admixture with barley and wheat in India, Italy, and Algiers. According to James Irvine, the symptoms supervene in India when the proportion exceeds $\frac{1}{12}$.

The symptoms are, first, gastro-intestinal irritation, then a condition of spastic paralysis, which may pass on to complete paraplegia. The arms are rarely, if ever, affected.

No associated morbid change has been made out.

Treatment of Grain Poisoning.

This consists, primarily, in the removal of the cause, and the substitution of suitable food; in removal, also, from the district, if possible, and suitable treatment of symptoms.

SECTION XIII.

EFFECTS OF EXPOSURE TO HIGH THOUGH BEARABLE TEMPERATURE.

Such effects are easily separable into two groups, covered by the terms heat exhaustion and thermic fever.

HEAT EXHAUSTION.

Definition.—A condition of syncopal exhaustion with vaso-motor paralysis and lowering of bodily temperature, due to exertion under high temperature. Such condition may arise quite independently of the direct rays of the sun. The heat may be that of confined rooms indoors and may be artificial heat.

Symptoms.—The sense of great weakness often experienced in hot weather after some unusual exertion exhibits the mildest degree of this condition. In the more severe forms a sense of faintness, associated with pallor, dizziness, at times blindness, and the starting of cold perspiration are the first symptoms. Sometimes the victim can get to a place where he may sit or lie down ; at others he faints away before assistance can reach him. Then follows a condition of unconsciousness or semi-consciousness, whence under favorable circumstances he may respond to gentle stimulus by ammonia or wine and then fall into a sleep, whence he will awake in an hour revived.

In more severe cases the collapse is more permanent, the pulse is extremely feeble and frequent, the skin continues leaky, while there may be great restlessness and muttering delirium. It is characteristic of this form of heat affection that there is extreme adynamia with lowered body temperature. H. C. Wood, whose name is inseparably associated with the subjects of heat exhaustion and thermic fever, reports a case with a temperature of as low as 95° F. (35° C.), with complete collapse.

Diagnosis.—That from thermic fever is based on the lowered temperature and feeble pulse in heat exhaustion as contrasted with the opposite in thermic fever. It is important that the two conditions should not be confounded, because of the widely different treatment required. The syncopal attack from cardiac failure or from concealed hemorrhage much more closely resembles heat exhaustion, being associated also with feeble pulse and lowered temperature, but as the treatment is identical,

the distinction is less important. The fall in temperature is, however, less decided in syncope.

Treatment.—The patient should at once be put to bed with his head horizontal or slightly raised. When possible, stimulants should be administered moderately by the mouth,—brandy, whiskey, and ammonia with digitalis. If this is not possible, digitalis and strychnine should be given hypodermically, ten to 30 minims (0.66 to two gm.) of the former and $\frac{1}{30}$ grain (0.0022 gm.) of the latter. Friction should be applied, and dry heat by hot-water bags or cans.

THERMIC FEVER.

SYNONYMS.—*Heat Fever; Sunstroke; Coup de soleil.*

Definition.—A state of high fever induced by exposure to heat, natural or artificial.

Etiology and Pathology.—In this country the majority of cases occur in the summer season in those exposed to the direct rays of the sun, though they occur also among those exposed to high heat within doors, as in sugar refineries, the fire-rooms of ocean steamers, laundries, and the like. A heated atmosphere charged with moisture, impeding, therefore, evaporation, produces fever much more rapidly than a dry heat, which is in fact slow to produce it. The habitual use of alcohol is found to be a potent predisposing cause, at least alcoholics very much sooner succumb to the influence of over-heat than temperate persons.

The pathology of the two conditions of heat exhaustion and thermic fever is thus explained by H. C. Wood: "There is in the pons or higher portion of the nervous system a centre whose function it is to inhibit the production of animal heat, and in the medulla a centre (probably the vaso-motor centre) which regulates the dissipation of bodily heat. Fever is due to a disturbance of these centres so that more heat is produced than normal and proportionately less thrown off. Let it be supposed that a man is placed in such atmosphere, that he is unable to get rid of the heat which he is forming. The temperature of the body will slowly rise and he may suffer from a general thermic fever. If early or late in this condition the inhibitory heat centre becomes exhausted by the effort which it is making to control the formation of heat, or becomes paralyzed by the direct action of the excessive temperature already reached, then suddenly all tissues will begin to form heat with the utmost rapidity, the bodily temperature rises with a bound, and the man drops over with one of the forms of coup de soleil."

"Heat exhaustion," on the other hand, "with lowered temperature, represents a vaso-motor palsy, *i. e.*, a condition in which the existence of the heat paralyzes the centre in the medulla, and the heat is dissipated more rapidly than it is produced." It must be admitted that the explanation of heat exhaustion is less satisfactory than that of thermic fever.

Morbid Anatomy.—The high temperature characteristic of heat fever remains a long while after death. Hence putrefaction sets in early. Rigor mortis also occurs promptly. The blood remains liquid. There

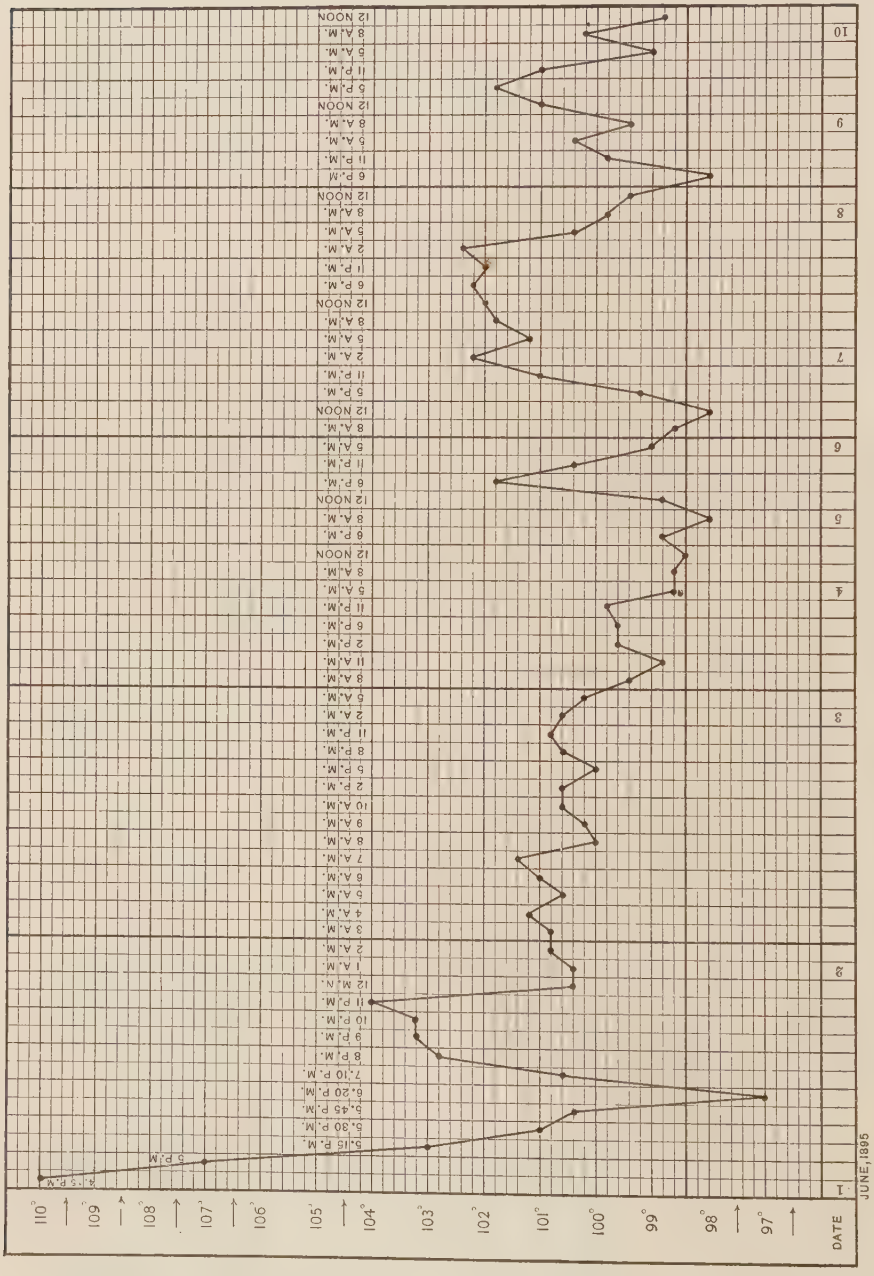


FIG. 90.—TEMPERATURE CHART FROM A CASE OF SUNSTROKE, TREATED BY ICE-WATER BATHS AND FRICTIONS. RECOVERY.

is general venous engorgement, especially of the lungs and cerebrum. In early autopsies the left ventricle is found contracted, the right dilated.

Symptoms.—A sense of uncomfortable burning heat and feeling of oppression may precede the "stroke" which fells its victim, who quickly becomes unconscious and comatose, perishing sometimes instantly, at others in a few hours. In other cases there are intense headache, dizziness, oppression, nausea, and vomiting, occasionally diarrhœa. Chromatopsia or colored vision may be present. Sooner or later unconsciousness sets in, and may be associated with muttering delirium, and intense restlessness. In this condition the patient is commonly admitted to hospital, with face flushed, eye suffused, skin hot and dry, temperature from 107° to 112° F. (41.6° to 44.4° C.), the breathing labored, sometimes stertorous, the pulse frequent and full. The pupils at this stage are usually contracted, though at first dilated. The urine is scanty, sometimes albuminous. Usually there is relaxation of the muscles, but at times there is a convulsive tendency, shown by twitching and jactitation, and occasionally epileptiform convulsions. The skin, usually dry, may become moist and bathed with perspiration, which does not, however, reduce the temperature. Wood speaks of a peculiar odor exhaled by the entire body as characteristic.

In fatal cases the stupor deepens, the pulse becomes more frequent and loses even its seeming strength, then is irregular, the inspiration labored and irregular, and toward the last shallow, or assumes the Cheyne-Stokes type previous to death. Death does not usually take place for several hours. In favorable cases improvement is indicated by a falling temperature and a return to consciousness.

Recovery may be complete, but more rarely a permanent condition results in which there may be more or less constant mental weakness, as evidenced by incapacity for sustained mental effort, while exposure to moderate degrees of temperature produces great excitement or headache or pain in the upper cervical region. Epileptic convulsions sometimes occur. In these cases there is probably a certain degree of meningitis.

Mention has already been made, when treating of fevers, of the form of continued fever occurring in the south of the United States, where it is known as "Florida fever" and "country fever," and in India and the West Indies as *fièvre inflammatoire*, for which John Guitéras proposes the name *continued thermic fever*, but which more recently he is inclined to ascribe to a septic origin.

Diagnosis.—The diagnosis of heat fever presents no difficulties. The distinction between it and heat exhaustion has been alluded to.

Prognosis.—The prognosis depends partly upon the severity of the case, and the promptness and thoroughness of treatment. A few cases are almost instantaneously fatal. If the cooling treatment can be applied properly, a decided majority, fully 60 per cent., recover. A temperature of 110° F. (43.3° C.), though indicating gravity, should not discourage.

Treatment.—The success of treatment of thermic fever depends altogether upon our ability to lower the temperature. To this end the patient should be placed in a bath of water, to which ice is freely added to keep the temperature down as low as it can be, which in summer is

not likely to be below 60° F. (15.54 C.). The surface of the body is further vigorously rubbed with ice. In the absence of bath facilities the patient should be placed on a mattress covered with a mackintosh and rubbed with pieces of ice. The refrigerating effect may be further increased by ice-water enemata. This treatment should be regulated by the thermometer in the rectum, and abated as the temperature approaches the normal, and renewed as it again rises.

After this, or in addition to this, treatment should be symptomatic: For convulsions, chloral or chloroform; for heart failure, digitalis and strychnine hypodermically; for asphyxia, bleeding. Mild cases or recurrent fever may be treated with the antipyretics represented by antifebrine, phenacetine, and antipyrine.

The sequelæ referred to should be treated symptomatically.

SECTION XIV.

ANIMAL PARASITES AND THE CONDITIONS CAUSED BY THEM.

While the number and variety of parasites which infest man are extremely large, the number of these which are directly harmful is very much less. Representatives of sufficient importance to deserve consideration are found among the

I. Protozoa.
II. Platyhelminthes.
III. Nematodes.
IV. Acanthocephali, and
V. Arthropoda.

I. PROTOZOA.

(1) *Psorospermiasis.*—The protozoa are represented by the psorosperms or sporozoa, and these by the *coccidia*. The latter are spherical or oval, nucleated organisms, composed of granular protoplasm. They vary in size, the smallest being 0.012 to 0.015 mm. (0.0005 to 0.0006 inch) long, and 0.007 to 0.01 mm. (0.003 to 0.004 inch) wide, and the largest 0.040 to 0.049 mm. (0.0016 to 0.0019 inch) long, and 0.022 to 0.028 mm. (0.0008 to 0.0011 inch) wide.

They lodge within the epithelial cells of man and the lower animals, one or more within a single cell, whence they are also called *cytozoa*. They are closely allied to the hæmatozoa or blood parasites which occupy the corpuscles of fish and amphibia. Other varieties are found outside of cells in the submucous tissues, and in the villi of the intestinal wall of dogs, cats, rabbits, man, and other animals, or in organs adjacent, such as the liver and kidney. Wherever they lodge they immediately become encysted, forming whitish, oval nodules, especially easily studied in the liver of the rabbit. These nodules vary in size from a pin's head to a split pea, and resemble the tubercles of tuberculosis. In most of the cases in man the liver is found to be the seat of invasion, and the resulting tumors may be felt during life. They have thus been mistaken for echinococci. The spleen, kidneys, omentum, peritoneum, and pericardium may also be invaded. Other symptoms are abdominal muscular pain, tenderness, sometimes diarrhœa, nausea, dry tongue, hæmaturia, and peritonitis. The effect appears to be one of intense irritation, like

that of general miliary tuberculosis or trichiniasis, causing death in variable periods, from fourteen days to several years. Cases of internal psorospermiasis, as above described, have been reported in France, Germany, Austria, and England, but not in America. Two cases of external or cutaneous psorospermiasis, affecting the skin only, were reported by Rixford and Gilchrist in the Johns Hopkins Hospital Reports, Vol. I. The affection attacked the skin of the face, trunk, and extremities, producing tubercular nodules. Subsequently, internal organs, including the lymphatic glands and lungs, were invaded, producing symptoms and lesions like those of tuberculosis, but enormous numbers of sporozoa were found in the caseous masses.

(2) *Parasitic Infusoria.*—The protozoa are also represented by the infusoria, and of these especially the subclass *flagellata*. These include the *plagiomonas urinaria*, found once only by Künstter in the fresh urine of a man who had suffered from chronic suppuration; also the *trichomonas vaginalis*, an irregular spindle-shaped organism 0.015 to 0.025 mm. (0.0006 to 0.0009 inch) long and 0.016 to 0.018 mm. (0.0006 to 0.0007 inch) wide, found in the acid vaginal mucus; the *trichomonas* or *circomonas hominis*, a pear-shaped organism 0.004 to 0.007 or 0.015 mm. (0.0002 to 0.0006 inch) long and 0.003 to 0.004 mm. (0.0001 to 0.0002 inch) wide, terminating behind in a pointed projection and in front in four cilia, found in intestines and stools; also the *lamblia intestinalis*, a similar-shaped organism found in the intestinal canal. Flagellates have also been found in the lungs, in gangrene, bronchiectasis, and pleurisy. Of the ciliated infusoria *balantidium* or *paramœcium coli*, an oval body 0.070 to 0.1 mm. (0.0027 to 0.0393 inch) long and 0.050 to 0.070 mm. (0.0019 to 0.0027 inch) wide, has been found in the large intestine in dysentery.

II. PLATYHELMINTHES, OR FLAT-WORMS.

The flat worms include the
(A) Trematodes, or flukes.
(B) Cestodes, or tapeworms.

(A) Trematodes, or Flukes.

These are divided into liver flukes, blood flukes, and bronchial flukes.

(*a*) *Liver Flukes.*

These include,—

(1) The *distomum hepaticum*, or fasciola hepatica, 25 to 32 mm. (0.9842 to 1.2598 inch) long, eight to 13 mm. (0.3149 to 0.5118 inch) wide, found in the liver, blood-vessels, and in abscesses in men.

(2) The *distomum buski*, large, four to 8.5 cm. (1.5748 to 3.3464 inch) long, 1.4 to two cm. (0.5511 to 0.8 inch) wide, found in the liver and fæces.

(3) The *distomum lanceolatum*, eight to ten mm. (0.3149 to 0.3937 inch) long, 1.5 to 2.5 mm. (0.059 to 0.0984 inch) wide, in the intestine, whence it has been passed per anum and even by the mouth.

(4) The *distomum sinense*, ten to 13 to 18 mm. (0.3937 to 0.5118 to 0.7086 inch) long, two to three mm. (0.0787 to 0.1181 inch) broad. This is a distoma especially prevalent in Japan, where it was described as distomum endemicum and distomum perniciosum. It occurs in 20 per cent. of the inhabitants of certain provinces, according to Baelz.

(5) *Distomum conjunctum*, ten mm. (0.3937 inch) long, 2.5 mm. (0.0984 inch) broad, found in the liver.

(6) *Distomum felineum*, eight to 18 mm. (0.3149 to 0.7086 inch) long, 1.5 to 2.5 mm. (0.059 to 0.0984 inch) wide, found in man and cats.

All of these are known as liver flukes, being found in the bile passages and upper part of the small intestine. When numerous they give rise to very serious disturbance, including cholangitis with resulting jaundice, enlargement of the liver, emaciation, diarrhœa, and often ascites.

(*b*) *Blood Flukes.*

SYNONYMS.—*Bilharzia hæmatobia, or schistosoma hæmatobium; Distomum hæmatobium; Distomum capense.*

This distoma was first described by Bilharz in 1852. The male is thread-like, four to 15 mm. (0.1574 to 0.5905 inch) long and 0.6 mm. (0.0236 inch) wide in the widest portion; the female is much longer than the male, 15 to 20 mm. (0.5905 to 0.7874 inch), but much narrower, so that it may enter the canalis gynæcophorus of the male.

It inhabits the venous system, especially the portal vein and the veins of the spleen, kidney, the venous plexuses of the bladder and rectum in boys of the lower classes, more rarely men and women in Egypt and Africa, 50 per cent. of the former being infested, according to Bilharz. It is geographically much more widely distributed in Africa, throughout the east coast as far as Capeland, in numerous districts in the interior, and from the Gold Coast to Algiers.

Symptoms.—The symptoms of the bilharzia disease begin usually with hæmaturia and burning pain in the urethra, the latter increased during micturition and due to the irritation of the worm. In addition to blood the urine contains pus, shreds of mucus, and the eggs of the bilharzia which are easily recognized with a low power of the microscope. They are ovoid and terminate in a thorn-like end. The larva may be seen in the interior of the egg. The ova may become the nucleus of stone in the bladder, a fact which is held responsible for the large number of cases of stone in the bladder in Egypt. Hypertrophy of the prostate may ensue. The large bowel may be invaded, causing dysentery. The hemorrhage may lead to anæmia, debility, and exhaustion, terminating fatally, though most cases recover. Attacking, especially, children, the disease commonly disappears at puberty.

(*c*) *Bronchial Flukes.*

SYNONYMS.—*Distomum Westermanni; Distomum pulmonalis.*

This is an ovoid fluke eight to ten mm. (0.3149 to 0.3937 inch) long and four to six mm. (0.1574 to 0.2362 inch) broad, of a reddish-brown

color. It is met with in various parts of China, Japan, and Formosa where it infests the bronchial tubes causing cough and hæmoptysis.

(B) Cestodes, or Tapeworms.

(*a*) *Intestinal Cestodes.*

Historical.—These when mature occupy the intestine of man. The larger forms at least of tapeworm were known to the ancients as animal parasites infesting the alimentary canal. The correct notion of the origin and development of the tapeworm is, however, comparatively recent, and for a long time the young worm was supposed to start from the links or proglottides of the adult worm. The cysticerci or larval forms have also been long known, but were regarded as simple cystic tumors until almost simultaneously Redi, in Italy, and Hartmann and Wepfer, in Germany, inferred their animal nature, after which they were regarded as worms and made by Zeder, in 1800, a separate class of bladder worms.

In 1683 Edward Tyson discovered the head with its double row of hooklets in a large tapeworm in a dog; in 1684 Redi recognized the head and the suckers of several tæniæ; in 1700 Andry recognized the head of the tænia saginata, and in 1777 Bonnet and in 1779 Gleichen-Russworm that of the bothriocephalus. From that time it was believed by most authors that the tapeworm was an animal which nourished itself by fastening its head into the intestinal wall. About the middle of the present century Küchenmeister, by his celebrated feeding experiments, proved that the cysticerci or bladder worms represented a larval stage in the development of the tapeworm, the latter having for its habitat the intestine of man, and the former the muscles and solid organs of certain lower animals and rarely of man.

Of the several varieties of tapeworm which infest man four only deserve special consideration, viz., the tænia saginata or mediocanellata, the tænia solium, the bothriocephalus latus, and the tænia flavopunctata.

The *tænia mediocanellata* or *tænia saginata* is also known as the beef tapeworm, because its embryos or cysticerci are found, as a rule, in cattle, producing the so-called "measled" veal and beef; also as the "unarmed" tapeworm, because the head is unprovided with hooklets. It has also been found in the flesh of the giraffe. The *tænia solium* is known as the pork tapeworm, because met in the larval state in the flesh of swine, producing "measled" pork; and as the "armed" tapeworm, because of the hooklets named. The beef tapeworm, or tænia mediocanellata, is the most common form of tapeworm occurring in the United States, and I believe also at this time on the Continent of Europe, especially in Western Europe. In the middle of Germany it is less unusual to eat uncooked pork, and although the laws of inspection there are very stringent, the tænia solium has been at least until recently the more common variety of tapeworm. But in Holstein, Heller has met the tænia saginata four times as often as the tænia solium. The late Professor Leidy, to whom large numbers of worms were constantly being sent for opinion, informed me that all specimens sent to him for examination during a period of fifteen years, were tæniæ mediocanellatæ.

The tænia mediocanellata, or unarmed worm, has a head quadrate in shape, 1.5 to two mm. (0.06 to 0.0787 inch) in diameter, provided with four suckers, placed one toward each angle of the head, and a small central rudimentary sucker, without rostellum or circlet of hooks. To this head succeeds a slender neck, then the links. The tænia solium has a smaller head, spherical in shape, 0.6 to one mm. (0.0236 to 0.0393 inch) in diameter, provided, in addition to the four suckers, with a proboscis or snout called a rostellum, on which are two rows of hooklets, the inner row being the longer. There are from 12 to 14 in each row, making in all 24 or 28. The heads of both worms are at times more or less pigmented. There is also a difference in the segments. The ripe segments of the tænia saginata average from 16 to 20 mm. (0.63 to 0.8 inch) long and from four to seven mm. (0.1574 to 0.275 inch) broad. The ripe segments of the tænia solium average from ten to 12 mm. (0.4 to 0.47 inch) long and five to six mm. (0.2 to 0.2362 inch) broad.

Tapeworms are hermaphroditic, both sets of reproductive organs being contained in one segment. Further peculiarities of these organs will be considered in speaking of diagnosis. As to the length of the entire worm, the tænia mediocanellata is put down at from 4.8 to 7.2 meters (16 to 24 feet), but I have known more than ten meters (40 feet) passed. The tænia solium is usually much shorter—from 1.8 to 3.6 meters (six to 12 feet), but may be longer. The number of links or proglottides in the tænia solium is 800 to 900, in the tænia saginata 1000.

How do these tapeworms obtain access to the intestine of man? As already stated, the larva or cysticercus of the mediocanellata is found in "measly" beef. The egg containing the larval or embryo worm is in some way ingested by cattle. The envelope with which it is surrounded is digested and the embryo released. The latter is provided with hooklets arranged in three pairs, one central and two lateral. The central pair are simply stilettos for boring, the lateral for tearing and propulsion. By these means it bores its way through the intestinal wall and invades the muscular tissue, where it becomes encysted, forming the *cysticercus bovis*. It is a singular fact, however, that the "measles" of tænia saginata very rarely come under observation. When found, the cysticercus of this worm is an oval cyst, scarcely as large as a pea, and is probably often overlooked. Notwithstanding its apparent rarity it is responsible for the majority of tapeworms in man.

In the case of the tænia solium the links of the worm thrown off by man in the fæces are eaten by the pig; during digestion the shell of the egg is dissolved and the embryo liberated. The embryo of the tænia solium is not provided with the double row of hooklets possessed by the mature worm, but, like that of the tænia saginata with six hooklets, similarly arranged in pairs. It invades the muscular tissue, like the tænia saginata, becomes encysted, and thus the *cysticercus cellulosæ telæ* is formed, and the pork becomes "measled." The "measle" of pork is larger than that of beef. In the former, it sometimes reaches a length of nine-tenths of an inch (22.86 mm.), while in the latter, it is, as a rule, not more than one-fourth of an inch (6.3 mm.) in length.

The "measles" of beef and pork are rendered inert by cooking.

But it constantly happens that imperfectly cooked beef or pork are eaten, when the capsules of the embryo are dissolved off in the stomach, the embryo passes into the small intestine, attaches itself, and grows. From twelve to sixteen weeks after the attachment of the larva, fragments of the worm may be discovered passing per anum.

The laws demanding the inspection of pork and beef are causing the tapeworm to become less common, but it is still quite often met. At the present day we are much less in the habit of eating uncooked pork than rarely done and raw beef. This is also the case in England. It is on this account that the pork tapeworm is less common in America and England than the beef tapeworm.

The third variety of tapeworm, the *bothriocephalus latus*, is distinguished by the greater width of its segments and their shortness in an antero-posterior direction. These statements refer to the average, for the links taken from different parts of the same worm vary much in their dimensions. The number of links of the bothriocephalus is from 3000 to 4000. Still more widely does the head of the bothriocephalus latus differ from that of either of the two which I have described. It has neither rostellum nor hooklets, is club-shaped, 1.5 mm. (0.06 inch) long and one mm. (0.04 inch) wide, and is marked by two lateral depressions. The head is, therefore, less formidable than that of either of the two other worms. This worm is not often met with in this country. It is more common in Switzerland and Russia, but is also found in Ireland, Sweden, and Germany, especially in Eastern Prussia, and recently in the neighborhood of the Starnberger See and Munich. It is called the Irish tapeworm as well as the Russian and Swiss tapeworm. It is believed that the cysticercus of this variety of tapeworm is found in certain fish of the salmon and trout family, and that man acquires the worm by eating these fish uncooked. The length of the bothriocephalus is put by Heller at from 4.5 to 7.5 meters (15 to 25 feet), but if it be the longest of the tapeworms, as is generally stated, it must at times far exceed this.

There is still another form of tapeworm, which, there is reason to believe, is more common than is supposed. This is the *tænia flavopunctata*. It is a little tapeworm, not exceeding 30 to 37.5 cm. (12 to 15 inches) in length. In the American Journal of the Medical Sciences for July, 1884, the late Dr. Leidy describes the segments of one of these worms from specimens sent to him. They were passed by a child three years of age, after the administration of santonin. In fact, all the specimens of this worm as yet described seem to have come from children fifteen months to three years of age. The joints are proportionately short in all cases, the breadth exceeding the length several times. They are transversely linear, slightly wider back of the middle, with the posterior angles rounded, and the posterior margin slightly but irregularly crenate. The anterior immature joints exhibit no distinct traces of sexual organs, while succeeding ones have a more coarsely granular appearance, due to the presence of immature eggs. The pair of longitudinal vessels common to all tapeworms are conspicuously visible in the segments. At the posterior part of the body, the joints in a ripe condition

are of a pale brown color, due to the contained eggs. The ripe joints are interrupted here and there by a variable number, one or more, of sterile joints. The ripe joints are longer, broader, and thicker. The eggs distend the joints, and are diffused throughout a simple cavity, bounded by thin parietes, and are not inclosed in ramified or dendritic uterine pouches, as is the case with most tapeworms. In Dr. Leidy's specimens, the mass of eggs so concealed everything in these joints that the generative apparatus could not be determined. But in the joints devoid of eggs there was to be seen a narrow, clavate organ, which Dr. Leidy suspected to be the seminal receptacle, together with four or five oval bodies, probably testiculæ. In these joints only, as a rule, could the genital aperture be recognized at the lateral margin of the joints, immediately behind the middle.

The joints of this tapeworm range in length from 0.2 mm. (0.008 inch) in the neck to 0.5 mm. (0.02 inch), an inch (2.5 cm.) from the posterior extremity; and in width from 0.63 mm. (0.025 inch) in the neck to 2.5 mm. (0.10 inch), an inch from the posterior extremity. The mature eggs are raw sienna-colored, mostly spherical, and 0.07 mm. (0.003 inch) in diameter. The specimens sent to Dr. Leidy were parts of three worms, so it would seem that more than a single worm may infest the host, which is the case with other cestoid worms.

Another instance of this worm has been described by Dr. Weinland of Cambridge, Mass., and a third by E. Parona, in Italy. It was named by Weinland tænia flavopunctata, in consequence of yellow spots lying near the middle line in each successive joint, and representing, according to Dr. Weinland, the testicle. This spot had disappeared in Dr. Leidy's specimen, as it had also in Dr. Weinland's when it reached Professor Leuckhart some years afterward. The enclosed embryos are oval and are provided with six hooks arranged in three pairs. The intermediate or larval condition of this worm is unknown. I have taken some pains to describe the tænia flavopunctata, because, as I have already said, it may be more common than is usually supposed, and it is desirable that it should become so familiar that it may be recognized when met. The larvæ are developed in lepidoptera and beetles.

Symptoms of Tapeworm.—Given a tapeworm in the human intestine, how is it to be recognized? It has often happened that the parasite has been present without giving rise to any symptoms, having been accidentally discovered by the expulsion of segments.

The symptoms occasioned by its presence may be put down in general terms as those of irritation. They may be either direct or reflex. Among the direct symptoms are vague abdominal pains, nausea, and meteorism. Nausea is very common, and both it and the pain are easily accounted for by the irritation of so large a mass of living matter in the intestine. The reflex phenomena include a great variety of nervous symptoms, among which may be named headache, vertigo, dizziness, epileptiform convulsions, reflex paralysis, and even insanity. Among other symptoms are loss of appetite, together with faintness, and other disorders of digestion, derangement of vision and of hearing, such as noises in the head. Itching and dryness of the nose and anus, and

vague pains throughout the body are sometimes noted. Sensations of weariness and vague discomfort may alone be present. The only distinctive symptom is, however, the passage of links of the worm. Notwithstanding the presence of these symptoms, we are often compelled, in the absence of positive demonstration—*i. e.*, the passage of pieces of the worm—to administer a purge, in the action of which segments will be passed, if a worm is present.

Diagnosis.—It is a matter of no small importance that physicians should be able to distinguish the different varieties of tapeworm, for the difficulties of treatment vary considerably with the different worms, the tænia saginata being much more easily dislodged than the tænia solium ; and the bothriocephalus latus, perhaps, easiest of all. In the first place, the frequent spontaneous expulsion of segments of worm may be taken as presumptive evidence that they belong to the tænia saginata, as those of the tænia solium rarely drop off. Again, the segments of bothriocephalus latus, while occasionally expelled, are seldom expelled singly, being discharged in pairs or several joined.

The segments of the tænia saginata are stronger, thicker, and less transparent, and, when expelled spontaneously, contain very few eggs. The uterus, which contains the eggs, and which may generally be demonstrated by allowing a segment to dry on a piece of glass, or, better, slightly compressed between two glass slides, is composed of a central canal with lateral branches which are more distinct and less numerous in the tænia solium, being from nine to 12 in the former, than in the tænia saginata, and from 15 to 20 in the latter. The genital pores are lateral in the case of the tænia solium and tænia saginata, but central in the bothriocephalus, which has also a brown rosette-shaped uterus very easily distinguished from that of the other two more common forms of tæniæ.

Prognosis.—This is favorable, as a rule, in all forms of tapeworm, but the tænia saginata and bothriocephalus latus are much more easily dislodged than the tænia solium, the head of which often resists removal for a long time, the segments continuing to reproduce themselves until the head is dislodged. From eight to twelve weeks are usually necessary for the worm to redevelop itself to a size at which segments are again discharged.

Treatment.—There is, perhaps, no morbid condition which has brought more opprobrium upon the regular profession and more "grist to the mill" for advertisers and those who use secret remedies than tapeworm, and to our humiliation it must be said that these persons do seem to have more success in promptly getting rid of tapeworm than we do. There are, I think, two reasons why this is so. In the first place, it is certain that they do not use different remedies from those commonly in use by the profession, but they give larger doses. In the second place, they see a large number of cases and develop a sort of specialty, which, like all specialties, produces greater skill in treatment. In order that a tapeworm may be successfully treated, it is necessary that we shall have a certain size ; so that, if a large part of the worm has been brought away by medicine, it is useless to give anything more until the remaining part increases sufficiently in size.

There are half a dozen remedies for tapeworm, and they are all good. I think that the two best are probably the ethereal extract of male fern and kooso flowers. Some prefer the first of these, while others prefer the second. In my hands, kooso has been decidedly the most efficient—that is, having failed with everything else and having succeeded with kooso, it has naturally become the remedy with which I usually begin the treatment. It is the dried flowers and immature fruit of the Brayera anthelmintica, a tree native to Abyssinia.

Patients require some preparation before any remedy is employed. In all cases they should be kept absolutely quiet during treatment. They should eat nothing from breakfast time of one day until the next morning, during which the bowels should be moved by a saline cathartic, when one ounce (30 gm.) of kooso and eight ounces (240 c. c.) of water are to be taken. A more pleasant way is to give 75 grains (five gm.) in a glass of white wine every half hour until four doses are taken. If at the end of six hours no movement of the bowels has taken place, a promptly-acting aperient, as a dose of oil, compound jalap powder, or elaterium, is taken, but generally kooso requires no purgative after it. The worm is usually discharged entire. Of course one is never certain that this is the case unless the head is found. At the same time, it does not follow because the head cannot be found that it has not been passed, for it is very small, and may be lost in the discharges. In the tænia solium the head is about the size of a small pin's head; in the mediocanellata it is a little larger, and in the bothriocephalus it is still larger. If the head has not been removed, it is certain that in ten to sixteen weeks of time the worm will grow out again and begin to discharge links. Kooso is said to have induced miscarriage; it should not, therefore, be given to pregnant women. Instead of kooso, the resin which it contains, called koosin, may be given; but I have had no experience with it. The dose is 20 to 40 grains (1.33 to 2.66 gm.), enclosed in a wafer. The fluid extract is also efficient in doses of half a fluid ounce (15 c. c.).

The next remedy in efficiency is the ethereal extract of the rhizome of aspidium felix mas, or male fern, whose active principle—an oleo-resin—is extracted by ether. The preparation of the patient is about the same as for kooso. The dose of the ethereal extract is two to 2½ fluid drachms (eight to ten c. c.), followed in a couple of hours by a purgative. It is a dark, thick liquid, bitter, slightly acrid, and nauseous. Instead of the ethereal extract of male fern, the oleo-resin may be given in a gelatine capsule. The dose is 1½ to two fluid drachms (3.88 to 7.4 gm.). A couple of hours later a dose of purgative medicine should be administered. An important point to be borne in mind is the varying quality of these drugs, and that they deteriorate with age.

The third remedy, in order of efficiency, is the bark of the root of the pomegranate. This has been given in the shape of a decoction, two to four ounces (60 to 120 c. c.) to the pint (0.5 liter). Boil the bark half an hour, strain, and drink. The fluid extract is more convenient in the dose of from 45 minims to two fluid drachms (three to eight c. c.). Two hours later a purgative should be given. An alkaloid is obtained

from pomegranate, named pelletierine, in honor of the chemist, Pelletier. This is sold in a single dose of eight to 25 grains (0.5 to 1.6 c. c.). When first introduced it was vaunted as a "sure cure," but the experience of practitioners has not been uniform, and success has been by no means invariable.

Kamala, the hair of the rottlera tinctoria, is said to be very efficient in tapeworm, but I have had no experience with it. It is given in doses of from one to two drachms (four to eight gm.) suspended in syrup, repeated in eight to ten hours if it does not purge. The fluid extract is also given in doses of half a drachm to a drachm (two to four c. c.). It is purgative, sometimes drastically so. It may also cause nausea and vomiting.

Another efficient remedy is the oil of turpentine. It is, however, apt to produce such unpleasant symptoms that it should be the last used. The dose is from an ounce to two ounces (30 to 60 c. c.) mixed with twice that amount of castor oil,—a horrid dose,—but if others fail it may be tried.

Still another is pumpkin-seed. There are two ways in which it may be given. Three or four ounces (30 to 120 gm.) of the seeds may be crushed in a mortar with water, then strained, and the emulsion taken fasting, after a day's dieting. A few hours later a brisk purge should be taken. Or the seeds may be made into an electuary, which is almost as pleasant as sugar candy, and often is about as effectual. I should place these different remedies in the order of their efficiency, as follows: kooso, male fern, pomegranate, kamala, turpentine, and, lastly, pumpkin-seed.

Combinations are sometimes very efficient. The following is recommended by Strümpell:—

℞. Granati corticis radicis, ... f℥ iv to v (120 to 150 gm.)
Aquæ, ... Oij (1000 c. c.)
Macerate for twenty-four hours and boil until it is reduced to f℥ v (150 c. c.).
Add Oleo-resinæ filicis, grains lxxv (five gm.).

The whole amount is to be taken in three or four doses as near together as possible.

Thymol in doses of ten grains (0.66 gm.) three times a day in a wafer has been recommended.

Prophylaxis is of the greatest importance. Great attention should be paid to the cooking of meats, especially of large joints, in order that they may be thoroughly "done." Rare meats should not be eaten.

(*b*) *Visceral Cestodes.*

(1) *Cysticercus Cellulosæ.*—It is not only as tapeworm that the system is in danger from these parasites. Human flesh may also become "measled." Although the beef measle has thus far never been found in man, some 60 or 70 cases have been noted in which the pork measle, or *cysticercus cellulosæ*, is said to have caused death. As I have explained, the embryos already contained in the links of the worm are developed into cysticerci in a second host, as swine in the case of the tænia solium, and cattle in that of the tænia saginata. When cysticerci infest human

beings the embryos may have been introduced with food or drink; but more frequently the links are regurgitated * into the stomach, and there dissolved by the gastric juice. The embryos, thus released, migrate into the tissues and become there encysted—cysticercus cellulosæ in human tissue. Among other situations in which cysticerci are found, the brain, spinal cord, and cavity of the eyeball may be mentioned. In the former a train of symptoms are developed which sometimes suggest a diagnosis. In other situations they seldom give rise to any symptoms. Even in the brain they may occupy the "silent region" without symptoms. Seated in the subcutaneous tissue they may give rise to stiffness and pain on motion.

(2) *Echinococcus Disease.*—Another effect in the human being, of tapeworm, is the echinococcus, or "hydatid cyst." This is the result of the ingestion of the embryo of a little tapeworm known as the *tænia echinococcus*, or hydatid-forming tapeworm. This little tapeworm, which is not more than $\frac{1}{5}$ inch (four to five mm.) in length, is very common in dogs. It is composed of three or four segments, of which the terminal one alone is mature. It is about two mm. (0.0787 inch) long, and 0.6 mm. (0.025 inch) wide. It contains about 5000 eggs. The head of the little worm is very much like that of the tænia solium, provided with a rostellum, two rows of hooklets, and four suckers. The embryo also has its six hooks, arranged in three pairs, by which it burrows through the intestinal wall and reaches the peritoneal cavity or muscles, or it may enter the portal vessels and be carried to the liver. By the systemic vessels it may reach the brain and other distant parts. Its most common seat is the liver. It may find entrance with food and drinking water, which may contain scores of these larvæ and yet appear pure; or the embryos may be inhaled with dust.

Distribution of Echinococcus Geographically.—Except in Iceland, where there are more dogs than persons, and Australia, where the intercourse between man and dogs is also very intimate, echinococcus disease is rare in human beings. In Iceland the ratio is put by various authorities as from one in six to one in 61 residents. In some parts of Germany it is not very rare. Thus, in Rostock, during a period of twenty-three years, there was one case of hydatid disease to 1414 residents; in Upper Mecklenburg, one in 129,000 persons, and in Greifswalde, one out of 75 autopsies disclosed the disease. It usually affects persons between twenty and forty. In Manitoba, North America, where there are settlements by Icelanders, the disease is not very infrequent. Thus, A. H. Ferguson reports that between 45 and 50 subjects have been treated in Winnipeg since 1874, when the Icelandic immigration occurred.

Of the organs and tissues invaded the liver is the most frequent, fully half of all hydatids being found in this organ. After this the kidneys, bladder, and genitals in about ten per cent.; intestinal canal, nine per cent.; lungs or pleura, eight per cent.; brain and spinal cord, 6½ per cent.; bones, 3½ per cent.; heart and blood-vessels, 3½ per cent.; other organs, about eight per cent. Of 85 cases collected by Wm.

* Portions of tapeworm thus regurgitated may even be passed by the mouth.

Osler in Canada and this country up to 1891 the liver was the seat in 59—considerably over 50 per cent.

The symptoms, diagnosis, and treatment of hydatids of the liver were given when treating Diseases of the Liver.

The symptoms of hydatids elsewhere are not distinctive. In the pleura and lungs the discharge of hooklets through a perforation in the chest wall, alone justifies a diagnosis of hydatids in the pleural cavity; the finding of hooklets in the urine, hydatid of the kidney or urinary passages. The symptoms of hydatids of the brain are those of brain tumor, the disease being more frequent on the right side. The presence of hydatid disease elsewhere is presumptive evidence that the brain symptoms are due to hydatid disease.

Treatment.—The treatment of hydatid disease, so far as any is possible, is given in connection with the diseases of the various organs in which it occurs.

III. NEMATODES, OR THREAD WORMS.

Of these the ascarides, the trichinæ, the anchylostoma, and the filariæ are the most important.

(A) The Ascarides.

(*a*) *The Ascaris lumbricoïdes.*

Description.—This, the ordinary round worm, is the most common human parasite. It is found more frequently in children, but is not rare in adults. The worm is also found in cattle, especially in hogs. It is cylindrical, pointed at both ends, yellowish, striated, and longitudinally marked by four bands. The female is 15 to 17 and 25 cm. (six, seven, and ten inches) long, the male ten to 20 cm. (four to eight inches). Its resemblance to the ordinary earth worm is so close that it is not easy to distinguish them. The ova, often very numerous in the fæces, are brownish-red and have a thick shell. They are 0.075 mm. (0.0029 inch) long and 0.058 mm. (0.0023 inch) wide.

The worms are probably developed from eggs which are swallowed in food and drink. They inhabit the upper part of the small intestine, commonly one or two, but sometimes large numbers. From this situation they may emigrate downward and pass out of the anus or upward into the stomach and œsophagus whence they may be withdrawn through the mouth or nose, or they may be vomited. They may even enter the trachea and produce suffocation, or the Eustachian tube passing out of the ear at the external meatus. They sometimes invade the common bile duct and its branches in the liver.

Symptoms.—Often there are none, the worms being accidentally discovered. At other times there are colicky pain, meteorismus, fretfulness in children, vertigo, headache, chorea, and even convulsions. Grinding of the teeth in sleep by children is regarded by the laity as a symptom of "worms." It is probably caused by gastro-intestinal irritation of any

kind. No symptom is to be relied upon which is not supported by ocular demonstration. Obstruction of the bowel has resulted from the presence of large numbers forming knotted masses or balls. If a worm enters the bile ducts obstructive jaundice is almost sure to be caused by it.

Treatment.—The remedy which has been most satisfactory in my hands is santonin in combination with calomel. Powders containing of santonin and calomel each one or two grains (0.066 to 0.132 gm.) may be prescribed rubbed up with sugar of milk. Of these one is given night and morning until the bowels are freely moved. The santonin may color the urine and produce yellow vision or xanthopsia, but I have never seen harmful results in a large experience, though poisoning manifested by convulsion is said to have been produced. For very young children the dose may be reduced to ¼ to ½ grain (0.0165 to 0.033 gm.). The worm tablets much advertised usually contain santonin as their basis. There is an officinal troche, U. S. P., containing ½ grain (0.033 gm.) of santonin. Santonica or Levant worm-seed, whence santonin is derived, is no longer used. What is known as worm-seed oil, the oil of chenopodium, another excellent remedy for round worm, is derived from the Chenopodium anthelminticum or American worm-seed. The dose is ten minims (0.65 c. c.) to a child of five years, on a lump of sugar or in emulsion,—before breakfast, dinner, and supper for two days, followed by a purge of which none is more suitable for children than calomel, itself a vermicide.

(*b*) *Oxyuris vermicularis.*

Synonyms.—*Thread Worm; Pin Worm.*

Description.—The oxyuris is a minute white worm well named thread worm, the male three to five mm. (0.1181 to 0.1968 inch) long, the female 20 mm. (0.7874 inch). Both sexes are rather blunt at one end, and finely pointed at the other. It infests the rectum and colon of children and adults, more frequently the former. It migrates and may be found in the bed, and its ova are sometimes carried to the nose under the nails used in scratching. The primary infection is believed to be by uncooked fruit and vegetables containing the ova, which may, however, be transferred from person to person, from hand to hand, or otherwise by contact. It has been suggested that they may be transmitted through the excrement of flies. The worms are very easily detected on the fæces, which contain also immense numbers of the ova. Their presence produces intense itching, especially at night, when the worms migrate. This causes restlessness and sleeplessness, and may even cause convulsions in children, and weary and exhaust adults.

Treatment.—Some perseverance is sometimes necessary to get rid of the thread worm. I usually prescribe the same powder of santonin and calomel as for the round worm, *i. e.*, one to two grains (0.066 to 0.132 gm.) of each, but at the same time order nightly injections into the rectum of vermicides, of which there are many. The infusion of quassia, lime-water, aloes, vinegar, corrosive sublimate (one to 5000), salt and water. The injection should be retained for some time, and to this end the buttocks should be raised, or the child may be placed on its

hands and knees. Only as much should be introduced—two to four ounces (60 to 120 c. c.)—as can be conveniently retained. Too large a quantity is promptly rejected.

(B) Trichiniasis.

Definition.—The name given to the invasion of the tissues of the body by the *embryos* of the trichina spiralis. The mature, sexually developed worm has its habitat in the small intestine of man and other mammalia.

Description.—The adult male is 1.4 to 1.6 mm. (0.055 to 0.063 inch) long, 0.04 mm. (0.0015 inch) thick, with a cloacum opening between two little projections in the hinder end. The female is three to four mm. (0.1181 to 0.1574 inch) long, and 0.06 mm. (0.0023 inch) thick. The head is pointed and the tail is rounded. The larva or muscle-trichina is 0.6 to one mm. (0.0236 to 0.0393 inch) long, and lies coiled in an ovoid capsule, which is at first translucent, but subsequently opaque by calcification. Man, hogs, rats, guinea-pigs, and rabbits are easiest infected.*

Man commonly acquires trichiniasis by eating infected ham insufficiently cooked. The capsules are digested, the trichinæ set free; they pass over into the small intestine and there develop into the sexually mature worm, attaining maturity about the third day. Each one discharges large numbers of embryos, of which the males die immediately after birth, while the females bore their way into the mucous membrane. A part perforate the intestinal wall and find their way into the mesentery and lymphatic glands of the mesentery. The young brood is carried away from the bowel in the lymph stream and is distributed partly through the blood stream and partly by active migration. Before birth the young trichinæ are 0.09 to 0.1 mm. (0.0035 to 0.0393 inch) long, growing slightly during migration, say to 0.12 to 0.16 mm. (0.0047 to 0.0063 inch). Their favorite seat of lodgment is the striated muscular tissue, within the striped muscular fasciculus itself, or between the muscular fasciculi and parallel to them. In nine or ten days after infection the first brood reaches its destination, to be followed by others, since the intestinal trichinæ continue to produce young throughout a life of seven weeks. A single worm, it is said, may bring forth 8000 to 10,000.

The young trichinæ begin to be encysted in the muscle about the second or third week after infection, by which time the parasite has grown to 0.8 mm. (0.0314 inch) in length. Each one arranges itself in a spiral, of which the outline is oval, and becomes surrounded by a capsule of corresponding shape, the worm cyst lying with the long axis parallel to the direction of the muscular fibres. The cyst is transparent, 0.4 mm. (0.0157 inch) long, and 0.25 mm. (0.0098 inch) wide. After five to eight months calcification may even involve the enclosed trichina itself. On the other hand, the capsule may undergo fatty degeneration, a pathological change which takes place at times early, in

* The history of the development of our knowledge of the trichina is very interesting, and may be found admirably condensed in the little book of Max Braun, "*Die Thierischen Parasiten des Menschen*," second edition, 1895.

others only after the lapse of years. The encapsulated trichina remains living and capable of development for a long time—according to Damman in hogs eleven years, while in man they have remained living twenty-five, twenty-seven, thirty, and forty years after infection. It has been shown by Zenker that the encysting is not a necessary condition to the mature development of young trichinæ.

Human infection having been conclusively shown to be due to the eating of raw pork infested with trichinæ, it is not at once evident how swine become infected. It is well known that the rats which infest slaughter houses are infected in large numbers, but it is plain also that they may acquire trichinæ by eating pork. The two probably contribute mutually to the perpetuation of the disease.

As to the distribution of the trichiniasis. Most epidemics have been in Germany. Even in America, where there have been two or three epidemics, it has been in German immigrant communities. Apparently it is more the imperfect cooking of the pork than the presence of trichinæ in it which is responsible, for although a larger percentage of American pork appears to be infected than German, yet, as already stated, the disease is much more infrequent in America than in Germany. It is to be remembered that while thorough cooking effectually destroys the parasites, thorough cooking may fail to reach the interior of large joints containing viable larva.

Symptoms.—The immigration of numerous active parasites in muscular tissue is followed by intense irritation, manifested at first by fever and muscular pain. The latter is especially severe during motion. The acts of chewing, swallowing, and breathing are particularly difficult because of the pain excited by these acts. In the early stage of the disease diarrhœa is quite common, so that certain epidemics have been mistaken for typhoid fever and as often also for rheumatism. In the very beginning of the immigration into the muscles œdema has sometimes been observed. The more general and thorough the invasion, the more intense the symptoms. Very high fever, delirium, infiltration of the lungs, and fatty degeneration of the liver have been observed. Death may take place either from exhaustion as the result of extreme irritation, or later in the disease from the same cause preceded by anæmia and gradual loss of strength. Usually, however, improvement sets in about the fourth or fifth week, though convalescence in bad cases is slow, and many weeks elapse before recovery is complete.

Diagnosis.—It is usually the unexpectedness of the disease which leads to delay in diagnosis. The resemblance of the symptoms to those of typhoid fever and muscular rheumatism has been referred to, yet in the presence of a possible cause, as, for example, a German picnic, or other feasting occasion where the favorite ham or sausage has formed part of the feast, such symptoms should immediately excite suspicion. Where doubt exists, the harpoon, devised for obtaining samples of muscle for examination, should be unhesitatingly used, under ether or local anæsthesia, and the part removed carefully examined with the microscope.

Treatment.—Here, as so often elsewhere, an ounce of prevention is worth a pound of cure. Such prevention consists in thorough

official inspection of all pork brought to market, for the evident reason that cooking may fail of its purpose for the reasons already mentioned. For a like reason swine should be grain-fed, rather than allowed to feed on offal. It is doubtful whether any direct measures can be used for arresting the disease after the muscles have been once invaded. It is a simple conflict of the mastery between the strength of the patient and the life of the trichinæ. In the majority of cases the former triumphs, though death is not infrequent from the causes named. If the disease is recognized early, the alimentary canal should be treated with vermicides and purgatives, with the view of getting rid of all the sexually mature worms which may happen to remain there, since it will be remembered that successive broods develop from the same mother-worm while in the intestinal tract. Glycerine, given in a tablespoonful (30 c. c.) dose hourly, is said to destroy the trichinæ. Benzine, in one to two drachm (four to eight gm.) doses in capsules, and picric acid in doses of five to eight grains (0.3 to 0.5 gm.), are also recommended, but regarded as less reliable. To relieve the pains, hypodermic injections of morphine, ¼ grain (0.0165 gm.), or warm baths, may be used. Restoratives and stimulants should be given to keep up strength.

(C) Anchylostomiasis.

Definition.—A term applied to the invasion of man by the anchylostoma or sclerostoma duodenale, also known as the unicinaria duodenalis, dochmius anchylostomum, strongylus quadridentatus, and strongylus duodenalis.

Description.—The male is eight to ten mm. (0.3149 to 0.3937 inch) long, and has a prominent expansion or bursa at the tail end. The female is 12 to 18 mm. (0.4724 to 0.7086 inch) long; both are provided with hook-like teeth, by which they attach themselves to the mucous membrane. The ova, which are abundant in the stools, are ovoid, 0.052 mm. (0.0020 inch) long and 0.03 mm. (0.0012 inch) broad, provided with a thin, transparent shell and deposited in a state of segmentation. The habitat of the worm is the duodenum and the upper jejunum of man and certain anthropomorphous apes, it is said, throughout the inhabited globe; but beyond some apochryphal descriptions by physicians of the South American States it is unknown here. Max Braun also says it is sporadic only in the colder countries, being more prevalent in southern latitudes, especially those of Italy and Poland; in fact it has been largely spread by laborers from these countries throughout Germany and Austria-Hungary, the West Indies, and Brazil, the St. Gothard tunnel being one of the localities in which it was first studied. The larvæ are developed in damp earth and distributed by wind and water. Many of the victims are earth-eaters.

Symptoms.—The parasite fastens itself to the mucous membrane by the hooked teeth described, and feeds upon blood drawn therefrom. At first only causing gastro-intestinal irritation, there gradually results an anæmia, variously known as the Egyptian chlorosis, brickmaker's anæmia, tunnel anæmia, and mountain anæmia. The rate of development of the anæmia

varies, being sometimes very rapid. There occur also colicky pains, diarrhœa, and small hemorrhages. Hypertrophy and dilatation of the heart have been found.

Diagnosis.—The diagnosis is rendered easy by the conditions surrounding the victims and confirmed by the discovery of eggs in the fæces. At autopsy they may be found clinging to the mucous membrane in the duodenum and upper ileum.

Treatment.—In treatment, prophylaxis is most important. All water used by laborers should be disinfected. Thymol is said to be a specific and should be given in 30-grain (two gm.) doses in wafers at 8 A. M. and 8 P. M., a purge of castor oil or magnesia being given after the second dose. Should the first effort fail it may be repeated in a week. The diet should be liquid, milk, and soups.

(D) Filariasis.

A condition constituted by the presence of several species of nematode worms, known as filariæ.

(*a*) *Filaria sanguinis hominis.*

A term applied to the several varieties of filaria which infest the blood of man. Of these, three species will be described :—

(1) *Filaria Bancrofti.*—The adult filaria thus named was first discovered by Bancroft in Queensland in 1876, and soon thereafter by T. R. Lewis in Calcutta. Before these discoveries the larva only was known, having been discovered by Demarquay in Paris in 1863, in the hydrocele fluid of a Havanese.

The adult male is 83 mm. (3.2677 inches) long and 0.4 mm. (0.0157 inch) in diameter, head rounded, tail pointed, and spirally rolled. The female is about 155 mm. (6.1023 inches) long and 0.7 mm. (0.0275 inch) thick. The egg is 0.038 mm. (0.0015 inch) long and 0.014 mm. (0.0005 inch) wide. The normal habitat of the sexually mature worm is the lymphatic vessel of different parts of the body of man, though it has been found also in the left ventricle of the heart. The female is viviparous, exceptionally oviparous, producing an enormous number of young larvæ, which pass from the lymphatic stream into the blood and are thus distributed over the body. They are 0.270 to 0.340 mm. (0.0106 to 0.0133 inch) long, 0.007 to 0.011 mm. (0.0003 to 0.0004 inch) broad, rounded anteriorly, pointed behind.

The larvæ are found in the peripheral circulation after sundown in a drop of blood taken for the purpose. The numbers increase until midnight when they again become less numerous. From midday to evening there are no filariæ to be found. The cause of this interesting fact cannot be ascribed to the periodic production of broods, since Mackenzie has shown the order is reversed if the patient sleeps during the day and is awake during the night, under which circumstance the filariæ are found in the blood in the day. This is, however, not without exception. It would seem, as suggested by Linstow, that the event has something to

do with sleep and that during sleep the peripheral blood-vessels widen and again contract during awaking ; that the filariæ cannot pass the contracted capillaries of the superficial skin, but rest in the larger blood-vessels in the deeper portion, to come out with the widening which accompanies sleep. Their number in the blood has been variably estimated at 140,000 by Carter, and by Mackenzie at 30,000 to 40,000.

Philip Manson discovered the young filaria, together with human blood, in the intestine of the mosquito, and thus secured the key to the problem of the transmission of the parasite from one person to another. He also noted that the larvæ underwent a certain degree of development in the intestine of the mosquito, changing in form and size. On the sixth or seventh day they are 1.5 mm. (0.0599 inch) long, and cylindrical, and at this time change their habitat to water through the death of the mosquito, which takes place after the deposit of eggs. Few mosquitoes, however, live long enough to permit the development in their intestine of the filaria to the stage at which it is sufficiently mature to survive in water. Manson concludes, however, that man becomes infected by drinking water which holds the mature filariæ. Manson's studies have been confirmed by Lewis, and, although his conclusions are not undisputed, they seem likely. The filaria Bancrofti is met with in the blood in all tropical countries, especially India, China, Japan, and Brazil. It has also been found in Southern United States in persons who have never been out of the country, first by Guiteras and since by others.

(2) *Filaria Perstans.*—This second variety of filaria sanguinis hominis was also found by Manson in a larval state in the blood of a negro in West Africa. It is distinguished from the other filariæ of man by its small size—0.2 mm. (0.0078 inch) long—its active motility and contractility. Manson is inclined to ascribe to this the sleeping disease of negroes, and to it also the skin affection known among negroes as *kraw-kraw*, which is a papillo-pustular eruption, probably the same as Nielly's *dermatose parasitaire*, the parasite of which was called *Rhabditis Nielly* Blanchard. The posterior extremity is obtuse ; the anterior has a retractile rostellum.

(3) *Filaria Diurna.*—Also described by Manson, is known only in the larval state, agreeing in this respect with filaria Bancrofti, differing, however, from the latter in that it appears in the blood only in the day. It was discovered by Manson in the blood of negroes on the west coast of Africa. Manson suggests *filaria loa* represents the adult stage.

Symptoms.—The early presence of filariæ in the blood does not occasion subjective symptoms, and may last years without impairment of health. Sooner or later, however, as a rule, there appear anæmia, enlargement of the spleen, and fever, with lymphatic tumors in different parts of the body, especially in the testicle and spermatic cord. Later there develops in consequence of lymphatic obstruction, possibly caused, according to Manson, by the ova, elephantiasis, especially of the scrotum (lymph scrotum) and lower extremities. To these succeed enlargement of the lymphatic glands, chyluria, or hæmato-chyluria already described, chylocele, more rarely nephritis, pyelitis, cystitis, and even peritonitis. Parasites are by no means always found in the blood in cases of elephantiasis or lymph scrotum.

Treatment.—No treatment appears to be of any value in exterminating the filariæ in the blood. The symptomatic treatment has been considered in treating of chyluria.

(*b*) *Filaria dracunculus.*

SYNONYM.—*Dracontiasis, or Guinea-Worm Disease.*

Description.—Dracontiasis is a term given to the presence of the filaria dracunculus or filaria medinensis, or guinea-worm.

The female, until recently alone known, is 50 to 80 cm. (20 to 32 inches) long and longer, and 0.5 to 1.7 mm. (0.0196 to 0.0669 inch) thick. Quite recently R. H. Charles found, along with two females removed from a dead body, at Lahore, a much shorter worm about four cm. (1.6 inch) long, attached by its hinder extremity to one of the females at a point about four cm. (1.6 inch) from its head, which he inferred to be the male attached to the vagina of the female, dying after impregnation, as is the case with some other parasites. Then, too, the vagina atrophies, as both it and the vulva are absent in the mature worm.

The mature female resides in the subcutaneous and intermuscular connective tissue, especially in the lower extremities, about the foot-joint. To this it obtains entrance through the stomach, whence it penetrates the intestine and passes to the subcutaneous connective tissue where it attains its full development, lying for a long time quiescent under the skin, where it can be felt like a cord. Later it excites suppuration, and with the rupture of the abscess is discharged. It is also found elsewhere, as in the back, scrotum, perineum, the upper extremities, eyelids, and tongue. Usually one worm only is found, rarely several. It is widespread, occurring in all races, ages, and both sexes. Van Harlingen described a case in a man who had always resided in Philadelphia. According to Braun, the fiery serpents described by Moses were guinea-worms. The term *Δρακοντιον* was applied by Agatharchides one hundred and forty years before Christ, and Galen called the disease dracontiasis.

As to the development of the worm, the mature female contains enormous numbers of living embryos, which are discharged into the water of ponds. After a few days they probably enter the cyclops, a small crustacean, and there reach a certain stage of development, when they are imbibed with drinking water by man and reach their ultimate destination as described.

They are easily recognized under the circumstances named.

Treatment.—As to treatment, the indication is to remove the worm intact after the abscess is opened, because of the irritation excited by the escape of the living embryos. The method of procedure directed is to roll the worm around a piece of smooth wood, each day a little, in order to prevent retraction, until the whole worm is withdrawn.

It is said that the leaves of the plant *amorpattee* are a specific cure, while asafœtida, when given in full doses, is also said to be poisonous to the worm.

(*c*) *Other Filariæ.*

Numerous other filariæ, of less importance, have been found from time to time in man. Among them I select from Braun :—

(1) The *filaria immitis*, the common filaria sanguinis of the dog, found twice in man by Bowlby ; in one instance associated with hæmaturia. He found in the case of an Arab numerous filariæ in the portal veins, eggs in the thickened bladder-wall, kidneys, ureters, and the lungs ; in another, eggs were found in a tumor of the rectum in a seventeen-year-old youth.

(2) *Filaria loa*, a delicate worm, 30 to 40, rarely, 70 mm. (1.11 to 1.57 to 2.75 inches) long, found between the conjunctiva and the eyeball in negroes on the West Coast of Africa, whence it has spread to South America and the West Indies.

(3) *Filaria oculi humani vel lentis*, which in several instances was removed with cataracted lenses, of which it seems to have been the cause.

(4) *Filaria labialis*, found in a pustule in the lips of a child.

(5) *Filaria hominis oris* (Leidy), obtained from the mouth of a child.

(6) *Filaria lymphatica vel bronchialis* found in lymphatic glands of the lungs, and in the trachea and bronchi.

(E) Other Nematode Worms.

(1) The *tricocephalus dispar*, an interesting worm of which the anterior portion, equal to three-fifths of the body, is very delicate and hair-like, while the hinder portion is much thicker. It is four to five cm. (1.6 to two inches) in length, the male being somewhat shorter. The hinder end of the female is conical and pointed, while in the male it is more obtuse and rolled like a spring. The ova are oval, 0.05 mm. (0.0019 inch) long, and provided with a button-like projection like that on the end of a lemon.

Its habitat is usually the cæcum of man, rarely the appendix vermiformis, and exceptionally the small intestine. Usually they are few in number and give rise to few or no symptoms. In some instances large numbers are found and serious brain symptoms have been ascribed to them,—in other cases anæmia. It is said to be one of the commonest parasites in man the world over, in either sex and at any age except infancy. Beriberi has been ascribed to it. The larvæ are probably developed from the eggs in water, and are possibly ingested in drinking water. The ova are very resisting to the destructive agents to which they are ordinarily subjected. Large numbers are found in the fæces.

(2) The *eustrongylus gigas* or strongylus gigas is an enormous nematode, the male of which measures 240 cm. (2.1 feet), and the female 100 cm. (40 inches) long and 12 mm. (0.5 inch) thick. It lives in the pelvis of the kidney, more rarely in the abdominal cavity of the seal, dog, wolf, horse, and other animals, and exceptionally in man.

(3) The *strongyloides intestinalis* or anguillula intestinalis et stercoralis is a small nematode worm, first found in 1876 in the stools of French soldiers in Cochin China suffering with severe diarrhœa. They

are found in all parts of the intestine and in the biliary and pancreatic ducts, producing diarrhœa only when present in large numbers.

IV. ACANTHOCEPHALI.

Synonym.—*Thorn-head Worms.*

Description.—These are nematode-like worms in the intestinal canal which are provided at the anterior end with a retractile proboscis provided with hooks, hence called thorn-headed.

Of these the *gigantorhynchus* or echinorrhynchus gigas is an enormous worm, the male being ten to 15 cm. (four to six inches), and the female 30 to 50 cm. (12 to 20 inches) long, which attains its full development in the intestine of the hog, attaching itself to the mucous membrane by its thorn head. The intermediate host is the cock-chafer grub in America and the June bug. As these insects may be accidentally swallowed by man it is not impossible that the parasite may develop in his intestine.

V. ARTHROPODA.

Of these both the arachnides and insecta contribute to human parasites.

(A) Arachnoidea.

(*a*) *Acarinæ.*

(1) *Sarcoptes or acarus scabiei,*—the itch insect. This is the most frequently met with of the arachnide parasites. Its oval, nearly circular little body, provided with horns and bristles, is barely visible to the naked eye under favorable circumstances, the male being 0.2 to 0.3 mm. (0.0078 to 0.0118 inch) by 0.145 to 0.19 mm. (0.0057 to 0.0074 inch); the female 0.33 to 0.45 mm. (0.0129 to 0.0177 inch) by 0.25 to 0.35 mm. (0.0098 to 0.0137 inch).

The female lies at the end of a burrow made for itself in the epidermis in situations where the skin is most delicate, as between the fingers, in the elbows, and under the knees, in the groin, and on the penis, very seldom in the face, but in any delicate part. In this burrow, some millimeters to a centimeter long, the female deposits her eggs. The male is seldom seen, dying after copulation, and the female after depositing her eggs. The eggs hatch in from four to eight days, and in about fourteen days the larvæ are sufficiently matured to make their own burrows. The disease is communicated by personal contact or by clothing.

Symptoms.—These are first an intense itching which incites to scratching, which, in turn, causes excoriations, papules, vesicles, and pustules.

Diagnosis.—The diagnostic feature is the shining little vesicle readily recognized by a moderate magnifier in the webs of the fingers, though it is often obscured and obliterated by the eruption and marks caused by scratching.

Treatment.—This is very simple. Sulphur ointment is a prompt specific. The body should be first bathed thoroughly with soft soap, and then as thoroughly anointed with the ointment, which should be left on until the next day, when there should be another bath, followed by another vigorous application of the ointment. Three or four days of this treatment should suffice. An ointment of naphthol, one drachm to the ounce (four gm. to 30 gm.), is recommended.

(2) *Demodex folliculorum*, a minute parasite 0.3 to 0.4 mm. (0.0118 to 0.0157 inch) long, which resides in the sebaceous follicles, with the grease of which it can sometimes be squeezed out. It is oftenest met with on the face and nose. It is said to be present in about 50 per cent. of persons, but this is probably exaggerated. It usually gives rise to no symptoms, but is said sometimes to be the cause of obstruction of the follicles and produce thus the little worm-like accumulations of fat which may be squeezed out of the follicles, and which cause inflammation and acne.

Treatment.—Acne is well treated by a lotion of corrosive sublimate, two to 1000, and it may be by its effect on the demodex that it is useful.

(3) *Leptus autumnalis*, or harvest bug. This is a minute red parasite 0.3 to 0.5 mm. (0.0118 to 0.0196 inch) long, which has three pairs of legs, with rows of bristles upon its back and belly. It prevails in summer on grasses and plants, attaches itself to the skin of man and animals by its hooklets, and gives rise to irritation.

Treatment.—It is successfully destroyed by sulphur ointment and corrosive sublimate, two to 1000.

(4) The *ixodes ricinus.*—This is a minute oval tick, the male 1.2 to two mm. (0.0474 to 0.0787 inch) long, and brownish-red in color, the female four mm. (0.1574 inch) long, yellowish-red, when distended with blood, bluish-grey, 12 mm. (0.4724 inch) long, six to seven mm. (0.2362 to 0.2755 inch) broad, which infests the skin of sheep, cattle, dogs, horses, and men, causing irritation and inflammation.

It may generally be removed by rubbing or greasing with any sort of oil or vaseline.

(*b*) *Linguatulidæ, or Pentastomes.*

The pentastomes include the pentastomum tænioides, or linguatula rhinaria, and the pentastomum constrictum, or porocephalus constrictus.

(1) The *pentastomum tænioides* is a lancet-shaped worm, already described in connection with parasites of the liver. The adult form infests the frontal sinuses and nostrils of the dog, more rarely of the horse, and has been found in the nostrils of man.

(2) The *pentastomum constrictum* has as yet been met with only in the larval state. It is milk-white in color, with golden-yellow hooklets, 13 mm. (0.5118 inch) long and 2.2 mm. (0.0866 inch) wide, provided with 23 rings. It has been found by Pruner encysted in the liver of two negroes in Cairo, and by Bilharz in two instances encysted in the liver and mucosa of the bowel, and by Aitken in the liver and lungs of an English soldier in the West Indies.

(B) Insecta.

Of these the order *rhyncota* is represented by pediculi or lice, and the cimex or bed-bug, the *diptera* by the pulex or flea.

(*a*) *Rhyncota.*

Pediculi (*phtheiriasis:* pediculosis).—Of these, three varieties infest human beings of filthy habits.

(1) The *pediculus capitis*, or head-louse.—The male is one to 1.5 mm. (0.0393 to 0.0590 inch) long, the female 1.8 to two mm. (0.07080 to 0.0757 inch) long. The color varies somewhat with the races. In the white it is grey with a dark border, in the negro and Chinaman darker. Its eggs are 0.6 mm. (0.0236) long, of which the female lays about 50, which mature in about a week and in eighteen days are ready to reproduce. The eggs are attached to the hairs and are easily visible, being known as nits.

The head-louse is found the world over, upon the hairy heads of men and sometimes in other parts of the body where there are hairs. Even when they are quite numerous they may produce no symptoms. Generally, however, they cause itching and scratching, especially when the louse bores deep into the skin and produces pustular dermatitis, with resulting crusts and scabs in which the hair becomes matted and tangled, forming the *plica polonica*, so called from its frequency in Poland.

(2) The *pediculus vestimenti*, or body-louse, is considerably larger, being two to five mm. (0.1574 to 0.1968 inch) long, and whitish-grey in color, the back part of the body being wider than the thorax. Its eggs are 0.7 to 0.9 mm. (0.275 to 0.0354 inch) long, and about 70 are laid by the female. It lives on the clothing in which it deposits its eggs, about the neck, back, and abdomen. The puncture incident to sucking is often covered by a hemorrhagic point. It, too, causes itching and scratching, with irritation and inflammation of the skin, and in old cases a roughness and pigmentation causing dark spots and a condition known as *morbus errorum* or vagabond's disease, which has been mistaken for Addison's disease.

(3) The *pediculus pubis*, phthirius inguinalis, or crab-louse, is smaller than the head-louse, greyish-yellow or greyish-white, the male 0.8 to one mm. (0.0314 to 0.0393 inch) long, the female 1.12 mm. (0.0441 inch) long. The eggs are pear-shaped, 0.8 to 0.9 mm. (0.0314 to 0.0354 inch) long, 0.4 to 0.5 mm. (0.0157 to 0.0196 inch) wide. They infest the parts of the body covered by shorter hairs, such as the pubis, axilla, and eyebrows. The pediculus pubis does not wander as much as the pediculus capitis or vestimenti, but adheres more closely to the skin and can often be removed with difficulty.

These lice rarely give rise to symptoms.

Treatment.—Of *pediculosis.* For the head-lice. The hair should be cut short and burned, the head thoroughly washed with soap and water, and then anointed with mercurial ointment, or washed with tincture of cocculus indicus, or with coal-oil or turpentine, or carbolic acid, one

to 50. Cocculus indicus is to be preferred because of its freedom from odor. The washing should be repeated for several days in succession.

The treatment for the crab-louse is the same, but as mentioned it adheres firmly to the skin and it is generally necessary to pick off the individual louse.

To get rid of the body-louse the clothing, if not too valuable, should be burned, but may be boiled, or when this is not admissible treated by superheated steam.

The itching promptly disappears with its cause, but if necessary it may be further allayed by a warm bath to which four or five ounces (120 to 150 gm.) of sodium bicarb. are added.

Repeated bathing with soft soap should be done until it is absolutely certain that the parasite and its ova are removed.

(4) *Cimex lectularius* or common bed-bug.—This familiar insect is reddish-brown, oval in shape, four to five mm. (0.0574 to 0.1968 inch) long, three mm. (0.1181 inch) wide. The female lays three or four times a year about 50 eggs, 1.12 mm. (0.0441 inch) long, which require about eleven months for their perfect development to the sexually ripe condition. They live in the crevices of beds and of floors and rafters, in furniture, behind wash-boards and wall-paper, in the habitations of man. During the day they lie concealed; at night they wander in search of the blood of the human being, which they draw by means of a long proboscis. The peculiar odor of the insect is due to a secretion of a special organ with which the bug is provided.

Human beings are variously susceptible to the bite of the bed-bug, some being quite indifferent to it, others being, as it were, special favorites of the little creature.

Treatment.—The irritation is confined to the moment of the bite. The aim to be sought is the extermination of the insect. This is often difficult when a thorough lodgment is secured, and it is often necessary that all wall-paper should be removed as well as loose woodwork. Bedsteads should be thoroughly scalded, and then treated with the following: two tablespoonfuls of metallic mercury should be thoroughly beaten up with the white of one egg until a froth is attained. Apply freely with a small paint brush, filling in carefully all cracks and crevices. The pest is less apt to invade iron bedsteads, but even these must not be neglected, for they, too, in careless hands, may become infested. Solution of corrosive sublimate, one to 1000 may also be applied in the same manner.

(*b*) *Diptera.*

(1) *Pulex irritans*, or common flea.—Of these little creatures the male is two to 2.5 mm. (0.0787 to 0.1181 inch) long, the female as much as four mm. (0.1574 inch), red or dark-brown in color. It also is highly capricious in its tastes, disturbing some persons not at all, others seriously. It is not a parasite of man and invades him usually because of its great abundance in certain places and countries. Though of world-wide distribution, it is more troublesome in hot countries where cleanliness of

household, city, and person are matters of indifference. The eggs are not laid on human beings, but in the cracks of boards, sweepings, and wooden spit-boxes.

Treatment.—The essential oils cause the retreat of fleas when applied to infested parts.

(2) The *pulex penetrans*, or sand-flea or jigger.—The female buries herself in the skin of human beings as well as dogs, swine, and other mammals, producing painful irritation, circumscribed swelling, and even suppuration. It especially attacks the feet. It prevails in tropical countries, especially in Central and South America. The eggs are land-hatched.

Treatment.—The flea may be picked out with a needle, after which the essential oils are rubbed in on the parts to keep it away.

(3) *Myiasis.*—The diptera also contribute to parasites through their larvæ, which are deposited sometimes in open sores which have been neglected, and sometimes in the nasal passages and cavities, the ear, pharynx, vagina, etc. This condition is called myiasis, from the Greek μυον, a fly.

The most common of these is *myiasis vulnerum*, in which an ulcer becomes filled with maggot, which are the larvæ of the blue bottle or common flesh fly, *sarcophaga carnivora.*

Myiasis narium, aurium, conjunctivæ, vaginæ, etc., is due to the *lucilia macellaria*, whose larva is deposited in these situations usually when they are diseased, and may produce serious mischief, perforating mucous membrane and even cartilage. The larvæ of the *lucilia nobilis* have also been found in the auditory passages, producing ringing of the ears as a symptom. The larvæ of *sarcophaga magnifica* have been found in ulcers and other situations, throughout Europe and especially in Russia.

Cutaneous myiasis is commonly due to the larva of the *hypoderma bovis* or bot fly, the female of which lays her eggs on the skin of cattle and sheep, in which the larva bores its ways and forms the gad boil, about as large as a pigeon's egg. Rarely in tropical countries this happens in the skin of man. Cutaneous myiasis is sometimes caused by the larva of the *musca vomitoria*, one of the domestic flies. More frequently it causes internal myiasis, having been swallowed and again discharged by vomiting. More rarely dipterous larvæ are found in the fæces, including those of the common house fly and the *trichomyza fusca*, which has also been vomited.

SECTION XV.

SUMMARY OF SYMPTOMS FOLLOWING OVER-DOSES OF POISONS.

(*Alphabetically Arranged.*)

To Which is Added a Table of Minimum Dose Which Has Caused Death, and Maximum Dose Followed by Recovery.

Alcohol.—Taken in the form of spirituous beverages. Acute alcoholic poisoning. A brief period of excitement, with flushing of the face, followed by unconsciousness, stertorous breathing, rapid and, finally, weak pulse, vomiting, a subnormal temperature, delirium, complete muscular relaxation, at times convulsions; the pupils are usually dilated. Usually recovery takes place in a day or two, but remissions may occur. Odor of alcohol on the breath.

Treatment.—Evacuation of stomach by pump; emetics, like apomorphine, $\frac{1}{10}$ grain (0.0064 gm.); washing out the stomach. Stimulation by ammonia, coffee, digitalis, strychnine, or even faradic current to muscles of respiration.

Delirium Tremens.—Delirium, with hallucinations; great restlessness and insomnia; slight fever, pulse rapid and soft.

Treatment.—Withdrawal of alcohol; bromides in large doses, or chloral, aided by a cold bath to produce sleep; nourishing food, and stimulation if the condition demands it, even alcohol.

Ammonia.—Taken by mistake, or with suicidal intent, in the form of "household ammonia," water of ammonia, spirit of hartshorn, and in liniments.

Symptoms.—At once, burning pain in the mouth, throat, œsophagus, and stomach; the lips and tongue are intensely swollen and inflamed; vomiting of blood-tinged mucus, suffocative cough, with rapidly increasing dyspnœa. The face is pale, pulse is rapid and thready, and collapse soon develops. Death may follow at once, or some days later, from the violent gastro-enteritis and stricture of the œsophagus.

Diagnosis.—Smell of ammonia on the breath, vapors of the corresponding salt when a rod dipped in hydrochloric acid is held before the mouth, together with the sudden onset of the symptoms.

Treatment.—Neutralization with vinegar, or some other dilute acid, at once, the acid being mixed with some bland oil, if possible. If the

patient lives, treat the results of the violent inflammation. Tracheotomy, if danger of death from œdema of the larynx.

ANTIMONY.—Taken as tartar emetic, the tartrate of antimony and potassium. A heavy, white, odorless, slowly soluble powder, having a sweetish, metallic taste; charring on heating to redness.

Symptoms.—Metallic taste in the mouth, burning pain in œsophagus, stomach, and abdomen, dysphagia, violent vomiting and purging of serous material, cramps in the stomach and muscles of the arms and legs. Finally the symptoms of collapse, cold, clammy skin, great depression, respirations shallow, pulse weak and thready. There may be convulsions, delirium, and coma.

Treatment.—If no vomiting, apomorphine $\frac{1}{10}$ grain (0.0064 gm.), or another emetic; tannic acid as a chemical antidote, followed by washing out of stomach; external heat, stimulants to combat the collapse, and opium when the acute symptoms have disappeared.

ARSENIC.—Used in the form of arsenious acid in rat-poisons, in fly-paper, to preserve stuffed birds and animals. Paris green, used as potato-bug poison, is an arsenite of copper, hence the symptoms are similar. Arsenious acid, or white arsenic, is an odorless, tasteless white powder, quite heavy, and but slowly soluble in water.

Symptoms.—Appear usually in the course of an hour and are those of violent gastro-enteritis, so severe as to suggest Asiatic cholera. Burning pain in throat and stomach, persistent vomiting of brown matter streaked with blood, though the vomited matter may be green from bile. Purging of serous and bloody material, and finally collapse. Fatal in a day or two, though a remission sometimes occurs on the third day. Nervous symptoms may appear, and at times a case of arsenic poisoning closely simulates acute yellow atrophy of the liver.

Treatment.—Early stage, stomach-tube or emetics. Chemical antidote, the freshly prepared hydrated oxide of iron, made by precipitating the solution of tersulphate of iron by ammonia; the tincture of chloride of iron may be used instead of the solution. Mix and strain, wash and administer the magma. The best antidote is the ferri oxidum hydratum, U. S. P., or ferri oxidum hydratum cum magnesia, the latter acting also as a purgative. Otherwise treat the collapse.

ATROPINE (*Belladonna*).—The deadly nightshade. Used as a mydriatic and in liniments. The leaves impart a narcotic odor to the tincture, but recognition depends upon the physiological effect.

Symptoms.—The throat is dry, the pupils are dilated; pulse rapid and hard; respirations quickened and deepened; great restlessness, occasionally talkative delirium. An erythematous rash is sometimes present. The urine contains the alkaloid, hence it will cause dilatation of the pupil if dropped into the eye of an animal.

Treatment.—Tannic acid, followed by the stomach-tube or emetics. Physiological antidotes: Morphine, physostigmine, and pilocarp-

ine. Artificial respiration for two hours. Catheterize, as patients frequently suffer from retention. Meet collapse by proper stimulation.

Aconite (*Monkshood, Wolf's Bane, Blue Rocket*).—All parts poisonous. The tincture may be mistaken for sherry or whiskey; it has an exceedingly acrid taste.

Symptoms appear quickly, consist of an acrid taste in the mouth, a feeling of warmth in the stomach, followed by a tingling sensation throughout the body; muscular weakness, slow, weak pulse; vomiting may be present; collapse. The mind is clear to the last.

Treatment.—Stomach-tube or emetics; recumbent posture, with feet elevated somewhat. Stimulants freely, such as ammonia, ether, digitalis, atropine, and strychnine. Heat to the extremities and artificial respiration for two hours.

Bromine.—A dark red, very heavy liquid, emitting reddish vapors resembling chlorine.

The fumes, when inhaled, cause convulsive cough, bloody expectoration, dyspnœa, and spasm of the glottis.

Treatment.—Fresh, moist air and cautious inhalations of ammonia.

Bromism.—The symptoms of chronic gastro-intestinal disturbance, such as fetor of the breath, anorexia, diarrhœa; great depression of all the functions, especially the sexual function, with languor and mental apathy. A general eruption of acne is an early sign.

Treatment.—Stop the administration and aid elimination.

Carbolic Acid (and Creasote) is a colorless or reddish liquid, or a crystalline solid, when pure; when impure, the color varies from this to black. Has a characteristic odor.

Symptoms appear quickly. Burning pain in the mouth and stomach, though there may be no pain. Vomiting may be absent. Soon coma sets in, with feeble respirations, collapse, and convulsions. The urine is smoky, but the most characteristic sign is the white eschar on mouth and lips, with the odor of the acid on the breath.

Treatment.—Any soluble, non-poisonous sulphate, such as sulphate of magnesium, or sulphate of sodium, as a chemical antidote. Stomach-tube or emetics, followed by washing out stomach with a solution of a sulphate. Oil to counteract the escharotic effect. Treat the collapse with heat, stimulation, etc.

Carbonic Acid Gas.—The choke damp or after damp of miners. May be accidentally inhaled in over-crowded rooms, in fermenting vats, over lime kilns, or wherever the products of complete combustion cannot escape.

Symptoms.—Headache, dizziness, vomiting, and great drowsiness, with relaxation of the muscles; hurried respiration, with violent action of the heart. Soon coma ensues.

Treatment.—Fresh air, if need be; artificial respiration, kept up steadily and unceasingly; ammonia by inhalation; oxygen, if obtainable; cold douche to the head and chest, with stimulation as occasion requires.

Carbonic Oxide (*Carbon Monoxide*) is formed during the incomplete combustion of carbon and is a direct poison, while carbonic-acid gas, the product of complete combustion, kills merely by exclusion of oxygen. Treatment as for carbonic-acid gas.

Caustic Potash, or Soda.—Taken in the form of "lye."

Symptoms.—An acrid, burning taste, the burning extending down to the stomach, followed by vomiting, purging, and collapse. The mucous membrane of the mouth shows evidence of corrosion.

Treatment.—Olive oil, to saponify the alkali; demulcent drinks, dilute acids, like vinegar and lemon-juice, to neutralize. If the patient lives, the resulting stricture of the œsophagus requires dilatation.

Cheese Poisoning.—Decayed cheese owes its poisonous properties probably to tyrotoxicon. Usually there is severe gastro-enteritis, with vomiting and purging.

Treatment.—The stomach-tube may be used if vomiting has not been very free; subsequent lavage; sedatives for the irritation.

Chloral.—A popular somnifacient and sedative. Occurs in deliquescent crystals with characteristic odor, and acrid, burning taste.

Symptoms.—Profound unconsciousness, complete muscular relaxation; sensibility diminished or lost; the pulse becomes feeble and rapid, the respirations are diminished in frequency and may be stertorous; the temperature is depressed more than by any other toxic agent.

Treatment.—Evacuate the stomach; keep up the temperature by application of heat; rouse the patient; stimulate, use artificial respiration in conjunction with the battery.

Chloroform.—Identified by its peculiar, ethereal odor and sweet, pungent taste; a heavy, volatile, non-inflammable liquid, not miscible with water.

Symptoms.—First stage of narcosis, excitement, muscular rigidity, lessened sensibility to pain. Second stage, muscular relaxation, anæsthesia of conjunctiva, insensibility to pain. Third stage, stertorous breathing, dilated pupils (not responding to light), abolition of all reflexes, muscles absolutely relaxed. Death usually by failure of circulation. The symptoms are the same if the chloroform is taken by the mouth.

Treatment.—In case the chloroform was used as an anæsthetic, lower the head, slap a wet towel on the patient's chest; pull out the tongue to see that the mouth is clear; artificial respiration at the rate of 20 a minute, aided by the cautious use of the battery (interrupted current).

When taken internally, stomach-tube or emetics; flicking with a wet towel; stimulation with coffee by rectum; whiskey by mouth, if possible.

Conium (*Poison Hemlock; Common or Spotted Hemlock*).—Not a native of this country, hence cases of poisoning must be restricted to the use of the preparations. The plant and some of the preparations have a peculiar odor, resembling the urine of mice.

Symptoms.—Prominent is the loss of muscular power, the patient staggering as if intoxicated; the arms and chest are affected later on, and death may ensue from paralysis of the muscles of respiration. In other cases there are delirium and stupor, coma and convulsions; the pupils are dilated, and ptosis is a peculiar symptom.

Treatment.—Stomach-pump or emetics; tannic acid, stimulants, heat, artificial respiration.

Copper.—The sulphate, blue-stone, or blue vitriol, is used in the arts. Recognized by its crystalline shape, blue color, and acrid, metallic taste. Articles of food are frequently prepared in imperfectly cleansed copper kettles; a bright piece of steel, such as a knife, will show a deposit of metallic copper a few minutes after immersion in a liquid containing copper. Verdigris (subacetate) is another source.

Symptoms.—A metallic, astringent taste in the mouth; griping and colicky abdominal pains; vomiting, purging, accompanied by tenesmus, the stools being mucous or bloody. Respiration embarrassed, small, quick pulse, weakness, great thirst, coma, death.

Treatment.—Stomach-pump or emetics, if vomiting has not emptied the stomach; demulcents, like milk and eggs; chemical antidotes are soap, sodium carbonate, and the yellow prussiate of potash; opium as a sedative.

Cocaine.—Solution used as a local anæsthetic, particularly in eye-surgery.

Symptoms.—Vertigo, headache, paroxysmal dyspnœa, rapid, weak pulse, elevated temperature, mental excitement, blindness, delirium, coma, and convulsions. Some of these may be caused by the local application of solutions to mucous membranes. The pupil is dilated, but the power of accommodation remains in part.

Treatment.—Nitrite of amyl, stimulants, atropine, caffeine, and ammonia. Death is unusual.

Digitalis (*Foxglove*).—A native of Europe. The tincture has a distinct odor of tea, the drug itself lacking a narcotic odor.

Symptoms.—Vomiting and purging of green material; pulse slow, later rapid and irregular; headache, occasionally delirium and convulsions; the skin is cold and clammy, the pupils dilated. Coma and death come on quite suddenly, though the mind may be perfectly clear.

Treatment.—Stomach-pump, or emetics, if necessary; tannic acid, 20 grains (1.33 gm.), repeated frequently; strong tea; stimulation with whiskey, ammonia, or hot coffee; maintain recumbent position for some time after all symptoms have subsided.

Ergot.—Used to produce abortion.

A large dose produces cramps in the legs, arms, and chest, dizziness, weakness, pulse small and pupils dilated, skin cold ; there may be vomiting and diarrhœa.

Chronic ergotism, produced by eating bread tainted with ergot of rye, causes muscular cramps, paresis, delirium, and convulsions ; in other cases a dry gangrene. Occurs in Europe.

Fish-Poisoning.—Tainted fish probably contains ptomaïnes, while several kinds of fish are constantly poisonous.

Symptoms.—Vomiting, irritation of the eyes, great depression, and severe nettle-rash.

Treatment depends upon the symptoms.

Hydrochloric Acid.—See Mineral Acids.

Hydrocyanic Acid (*Prussic Acid*).—The pure acid is an exceedingly poisonous gas ; in medicine it is employed as a two per cent. aqueous solution. Contained in oil of bitter almonds distilled from the seed ; not found in the artificial or the purified natural product. Its salt, the cyanide of potassium, is employed as a quickly-acting poison for the destruction of animals. Contained also in cherry-laurel water.

Symptoms.—Patient is nearly always insensible in two minutes if a fatal dose is taken. Loss of motor power, giddiness, slow respirations, pupils insensible to light, eyes protruding, pulse weak ; frothing at the mouth, perhaps tetanic convulsions. The odor of bitter almonds is about the patient.

Treatment.—Stomach-tube or emetics, stimulants, hot and cold douche ; artificial respiration, kept up steadily, as the patient is probably safe if tided over the first half-hour. Mild interrupted current to region of heart.

Iodine.—May be taken by mistake ; the tincture has the odor of iodine, which somewhat resembles chlorine.

The symptoms of a violent gastro-enteritis, such as burning pain in the throat, stomach, and abdomen, with vomiting and purging. The vomited matter may be yellow from iodine, or blue if starch is present in the stomach.

Treatment.—Starch (arrow-root) in any form as chemical antidote ; stomach-tube or emetics ; opium as sedative.

Iodoform.—Rarely taken internally, but toxic symptoms may appear when used freely as an antiseptic dressing.

Most marked among these are great somnolence, slight nocturnal delirium, headache, hurried breathing, and rapid pulse. These, with a slight elevation in temperature, often resemble cerebral meningitis. A rash may accompany the intoxication.

Treatment.—If taken internally, emetics, etc. Substitute another antiseptic as a dressing and aid elimination.

Lead.—Taken as sugar of lead (acetate), Goulard's solution (subacetate), lead water (subacetate), white lead (carbonate).

Symptoms.—In acute poisoning there are the symptoms of a violent gastro-enteritis, with colicky pains, especially about the umbilicus, and relieved by pressure; abdominal walls hard; cramps in the legs; convulsions.

Treatment.—Rid the stomach of the poison; use dilute sulphuric acid or a non-poisonous sulphate as a chemical antidote; demulcents with opium for the pain.

Chronic Poisoning.—Constipation with colicky pains centring around the navel; abdominal walls retracted and hard; pulse apt to be hard and corded; headaches; paralysis of extensor muscles of fore-arm (bilateral wrist-drop); a blue line on the gums, due to a deposit of sulphide of lead.

Treatment.—A thorough course of potassium iodide; treat the constipation; avoid further trouble by cautioning lead-workers to thoroughly clean their hands, and by the use of sulphuric acid internally. The colic may require opium.

Meat Poisoning.—*Vide* Ptomaïne Poisoning.

Mercury (*Corrosive Sublimate, Bichloride of Mercury, Mercuric Chloride, or the Perchloride*).—Used in aqueous solution as bed-bug poison, as an insecticide, to preserve specimens, as an antiseptic surgical dressing.

Symptoms.—In concentrated form it is corrosive, hence mouth and lips are swollen and white; a metallic taste; œsophagus, stomach, and abdomen are the seat of intense pain; there is vomiting and purging of mucus, bloody material; scanty, albuminous urine; collapse.

Treatment.—Stomach-pump or emetics; white of egg as chemical antidote; demulcents; stimulants.

Mineral Acids.

Hydrochloric Acid (*Muriatic Acid, Spirit of Salt*).

Symptoms.—Similar to those mentioned under sulphuric acid, though the acid is not as powerful and leaves no distinctive stain. It may be recognized by its odor and by the white fumes formed when the gaseous acid comes into contact with ammonia. Medicinally, and in the arts, hydrochloric acid is used in aqueous solution, the commercial variety tinted yellow from a trace of iron.

Treatment.—Same as for sulphuric acid.

Nitric Acid (*Aqua fortis*).—A colorless, moderately heavy liquid of peculiar and characteristic odor, staining organic tissues yellow.

Symptoms.—The same as those mentioned under sulphuric acid, though the characteristic yellow stain may be found on the lips.

Treatment.—As for sulphuric acid.

Sulphuric Acid (*Vitriol, Oil of Vitriol*).—A very heavy, colorless, odorless liquid, having a very acid taste and mixing with water with the production of great heat. Used largely in the arts. Turns organic matter black.

Symptoms.—Burning pain from mouth to stomach; lips and

mouth white, the vomited matter stained bloody and black; dysphagia; unconsciousness; collapse.

Treatment.—Immediate neutralization, or at least dilution, of the poison; soap and water, chalk, magnesia, lime-water, bicarbonate of sodium, or, in their absence, water in large quantity; demulcents and opium as a sedative. N. B.—Sulphuric acid may kill by œdema of the glottis, or by the secondary effects resulting from œsophageal stricture and destruction of the gastric mucous membrane.

Morphine Poisoning.—See Opium Poisoning.

Mushroom Poisoning.—Harmless varieties may prove poisonous to some individuals. Agaricus muscarius is the most poisonous variety, containing the active principle muscarine. The fungus is bright red with yellow spots. As a rule highly colored fungi, with an astringent, styptic taste, a pungent odor, should be avoided; they frequent especially dark and shady places.

Symptoms.—Excitement, violent colic, vomiting, and diarrhœa; breathing stertorous, surface cold; pulse slow; death from cardiac failure.

Treatment.—Evacuate stomach and bowel; heat; stimulation; atropine as physiological antidote.

Nicotine.—The liquid, volatile alkaloid of tobacco. An acrid, oily liquid, of amber color, smelling of tobacco. A very deadly and quickly-acting poison.

Symptoms.—Nausea, vomiting, faintness, great weakness; pulse rapid and feeble; mental confusion; sight dimmed; skin cold and clammy.

Treatment.—Rid the stomach of the poison; administer tannic acid; strychnine; heat; stimulants; place patient in the recumbent posture.

Nitro-Benzol (*Nitro-Benzene, Oil of Mirbane, Artificial Oil of Bitter Almonds*).—Used in the preparation of aniline dyes and in the arts on account of its flavor (soaps, etc.). Recognized by its highly characteristic odor.

Symptoms.—Usually not evident for an hour or two. Headache, weariness, nausea; the mind gradually becomes confused; there is great anxiety and cyanosis appears, the latter becoming extreme, until the whole body is blue. Stertorous breathing, coma, death by asphyxia or failure of heart.

Treatment.—As the poison is slowly soluble, wash out the stomach, even if a long time has elapsed since its administration; stimulants (whiskey, not until after evacuation of stomach, because it renders the poison more soluble); artificial respiration; interrupted current.

Opium Poisoning (Acute).—Taken accidentally or with suicidal intent in the shape of morphine or laudanum.

Symptoms.—Drowsiness, stupor, deep breathing, if due to laudanum, smell of it in the breath, contracted pupil, slow, full pulse. For a time patient may be aroused by shouting into ear.

Treatment.—Evacuate stomach by stomach-pump or tube, wash out thoroughly ; administer strong coffee and flagellation and electricity to keep patient awake ; if respiration fails, atropine and strychnine hypodermically ; also electricity to phrenic nerve ; artificial respiration.

Oxalic Acid.—Mistaken for Epsom salt ; taken with suicidal intent. Occurs in small, prismatic, colorless, odorless crystals, with a very sour taste. Volatile without charring at a red heat.

Symptoms.—Those of a violent, rapidly fatal gastro-enteritis, associated with cramps in the legs ; the mouth may be white. Collapse comes on quickly. Convulsions occur occasionally.

Treatment.—Carbonate of calcium in any form ; chalk, whitening, or marble dust ; lime, lime-water. In an emergency, white-wash from a wall. Follow by a purgative of castor oil.

Phosphorus.—Employed in the form of a paste as a rat and roach poison ; matches are sometimes sucked for suicidal purposes. The paste is recognized by its peculiar garlicky odor, and by the luminous fumes it emits in the dark.

Symptoms.—Usually do not appear until after the lapse of a few hours. At first the ordinary signs of irritant poisoning appear, such as pain and vomiting ; the ejected material being luminous in the dark. A garlicky taste in the mouth, and the odor of phosphorus may be perceptible on the breath. Later on there is severe abdominal pain, also pain in the region of the liver ; this organ may be enlarged. The symptoms subside, but from the third to the fifth day more serious ones develop ; jaundice, accompanied by pain and vomiting ; discharges of blood from the bowel, with extravasations below the skin. The urine may be suppressed or scanty, albuminous, and bile-stained ; bowels constipated or loose, the stools clay-colored and sometimes phosphorescent. Death after grave nervous symptoms, such as headache, delirium, convulsions, stupor, and coma, and may occur very suddenly. Convalescence is much protracted if patient recovers.

Treatment.—Emetic of five grains (0.33 gm.) of sulphate of copper, which acts also as a chemical antidote. Permanganate of potassium, in dilute solution. French oil of turpentine acts also as an oxidizing agent, but cannot be obtained in this country. Otherwise the treatment is symptomatic.

Potassium Nitrate (*Nitre, Saltpetre*) in large dose produces severe abdominal pain, vomiting and purging of blood-stained material, partial paralysis, convulsions, and collapse.

Treatment.—Stomach-tube or emetics ; demulcents ; stimulants ; heat.

Ptomaïne Poisoning.—Ptomaïnes are alkaloidal bodies, the products of the decay of animal tissues. These are probably the cause of the poisonous action of tainted meat and fish, cream-puffs, ice-cream, *blanc mange*, and cheese.

Poisonous Fish.—Sickness and vomiting, great depression, irritation

of the eyes, severe nettle-rash. Shell-fish are especially apt to produce a rash in susceptible people.

Poisonous Meat contains ptomaïnes, those produced when stale meat is just beginning to decay being more virulent than others which replace the first when decomposition is well under way.

Ices, Ice-Cream.—The cheap varieties are colored with aniline dyes, which may not be free from arsenic. Again, the highly poisonous alkaloid, tyrotoxicon, may have formed in the milk used, and to this principle most cases of poisoning of this kind are traced. Symptoms vary—dryness of throat, vomiting, and purging, are those most commonly observed.

The treatment must be symptomatic.

SULPHURETTED HYDROGEN has the characteristic odor of rotten eggs. Forms the bulk of the gas emanating from sewers and cesspools, some ammonium sulphide and nitrogen occurring with it. If dilute, the symptoms are nausea, diarrhœa, depression, and headaches. In more concentrated form it produces unconsciousness, frequent respiration, rapid pulse; dysphagia; dilated pupils, insensible to light; tonic convulsions; a temperature as high as 104° F. (40° C.) during the convulsions. In a state approaching purity, sewer gas kills almost instantly; in moderate amount it frequently causes death in twenty-four hours, all efforts to restore consciousness proving useless.

Treatment.—Fresh air; artificial respiration for many hours; ammonia by inhalation; stimulation.

STRYCHNINE.—An alkaloid occurring in nux vomica and ignatia. Appears in the form of white, prismatic, odorless crystals, which have an intensely bitter taste. Used as a vermin killer.

Symptoms.—Ordinarily these appear quickly. A sense of suffocation, great difficulty in breathing, from paralysis of the muscles of respiration; great anxiety, though the mind is perfectly clear; twitching of the muscles, finally amounting to tetanic convulsions, with intervals in which the patient is exhausted and bathed in perspiration; opisthotonus; death from asphyxia, or, between the attacks, from exhaustion.

In tetanus there is usually the history of a wound; symptoms develop much more gradually; the muscles of the jaw are early involved, and trismus is much more marked than spasm of the respiratory muscles and the convulsions are tonic.

Treatment.—Emetics or stomach-tube at once,—after the spasms have once set in, the introduction of a tube would excite them; tannic acid; nitrite of amyl; chloroform or ether, by inhalation; artificial respiration, if possible.

ZINC.—The chloride, in solution, is used as a disinfectant (Burnett's Disinfecting Fluid). A very heavy, corrosive, colorless liquid, of astringent taste. It partakes of the nature of a corrosive poison.

Symptoms.—Burning sensation in throat and stomach, perhaps signs of local corrosion. Nausea, vomiting, purging, dyspnœa, convulsions, coma, death.

Treatment.—Carbonate of potassium or sodium, in water; milk; eggs; tannic acid; opium as a sedative.

MINIMUM DOSE WHICH HAS CAUSED DEATH, AND MAXIMUM DOSE FOLLOWED BY RECOVERY.

NAME OF POISON.	MINIMUM DOSE WHICH HAS CAUSED DEATH.	MAXIMUM DOSE WITH RECOVERY.	USUAL FATAL DOSE.	FATAL PERIOD.
Aconite, . . .	From merely tasting Flemming's tincture to f℥j (3.7 c. c.) Flemming's tincture.	f℥iij (11.1 c. c.) Flemming's tincture.		Usually 3–4 hours.
Alcohol, . . .	½ pint gin (236.56 c. c.), two bottles of port (= 11 oz.) (29.57 c. c. alcohol). In a boy of 7 years, f℥iv (120 c. c.) brandy.	1 quart gin (946.24 c. c.); 1 quart whiskey (946.24 c. c.); 2 bottles port; 1½ pints (236.56 c. c.) mixed gin and brandy.		½ hour to several days.
Ammonia, . .	f℥ij (7.4 c.c.) aqua ammoniæ fortior.	f℥j (29.57 c. c.) aqua ammoniæ fortior.	f℥ss (15 c.c.) aqua ammoniæ fortior.	A few hours, months, even years.
Antimony, . . (Tartar Emetic),	2 grains (0.132 gm.). ¾ grain (0.0495 gm.) in a child.	℥ss (15.84 gm.).	℥j (4 gm.).	An hour up to several days.
Arsenic, . . .	Less than 1½ grains (0.099 gm.).	℥j to ℥ij (3.96 to 7.92 gm.). Recovery probable after 1 grain.	1½ to 2½ grains (0.099 to 0.165 gm.).	Death usually within 24 hours.
Atropine, . . .	2 grains (0.132 gm.).	1 grain, 1½ grains (0.066 to 0.099 gm.). Child 2 years old, 1 grain (0.066 gm.); child 4 years old, 1½ grains (0.099 gm.).	½ to ¾ grain (0.033 to 0.0495 gm.).	Death usually within 24 hours.
Belladonna, .	f℥j (3.7 c. c.) liniment.	f℥ss (15 c. c.) liniment; ℥iij (11.88 gm.) extract.		Death usually within 24 hours.
Carbolic Acid,	f℥j (3.7 c. c.).	f℥j (3.7 c. c.).	f℥ss (14.785 c. c.) of the pure, f℥j (29.57 c.c.) of the impure acid.	Death usually within an hour.
Chloroform, .	15 drops (0.9 c. c.) inhaled; 30 drops (1.8 c. c.) inhaled; f℥j (0.3 c. c.) internally.	f℥ij (60 c.c.) taken internally.		
Chloral, . . .	30 grains (1.98 gm.).	180 grains (11.88 gm.); 460 grains (30.36 gm.).		
Copper Subacetate (Verdigris),	℥ss, ℥j (16 to 32 gm.).			4–12 hours.
Copper Sulphate, . . .	Child 16 months old, from merely sucking crystals; ℥j (32 gm.) in an adult.	℥j (32 gm.) in an adult.		4–12 hours.
Digitalis, . . .	Uncertain.	f℥ij (60 c. c.) tincture; ℥j (4 gm.) powdered digitalis.		About 24 hours.
Hydrochloric Acid, ...	f℥ss (15 c. c.) in the adult; f℥j (3.7 c. c.) in a child.		f℥ss (15 c.c.) for an adult; f℥j (3.7 c.c.) for a child.	From a few hours to many weeks.

MINIMUM DOSE WHICH HAS CAUSED DEATH, AND MAXIMUM DOSE FOLLOWED BY RECOVERY.—(*Continued.*)

NAME OF POISON.	MINIMUM DOSE WHICH HAS CAUSED DEATH.	MAXIMUM DOSE WITH RECOVERY.	USUAL FATAL DOSE.	FATAL PERIOD.
Hydrocyanic Acid,	45 minims (2.7 c. c.) diluted acid (2 per cent.). Insensibility from merely smelling the strong acid.	f℥j (3.7 c. c.) Scheele's acid (4 per cent.).	45 minims to f℥j (2.7 to 3.7 c. c.).	10–15 minutes.
(Cherry Laurel Water),	f℥ij (60 c. c.).			
(Oil Bitter Almonds) (natural unpurified),	(contains about 10 per cent. HCN).		10 to 30 drops (0.6 to 1.80 c. c.).	
(Potassium Cyanide),	5 grains (0.33 gm.).		3 to 5 grains (0.198 to 0.33 gm.).	
Iodine,	20 grains (1.32 gm.).			
Iodoform,		℥iv (16 gm.).		
Lead, (Acetate), (Goulard's Solution),		℥j (32 gm.).		Few hours to several days.
(White Lead),		f℥xij (360 c. c.). ℥j (32 gm.).		
Mercury Bichloride,	3 grains (0.198 gm.).	℥j (32 gm.).	3 to 5 grains (0.198 to 0.33 gm.). 50 per cent. of cases fatal.	From 10 hours to 11 days.
Morphine,	¾ grain (0.0495 gm.); 1 grain (0.066 gm.).	75 grains (4.95 gm.); 20 grains (1.32 gm.).	2 to 6 grains (0.132 to 0.396 gm.).	7–12 hours.
Opium,	f℥ij (7.4 c. c.) laudanum; 2½ grains (0.165 gm.) extract; ℥j (3.96 gm.) laudanum; 2 to 3 drops (0.12 to 0.18 c. c.) laudanum in a very young child.	f℥ij (60 c. c.) laudanum; f℥iv to f℥v (120 to 150 c. c.) laudanum.	4 to 5 grains (0.264 to 0.33 gm.).	7–12 hours.
Nitrobenzol (Oil of Mirbane),	From merely tasting it. 8 to 9 drops (0.40 to 0.54 c. c.).	f℥ss (1.85 c. c.).		4–5 hours.
Oxalic Acid,	℥ij (0.132 gm.); ℥j (3.96 gm.) in a boy of 16.	℥ss (16 gm.).	℥ss (16 gm.).	Within one hour.
Phosphorus,	Less than 1 grain (0.066 gm.). Child died from sucking 2 matches. Another from swallowing the tops of 8 matches.	After sucking 300 matches.	A little less than 1 grain.	1–5 days.
Potassium Hydroxide, (Caustic Potash).	40 grains (2.64 gm.).		℥ss (16 gm.).	
Saltpetre,	℥j (32 gm.).	℥ij (64 gm.).	℥j to ℥iss (32 to 48 gm.).	A few hours.
Strychnine,	1/16 grain (0.0041 gm.) in a child of 2½ years; ½ grain (0.033 gm.) in an adult.	10, 12, 40, grains (0.66 to 0.792 to 2.64 gm.).	½ to 1 grain (0.033 to 0.066 gm.).	5 minutes to several hours. In adults during or after the 4th or 5th paroxysm; children 1–3d convulsion.

APPENDIX.

TABLES FOR REDUCING THE METRIC SYSTEM INTO THE ENGLISH.

(*Troy Weight.*)

Grains to Grams.		Grains to Grams.		Grains to Grams.	
$\frac{1}{200}$	= 0.00033	$\frac{1}{105}$	= 0.000628	$\frac{1}{12}$	= 0.0055
$\frac{1}{195}$	= 0.00034	$\frac{1}{100}$	= 0.00066	$\frac{1}{10}$	= 0.0066
$\frac{1}{190}$	= 0.00035	$\frac{1}{95}$	= 0.00694	$\frac{1}{8}$	= 0.0082
$\frac{1}{185}$	= 0.000357	$\frac{1}{90}$	= 0.0073	$\frac{1}{7}$	= 0.0094
$\frac{1}{180}$	= 0.00036	$\frac{1}{85}$	= 0.0077	$\frac{1}{6}$	= 0.011
$\frac{1}{175}$	= 0.000377	$\frac{1}{80}$	= 0.0082	$\frac{1}{5}$	= 0.0132
$\frac{1}{170}$	= 0.000388	$\frac{1}{75}$	= 0.0085	$\frac{1}{4}$	= 0.0165
$\frac{1}{165}$	= 0.0004	$\frac{1}{70}$	= 0.0094	$\frac{1}{3}$	= 0.022
$\frac{1}{160}$	= 0.000413	$\frac{1}{65}$	= 0.001	$\frac{1}{2}$	= 0.033
$\frac{1}{155}$	= 0.000425	$\frac{1}{60}$	= 0.0011	1	= 0.066
$\frac{1}{150}$	= 0.00044	$\frac{1}{55}$	= 0.0012	2	= 0.132
$\frac{1}{145}$	= 0.000455	$\frac{1}{50}$	= 0.00132	3	= 0.198
$\frac{1}{140}$	= 0.00048	$\frac{1}{45}$	= 0.00146	4	= 0.264
$\frac{1}{135}$	= 0.00049	$\frac{1}{40}$	= 0.00165	5	= 0.33
$\frac{1}{130}$	= 0.0005	$\frac{1}{35}$	= 0.00188	6	= 0.396
$\frac{1}{125}$	= 0.000528	$\frac{1}{30}$	= 0.0022	7	= 0.462
$\frac{1}{120}$	= 0.00055	$\frac{1}{25}$	= 0.00264	8	= 0.528
$\frac{1}{115}$	= 0.000574	$\frac{1}{20}$	= 0.0033	9	= 0.594
$\frac{1}{110}$	= 0.0006	$\frac{1}{15}$	= 0.0044	10	= 0.66

Grams to Grains.	
1	= 15.43
2	= 30.86
3	= 46.29
4	= 61.72
5	= 77.15
6	= 92.58
7	= 108.01
8	= 123.44
9	= 138.87
10	= 154.3

Grains to Milligrams.	
1	= 64.8
2	= 120.6
3	= 194.4
4	= 259.2
5	= 324
6	= 388.8
7	= 453.6
8	= 518.4
9	= 583.2
10	= 648
1 drachm or 60	= 3.89 gm.
1 ounce or 480	= 31.1 gm.*

FLUID MEASURES.

1 teaspoonful distilled water	=	1 fluid drachm	=	3.7 c. c.
1 dessertspoonful distilled water	=	2 fluid drachms	=	7.4 c. c.
1 tablespoonful distilled water	=	4 fluid drachms	=	14.8 c. c.
1 wineglassful distilled water	=	2 fluid ounces	=	59.14 c. c.
1 fluid ounce distilled water			=	29.57 c. c. (*circa*, 30 c.c.*)
16 fluid ounces distilled water	=	1 pint	=	473.11 c. c. (*circa*, 480 c.c.)

*A fluid ounce of water which measures 30 c. c. does not weigh 31.1 gm., because an ounce of water really weighs but 455.7 grains Troy, and not 480 grains.

TABLES FOR REDUCING THE METRIC SYSTEM INTO THE ENGLISH.

FLUID MEASURES.—(*Continued.*)

MINIMS TO CUBIC CENTIMETERS.			CUBIC CENTIMETERS TO MINIMS.			FLUID DRACHMS TO CUBIC CENTIMETERS.			CUBIC CENTIMETERS TO FLUID DRACHMS.		
1	=	0.06	1	=	16.2	1	=	3.7	1	=	0.27
2	=	0.12	2	=	32.4	2	=	7.4	2	=	0.54
3	=	0.18	3	=	48.6	3	=	11.1	3	=	0.81
4	=	0.24	4	=	64.8	4	=	14.8	4	=	1.08
5	=	0.31	5	=	81	5	=	18.5	5	=	1.35
10	=	0.62	6	=	97.2	6	=	22.2	6	=	1.62
15	=	0.92	7	=	113.4	7	=	25.9	7	=	1.89
$16\frac{1}{4}$	=	1	8	=	129.6	8	=	29.6	8	=	2.16
20	=	1.23	9	=	145.8	9	=	33.3	9	=	2.43
30	=	1.85				10	=	37	10	=	2.7
40	=	2.46									

FLUID OUNCES TO CUBIC CENTIMETERS.			LITERS TO FLUID OUNCES.			LITERS TO PINTS.			PINTS TO LITERS.		
1	=	29.57	1	=	33.8	1	=	2.1	1	=	0.473
2	=	59.14	2	=	67.6	2	=	4.2	2	=	0.946
3	=	88.71	3	=	101.4	3	=	6.3	3	=	1.419
4	=	118.28	4	=	135.2	4	=	8.4	4	=	1.892
5	=	147.75	5	=	169	5	=	10.5	5	=	2.365
6	=	177.42	6	=	202.8	6	=	12.6	6	=	2.838
7	=	206.99	7	=	236.6	7	=	14.7	7	=	3.311
8	=	236.56	8	=	270.4	8	=	16.8	8	=	3.784
9	=	266.13	9	=	304.2	9	=	18.9	9	=	4.257
10	=	295.7	10	=	338	10	=	21	10	=	4.73
12	=	354.84									
16	=	473.12									

LINEAR MEASURES.

CENTIMETERS TO INCHES.			INCHES TO CENTIMETERS.			INCHES TO MILLIMETERS.			MILLIMETERS TO INCHES.		
1	=	0.3937	1	=	2.54	1	=	25.4	1	=	0.03937
2	=	0.7974	2	=	5.08	2	=	50.8	2	=	0.07874
3	=	1.1817	3	=	7.62	3	=	76.2	3	=	0.11811
4	=	1.5784	4	=	10.16	4	=	101.6	4	=	0.15784
5	=	1.9685	5	=	12.7	5	=	127	5	=	0.19685
6	=	2.3622	6	=	15.2	6	=	152.4	6	=	0.23622
7	=	2.7559	7	=	17.78	7	=	177.8	7	=	0.27559
8	=	3.1496	8	=	20.32	8	=	193.2	8	=	0.31496
9	=	3.5433	9	=	22.86	9	=	228.6	9	=	0.35433
10	=	3.9370	10	=	25.4	10	=	254	10	=	0.3937

FEET TO METERS.			METERS TO FEET.		
1	=	0.3048	1	=	3.28
2	=	0.6096	2	=	6.56
3	=	0.9144	3	=	9.84
4	=	1.2192	4	=	13.12
5	=	1.524	5	=	16.4
6	=	1.8288	6	=	19.68
7	=	2.1336	7	=	22.96
8	=	2.4348	8	=	26.24
9	=	2.7432	9	=	29.52
10	=	3.048	10	=	32.8

TO CONVERT DEGREES OF FAHRENHEIT'S THERMOMETER TO CENTIGRADE, AND VICE VERSA.

Centigrade to Fahrenheit.		
1	=	1.8
2	=	3.6
3	=	5.4
4	=	7.2
5	=	9
6	=	10.8
7	=	12.6
8	=	14.4
9	=	16.2
10	=	18

To use this table, convert the given number of degrees Centigrade into degrees Fahrenheit, and add 32°.

Fahrenheit to Centigrade.		
1	=	0.555
2	=	1.11
3	=	1.665
4	=	2.22
5	=	2.775
6	=	3.33
7	=	3.885
8	=	4.44
9	=	4.95
10	=	5.55

To use this table, subtract 32° from the given number of degrees Fahrenheit and convert the remainder into degrees Centigrade.

INDEX.

A.

Abasia-astasia, 1083
Abscess, mediastinal, 505; of heart, 557; of liver, 356; perinephric, 675; post-pharyngeal, 224; of spleen, 375
Absorption, to determine rate of, 242
Acanthocephali, 1155
Acidity of gastric contents, estimation of, 236
Acromegaly, 1089
Actinomycosis, 196
Active congestion of kidney, 629
Acute chorea, 1032; delirium, 1028; phthisis, 470; rheumatism, 715; bulbar palsy, 893
Addison's disease, 712
Æstivo-autumnal fever, 67
Agraphia, motor, 914
Ainhum, 1093
Albumin-digestion, products of, 239; examination of products of, 239
Albumin, tests for, 627
Albuminuria, general remarks on, 625; extra renal, 625; physiological or functional, 627
Alcoholism, acute, 1109; chronic, 1110
Alalia, 914
Amauroses, 926
Amblyopia, 934
Amimia, 915
Amyotrophic lateral sclerosis, 894
Anæmia, progressive pernicious, 588; of brain, 985; lymphatic, 604; primary or essential, 584; secondary, 609; splenic, 607; symptomatic, 609
Anæmias, the, 583
Anæsthesia as a symptom of nervous disease, 811
Anarthria, 909
Aneurism, of heart, 558; varieties of, 570; of descending aorta, 577; of abdominal aorta, 577; of the innominate, 577; intracranial, 1001; pulmonary, 578; of the subclavian, 578; transverse part of aorta, 576
Angina Ludovici, 215
Angina pectoris, 565
Aniscoria, 939
Anorexia nervosa, 259
Anthrax, 166; bacillus of, 167; in animals, 166; period of incubation, 167
Aortic insufficiency or incompetency, 528; stenosis and regurgitation, 533; stenosis, 532
Aphasia, 909, 913; motor, 914
Aphemia, 914
Apoplexy, 987
Appendicitis, 293; complications of, 304; definition of, 293; history of, 293; morbid anatomy of catarrhal, 296; of interstitial, 297; of ulcerative, 297; symptoms of, 299; obliterans, 296; perforation in, 297; ulcerative, 297; recurring and relapsing, 304; chronic, 304
Arm jerks, 803
Arsenical poisoning, acute, 1124; chronic, 1125
Arterio-capillary fibrosis, 567
Arterial sclerosis, 567
Arthritis, deformans, 729; gonorrhœal, 727
Arthropathies, 875
Arthropoda, 1155
Ascarides, 1146
Ascaris lumbricoïdes, 1146
Ascites, 385
Aspiration-pneumonia, 448
Associated movements, 800
Asthma, bronchial, 419; cardiac, 507
Ataxia, hereditary, 879
Ataxia, progressive locomotor, 869
Atheroma of blood-vessels, 567
Athetosis, 800, 1067
Auditory hyperæsthesia, 954

B.

Ballismus, 1032
Basedow's disease, 614
Bed-bug, 1158
Beef tapeworm, 1138
Bell's, mania, 1028; palsy, 944
Beri-beri, 828
Bilateral spastic hemiplegia, 1006
Bile ducts, affections of, 342
Bilious headache, 1054
Birth palsies, 1006
Bladder, catarrh of, 700; diseases of, 700; hemorrhoidal veins in, 710; morbid growths of, 711; muscular spasm of, 708; neuroses of, 707; stone in, 706
Bladder and rectum, mechanism of action, 800
Blood and blood-making organs, diseases of the, 583
Blood-vessels, diseases of, 567
Body louse, 1157
Bothriocephalus latus, 1140

Bowel, carcinoma of, 326; deranged sensibility of, 323; embolic ulcer of, 892; hemorrhagic infarct of, 291; intussusception of, 309; invagination of, 309; motor derangements of, 322; nervous affections of, 322; obstruction of, 308; by fecal matter, 311; by foreign bodies, 310; by morbid growths, 311; by stricture, 311; secretion neuroses of, 325; strangulation of, 308; syphilitic ulcer of, 292; tubercular ulcer, 291; twists and knots in, 310; ulceration of, 291
Brachial plexus, lesions of, 972
Brachycardia, 560
Brain, abscess of, 1022; affections of blood-vessels of, 984; anæmia of, 985; diseases of, 902; general and functional diseases of, 1028; hyperæmia of, 984; inflammation of, 1022; œdema of, 986; sclerosis of, 1009; syphilis of, 1097; tumors of, 1015
Breathing, alterations in, in nervous disease, 814
Bronchial asthma, 419; tubes, diseases of, 410
Bronchiectasis, 417
Bronchitis, acute, 410; capillary, 448; chronic, 412; fibrinous, 423; plastic, 423
Bronchocele, 612
Broncho-pneumonia, 448; tubercular, 461
Broncho-pneumonic phthisis, 470
Brown atrophy of the heart, 555
Bubonic plague, 172; bacillus of, 172; incubation period of, 172
Bubo, parotid, 214
Buhl's disease, 791
Bulbar palsy, acute, 893; progressive, 890

C.

Cachexia, malarial, 70; strumipriva, 621
Caisson disease, 853
Cancrum oris, 211
Capillary bronchitis, 448
Cardiac asthma, 507; muscle, degeneration of, 553; albuminoid degeneration of, 553; amyloid degeneration of, 554; calcareous, 555
Catarrh, acute bronchial, 410; chronic bronchial, 412; nasal, chronic, 391; of the bladder, 700
Catarrhal pneumonia, 448
Cauda equina, lesions of, 886
Caudate nucleus, 920
Cavities in lung, 459
Central ganglia, 920
Centrum ovale, 920
Cerebellum, 922
Cerebral hemorrhage, 987; hyperæmia, 984; localization, 902; palsies of children, 1003; softening, 996; localization, summary of facts bearing on, 923; vessels, summary of effects of plugging of, 998
Cerebritis, 1022
Cerebro-spinal fever or meningitis, 134; period of incubation, 136; abortive form, 137; chronic form, 138; complications and sequelæ, 139; intermittent form, 138; malignant form, 137; mild form, 137; morbid anatomy of, 135; ordinary form, 136
Cestodes, visceral, 1144
Cheyne-Stokes breathing, 637
Chiasm, optic, and tract, lesions of, 930
Chicken-pox, 125; incubation of, 125
Children, reflex convulsions of, 1052
Chill, the congestive, 68
Chloralism, 1117
Chlorosis, 584
Choked disc, 927
Cholangitis, chronic, 339; suppurative, 340
Cholera, 72; bacillus of, 84; collapse in, 75; diarrhœa in, 75; epidemics of, 72; infantum, 285; morbus, 288; nostras, 76, 288; postmortem test for bacillus, 85; protective inoculations against, 79; prophylaxis in, 78; special directions to nurses in, 80
Chorea, acute, 1032; chronic hereditary, 1041; chronic progressive, 1041; hysterical, 1042; major, 1042; minor, 1032; pre- and post-hemiplegic, 1043
Choreic movements, 800
Choreiform affections, 1038
Chronic endocarditis, 525; progressive chorea, 1041; rheumatism, 725
Chyluria, 693
Cimex lectularius, 1158
Circumflex nerve, lesions of, 972
Cirrhosis, Glissonian, 371; of the lung, 440, 446
Coated tongue, 205
Cocainism, 1118
Coccydinia, 1060
Cœliac affection, the, in children, 288
Colitis, mucous, 280
Colon, dilatation of, 322
Congenital absence of kidney, 687
Congestion of brain, 984; of kidney, 629
Congestive chill, 68
Constipation, 318, 323
Constitutional diseases, 715
Conus medullaris, lesions of, 886
Convulsion, epileptiform, 799
Convulsive tic, 950
Coprolalia, 1039
Cord, spinal, diseases of membranes of, 842
Coronary arteries, sclerosis of, 555
Corpora quadrigemina, 921
Corpulence, 776
Corpus striatum, 920
Cortex, functional assignments of, 903; motor areas of, 903
Cortical areas covering speech, 909; of unknown function, 919; epilepsy, 1047
Coryza, 390
Coup de soleil, 1131
Crab louse, 1157
Cranial nerves, diseases of, 924
Cretinism, 623; congenital, 622; sporadic and endemic, 623
Crises, tabetic, 874
Croup, spasmodic, 399; catarrhal, 399; false, 399
Croupous pneumonia, 431

Crura cerebri, 921
Cyanotic induration of kidney, 629
Cycloplegia, 938
Cysticercus cellulosæ, 1144
Cystitis, 700

D.

Deglutition-pneumonia, 448
Delayed conduction of sensation, 810
Delirium, 813; acute, 1028; cordis, 562; tremens, 1112
Delusions, 813
Dementia paralytica, 1011
Demodex folliculorum, 1156
Dengue, 141
Dentition, derangements due to, 206
Diabetes insipidus, 771; mellitus, 750; acetone in, 760; beta-oxybutyric acid in, 760; blood in, 757; coma in, 757; diacetic acid in, 760; dietetic treatment of, 764; hygienic treatment of, 768; medicinal treatment of, 769; neuritis in, 756; pathology, 751; pathogenesis, 751; treatment of, 764; test for diacetic acid in, 760; test for beta-oxybutyric acid in, 760; for glycogen in, 761; test for inosit in, 760; urine in, 757
Diarrhœa, acute dyspeptic, 283; chronic, 280; of children, 283; nervous, 323
Dilatation, bronchial, 417; of colon, 322
Diphtheria, 104; bacillus of, 104; complications and sequelæ, 109; contagiousness of, 105; croup in, 107; in animals, 105; latent form, 109; laryngeal form, 107; nasal form, 108; nephritis in, 109; neuritis in, 109; pharyngeal form, 107; prophylaxis against, 115; serum therapy in, 113
Diphtheroid sore throat, 108
Diplegia, 797; facialis, 892
Diptera, 1158
Disseminated nodular sclerosis, 1009
Divers' paralysis, 853
Double vision in disease of motor nerves of the eye, 940
Dracontiasis, 1153
Dysentery, 147; abscess of liver in, 150; acute catarrhal, 147; amœbic or tropical, 149; chronic catarrhal, 153; complications, 150; complications and sequelæ of diphtheritic dysentery, 152; diphtheritic dysentery, 151
Dyspepsia, nervous, 252

E.

Echinococcus disease, 1145
Echolalia, 1039
Eighth nerve, lesions of, 951
Electrical excitation of motion, 805
Eleventh nerve, lesions of, 965
Embolic pneumonia, 453
Embolism of cerebral vessels, 996
Emphysema of the lungs, 425; interlobular, 428; pseudo-hypertrophic, 425; vesicular, 425
Encephalitis, suppurative, 1022
Endocarditis, 514; acute, mild, or simple form of, 515; cardiac type, 520; cerebral type, 520; chronic, 521; chronica deformans, 567; infectious, 517; mycotic, 517; severe or malignant form, 517; septic type, 520; typhoid type, 520; ulcerative, 517
Endocardium, diseases of, 514
Enteralgia, 323
Enteritis, acute catarrhal, 276; chronic catarrhal, 280; croupous, 290; diphtheritic, 290; follicular, 287; mucous, 280; phlegmonous, 290; pseudo-membranous, 290; simple acute catarrhal, 276
Entero-colitis, acute, 287
Ephemeral fever, 198
Epidemic cerebro-spinal meningitis, 134; hæmoglobinuria of infants, 790
Epilepsy, 1044; hysterical, 1074
Erb's form of juvenile hereditary atrophy, 1105
Erroneous projection, 940
Erysipelas, 142; abscess in, 144; complications and sequelæ, 145; facial, 144
Eustrongylus gigas, 1154
Exophthalmos, 616
External popliteal nerve, lesions of, 976
Eyeball, lesions of motor nerves of, 936

F.

Facial hemiatrophy, 1089; nerve, lesions of, 944; infranuclear paralysis of, 946; nuclear paralysis, 945; paralysis of, 944; spasm, 950
Falling fits, 1044
Farcy, 169; acute and chronic, 170
Fatty infiltration of heart, 554; metamorphosis of heart, 553
Febricula, 198
Fehling's test solution for sugar, 762
Fever, æstivo-autumnal, 67; break-bone, 141; cerebro-spinal, 134; ephemeral, 198; intermittent, 63; malarial, 55; Malta, 202; miliary, 203; mountain, 46; pernicious malarial, 68; relapsing, 51; remittent, 67; simple continued, 199; scarlet, 95; typhoid, 17; typhus, 47; yellow, 86
Fibrillary contractions, 799
Fifth nerve, lesions of, 942; paralysis of sensory portion, 943; paralysis of motor portion of, 943
Filaria Bancrofti, 1151; diurna, 1152; dracunculus, 1153; hominis oris, 1154; immitis, 1154; loa, 1154; labialis, 1154; lymphatica, 1154; oculi humani vel lentis, 1154
Filariasis, 1151
Flea, common, 1158
Flint's murmur, 527
Floating kidney, 688
Flukes, 1136; liver, 1136; blood, 1137; bronchial, 1137
Foot and mouth disease, 171; bacillus of, 171; period of incubation of, 171
Fourth nerve, lesions of, 939

G.

Gall-bladder, cancer of, 343
Gallop-rhythm, 562
Gall-stone, acute impacted, 336; chronic impacted, 339
Gangrene of the lung, 440
Gastralgia, 254
Gastrectasia, 273
Gastric contents, chemical examination of, 234
Gastritis, acute catarrhal, 243; chronic catarrhal, 245; diphtheritic, 252; mycotic, 252; phlegmonous or suppurative, 250; traumatic or toxic, 251
General paresis, 1011
Giant urticaria, 1086
Gigantorhynchus, 1155
Girdle pains, 874
Glanders, 169; acute, 170; chronic, 170; bacillus of, 169; period of incubation of, 170
Globulin, test for, 628
Glossitis, 212; desiccans, 213
Glosso-pharyngeal nerve, lesions of, 957
Glossy skin, 1091
Glottis, œdema of, 405
Goitre, exophthalmic, 614; simple, 612
Gonorrhœal arthritis, 727; rheumatism, 727
Gout, 734; irregular or atypical, 741; chronic, 741; thread-test for uric acid in, 736, foot note; urine in, 740; pharyngitis in, 740; retrocedent, 740; metastatic, 740
Grain poisoning, 1128
Graves's disease, 614
Grippe, 130
Guinea-worm disease, 1153

H.

Habit-chorea, 1038; habit-spasm, 1038
Hæmatothorax, 497
Hæmaturia, idiopathic, 690
Hæmoglobinuria, 71, 691; paroxysmal, 692; toxic, 691
Hæmopericardium, 513
Hæmophilia, 792
Hallucinations, 813
Hammond's disease, 1067
Harvest bug, 1156
Hay fever, 393
Headache, bilious, 1054; paroxysmal, 1054; sick, 1054
Head louse, 1157
Hearing, modifications of, in nervous disease, 814
Heart, abscess of, 557; aneurism of, 558; atrophy of, 552; chronic valvular defects of, 521; congenital defects of, 536; diseases of, 507; dilatation of, 549; disease, relation of to kidney disease, 694; fatty degeneration of, 549; fibroid degeneration of, 555; irritable, 550; nervous palpitation of, 559; neuroses of, 559; rupture of, 558
Heat, exhaustion, 1130; fever, 1131
Hemeralopia, 926
Hemiachromatopia, 932, 934
Hemianopia, 930; heteronymous, 930; lateral, 930
Hemicrania, 1054
Hemiplegia, 797; bilateral spastic, 1006; infantile, 1003
Hemorrhagic infarct of the lung, 453; syphilis, 790
Hepatic fever, 339
Hepatitis, suppurative, 356
Hereditary ataxia, 879
Hodgkin's disease, 604
Huntingdon's chorea, 1041
Hydrocephalus, acquired, 1026; chronic, 1025; congenital, 1025; internal, 1025
Hydrochloric acid, test for, 235
Hydronephrosis, 685
Hydropneumothorax, 497
Hydropericardium, 513
Hydrophobia, bacillus of, 158; period of incubation in, 158; paralytic stage, 160; Pasteur's treatment by attenuated virus, 161; premonitory stage, 159; spasmodic stage, 159
Hydrothorax, 497
Hyperchlorhydria, 256
Hypertrophy of heart, 546
Hypnosis and suggestion, 1074
Hypo-glossal nerve, paralysis of, 969; spasm, 970
Hysteria, 1070; cardio-vascular symptoms in, 1073; convulsive seizures in, 1074; derangements of motion in, 1072; derangements of sensation, 1071; digestive derangements in, 1073; mental symptoms in, 1073; traumatic, 1082; vaso-motor derangements in, 1073; visceral derangements in, 1073
Hysterical epilepsy, 1074
Hysterogenous zones, 1075

I.

Icterus neonatorum, 334
Illusion, 813
Infantile convulsions, 1052; hemiplegia, 1003
Infectious diseases of doubtful nature, 198; processes in relation to diseases of the nervous system, 1093
Influenza, 130; bacillus of, 131; period of incubation in, 131
Infusoria, parasitic, 1136
Inhalation tuberculosis, 456
Insecta, 1157
Insular sclerosis, 1009
Interlobular emphysema, 428
Intermittent fever, 63; period of incubation, 63; quotidian, 56; tertian, 56; double tertian, 56; hæmaturia, 71
Internal capsule, lesions of, 920
Intestines, disease of, 276; obstruction of, 308
Intrathoracic tumors, 503
Iridoplegia, 938
Itch insect, 1155
Ixodes ricinus, 1156

J.

Jaundice, hæmatogenous, 333; hepatogenous, 332; of the new-born, 334; simple catarrhal, 335

K.

Keloid of Addison, 1092
Kidney, amyloid, 664; anomalies of form and position of, 687; cirrhotic, 654; contracted, 654, 655; cysts in, 657; disease, dimness of vision in, 659; diseases of, 629; echinococcus disease of, 686; fatty and contracted, 646; gouty, 654; granular, 654; iodine test for amyloid, 665; lardaceous, 664; large white, 645; movable, 688; relation of diseases of, to heart disease, 694; stone in, 677; tuberculosis of, 676; tumors of, 682; waxy, 664
Knee jerk, 802

L.

Labyrinthine vertigo, 955
Landry's paralysis, 864
Laryngeal nerves, lesions of, 959; muscles, paralysis of, 406
Laryngitis, acute catarrhal, 397; simple chronic catarrhal, 400; syphilitic, 405; tubercular, 403
Laryngoplegia, 407
Larynx, adductor paralysis of, 961; bilateral abductor paralysis of, 960; diseases of, 396; examination of, 396; spasm of, 962; tensor paralysis of, 961; total paralysis of, 960; unilateral abductor paralysis of, 961
Lateral sclerosis, amyotrophic, 894
Lead poisoning, 1119
Leprosy, 193; anæsthetic, 195; bacillus lepræ, 194; contagiousness of, 194; macular form, 195; tubercular form, 195
Leprous neuritis, 829
Leptomeningitis, acute, 979; chronic, 983
Leptus autumnalis, 1156
Leucæmia, blood in, 598; lieno-myelogenous, 594, 602; liver in, 595, 596; lymphatic, 594, 602; lymphatic enlargement in, 595; marrow changes in, 595; myelogenic form, 594, 596; spleno-medullary, 602; spleen in, 595; splenic form, 594, 602
Leucocythæmia, 594
Leucoplacia, 213
Linguatulidæ, 1156
Lithæmia, 748
Liver, abscess of, 356; active hyperæmia of, 345; acute yellow atrophy of, 359; altered shape, 330; amyloid, 348; atrophic cirrhosis of, 352; biliary cirrhosis of, 352; carcinoma of, 362; changes in hepatic artery and vein, 346; cirrhosis of, 350; diseases of, 330; diseases of blood-vessels of, 345; dislocation of, 330; fatty, 347; fatty infiltration of, 347; fatty metamorphosis of, 348; floating, 330; hydatid cyst of, 367; hyperæmia of, 343; hypertrophic cirrhosis of, 351, 355; lardaceous, 348; morbid growths of, 361; parasites of, 367, 370; passive hyperæmia of, 343; red atrophy of, 343; sarcoma of, 363; syphilis of, 365
Lobar pneumonia, 431
Localization of cerebral disease, 902
Location, sense of, 809
Lock-jaw, 162
Locomotor ataxia, 869
Long thoracic nerve, lesions of, 972
Lumbar plexus, lesions of, 975
Lung, abscess of, 440; cavities in, 459; cirrhosis of, 440, 446; diseases of, 425; emphysema of, 425; fibroid induration of, 440; gangrene of, 440; hemorrhagic infarct of, 453; metastatic abscess of, 455; tuberculosis of, 456; tumors of, 486, 504
Lymphatic glands, tuberculosis of, 191

M.

Malarial cachexia, 70; fever, 55; æstivo-autumnal form of, 67; algid form of, 69; blood changes in, 63; clinical varieties of, 63; comatose form of, 68; chronic form of, 62; description of the paroxysm of, 63; favoring causes of, 61; geographical distribution of, 61; hæmaturia in, 71; intermittent form, 63; irregular forms of, 71; kidneys in, 62; latent form, 71; liver in, 62; organism of, 58; parasite of, 58; plasmodium of, 56; period of incubation, 63; prophylaxis against, 72; quartan, 56; quotidian, 56; remittent form, 67; seasons favoring, 61; specific germs of, 56; spleen in, 62; telluric conditions, 62; tertian, 56; double tertian, 56; varieties of, 55
Malignant pustule, 167
Malta fever, 202
Mania a potu, 1112
Mastication, spasm of muscles of, 943
Measles, 90; as a cause of tuberculosis, 92; complications and sequelæ of, 92; contagiousness of, 90; eruption of, 91; incubation of, 91; German, 94; period of incubation of German, 94; rubella, 94
Median nerve, lesions of, 974
Mediastinal abscess, 505; disease, 500; lymph adenitis, 506; tumors, 502
Membranes of the brain, diseases of, 977
Menière's disease, 955
Meningo-encephalitis, chronic diffuse, 1011
Metastatic abscess of lung, 455
Migraine, 1054
Miliary fever, 203
Milk sickness, 201
Mind atactilia, 912; blindness, 912; deafness, 912; amusia, 912; anosmia, 912; ageusia, 912
Mitral insufficiency or incompetency, 523; insufficiency and stenosis, 528; stenosis, 525
Morbus maculosus neonatorum, 791
Morphine habit, 1115
Morphœa, 1092
Morvan's disease, 883
Motor areas of the cortex, 903; nerves of the eye-ball, lesion of, 936; function of the stomach, to test, 242
Mountain fever, 46; sickness, 47
Mucous colitis, 280; enteritis, 280; patches, 214
Multiple sclerosis of brain and cord, 1009

Mumps, 129; secondary, 130
Muscle jerk, 803; pseudo-hypertrophy of, 1104
Muscular atrophy, facio-scapulo-humeral type, 1106; juvenile hereditary, 1105; idiopathic, 1104; progressive, 896; peroneal, type of, 1106; progressive neural, type, 1106; sense, 811; system, diseases of, 1103
Musculo-spiral nerve, lesions of, 973
Myalgia, 723
Mydriasis, 938
Myelitis, acute and chronic, 854; compression, 883; dorsal, 859; lumbar, 859; of the anterior horns, 861
Myiasis, 1159
Myocarditis, 555; acute suppurative, 557
Myocardium, diseases of, 545
Myositis, acute and chronic, 1103; infectious, 1103; progressive ossifying, 1103; rheumatic, 723, 1103
Myotonia congenita, 1107
Myxœdema, 619; pure, 620; associated with cretinism, 620; congenital, 620; acquired, 620; operative, 621

N.

Naming centre, 911
Nephritis, acute catarrhal or parenchymatous, 631; chronic catarrhal, 644; chronic diffuse, 644; chronic parenchymatous, 644; fever in the acute form, 636; glomerular changes in acute, 634; in chronic, 634; hemorrhagic, 647; hypertrophy of left heart in, 638, 658; interstitial changes in acute, 634; interstitial form, 654; suppurative interstitial, 669; tubular changes in acute, 633; uræmia, 636, 643; urine in, 635, 648, 658
Nephro-lithiasis, 677
Nervous deafness, 952; disease, investigation of a case of, 797; exhaustion, 1078; system, diseases of, 794; general symptomatology of diseases of, 797; histology of, 794; relation of infectious processes to diseases of, 1093; syphilis of, 1097; topical diagnosis or localization of disease in, 796; vomiting, 259
Neuralgia, 1057; brachial, 1059; cervico-brachial, 1059; cervico-occipital, 1059; dorso-intercostal, 1059; epileptiform, 1058; infra-orbital, 1058; lumbo-abdominal, 1059; ocular, 1058; of rectum, 325; of the feet, 1060; of the spinal column, 1060; phrenic, 1059; reflex, 1060; sacral, 1060; tender points in, 1057; trifacial, 1058; visceral, 1060
Neurasthenia, 1078
Neuritis, 815; brachial, 818; endemic, 828; leprous, 829; localized, 815; malarial, 828; multiple, 821
Neuromata, 830
Neuroses, traumatic, 1082
New-born, hemorrhagic diseases of, 790; spastic rigidity of, 1003
Ninth nerve, lesions of, 957
Nose, diseases of, 389
Nyctolopia, 926
Nystagmus, 937

O.

Obstruction of bowel, 308
Occupation neuroses, 1063
Œdema, angeioneurotic, 1086; of brain, 986
Œsophagitis, acute, 225; chronic catarrhal, 226
Œsophagus, cancer of, 227; dilatation of, 228; diseases of, 225; exploration of, 225; spasm of, 226; stricture of, 227
Olfactory nerves, lesions of, 924
Ophthalmoplegia, 940
Optic atrophy, 929; nerve and tract, lesions of, 925; neuritis, 927; tract and centres, lesions of, 932
Organic acids of stomach, determination of, 237
Osteomalacia, 783
Oxyuris vermicularis, 1147

P.

Pachymeningitis, cerebral, 977; cervical hyperplastic, 842; external, 842, 977; hemorrhagic, 843, 977; internal, 842, 977; pseudo-membranous, 977; purulent, 977; spinal, 842
Painters colic, 1119
Palsies, cerebral, of children, 1003
Pancreas, cancer of, 374; cysts of, 374; diseases of, 372
Pancreatitis, acute, 372; chronic, 374; gangrenous, 373; hemorrhagic, 372; suppurative, 373
Paradoxical contractions, 805
Paragraphia, 915
Paralysis agitans, 1029; of laryngeal muscles, 406; periodical, 1084
Paranephritis, 675
Paraphasia, 915
Paraplegia, 797
Parasites, animal, 1135
Parenchymatous, or albuminoid degeneration, 553; nephritis, acute, 631; chronic, 644
Paretic dementia, 1011
Parotid bubo, 214
Parotitis, acute, 214; chronic, 215; epidemic, 129; secondary, 130
Pediculus capitis, 1157; pubis, 1157; vestimenti, 1157
Peliosis rheumatica, 789
Pentastomes, 1156
Pentastomum, constrictum, 1156; tænioides, 1156
Pericarditis, 508
Pericardium, diseases of, 508
Perihepatitis, 370
Perinephric abscess, 675
Peripheral nerves, affections of, 815
Peritoneum, cancer of, 384; diseases of, 377; tuberculosis of, 383

Peritonitis, acute, 377; adhesive, 380; circumscribed, 380; chronic, 382; primary, 377; secondary, 377
Pernicious malarial fever, 68; algid type, 69
Pertussis, 126
Petit mal, 1047
Pharyngitis, acute, 222; chronic, 223; phlegmonous, 224
Pharynx, diseases of, 216; circulatory derangement of, 221; hypertrophy of adenoid tissue of, 219; spasm of, 959; ulceration of, 223
Phrenic nerve, lesions of, 971
Phthisis, acute, 470; broncho-pneumonic 470; chronic ulcerative, 458; fibroid, 471; pneumonic, 470
Plague, bubonic, 172; bacillus of, 172; period of incubation, 172
Platyhelminthes, or flat-worms, 1136
Pleura, diseases of, 488; morbid growths of, 499
Pleurisy, acute, 488; chronic, 496; exudative, 496; plastic, 496; suppurative, 496; varieties of, 492
Pleurodynia, 1059
Plumbism, 1119
Pneumogastric nerve, lesions of, 958; lesions of cardiac branches of, 963; of œsophageal branches, 963; of pharyngeal branches, 958
Pneumonia, abscess of lung in, 440; blood in, 439; broncho-, 448; catarrhal, 448; complications of, 440; chronic interstitial, 446; croupous, 431; diplococcus of, 432; endocarditis in, 441; embolic, non-septic, 453; embolic septic, 455; fibroid induration or cirrhosis in, 440; gangrene in, 440; herpes in, 439; infectious nature of, 432; jaundice in, 439; lobar, 431; lobular, 448; meningitis in, 441; physical signs of, 436; stage of congestion, 434; stage of grey hepatization, 434; stage of red hepatization, 434; stage of yellow hepatization, 434; special symptoms of, 438; symptoms of, 435; termination, 439; treatment, 442; by ice applications, 444; treatment by serum, 446; tubercular phthisis in, 440; typhoid, 439; typhoid fever in, 442; urine in, 439
Pneumonic phthisis, 470
Pneumopericardium, 514
Pneumothorax, 497
Podagra, 734
Poisons, summary of symptoms following overdoses of, alphabetically arranged, 1160
Poliomyelitis, acute, in adults, 864; in children, 861; subacute and chronic, 864
Pollen catarrh, 393
Polyneuritis, 821
Popliteal nerve, external, lesions of, 976; internal, lesions of, 977
Pork tapeworm, 1138
Posterior spinal sclerosis, 869
Post-pharyngeal abscess, 224
Pressure, variations in sense of, 81
Presystolic murmurs, 527
Progressive bulbar palsy, 890; muscular atrophy, 896; paralysis of insane, 1011
Protozoa, diseases caused by, 1135
Protracted simple continued fever, 199
Pseudo-angina, 566
Pseudo-hypertrophic emphysema, 425; muscular paralysis, 1104
Pseudoleucæmia, 604; infantum, 596, 603; splenic, 607.
Psorospermiasis, 1135
Psychical hysteria, 1048
Ptomaïne poisoning, 1125
Ptosis, 937
Ptyalism, 214
Pulex penetrans, 1159
Pulmonary concretions, 460; incompetency or regurgitation, 536; stenosis, 535; tuberculosis, 456; treatment of, 472. See, also, tuberculosis.
Purpura, 785; arthritic, 789; hemorrhagic, 789; Henoch's, 789; scorbutic, 786; symptomatic, 785
Pyæmia, 155; abscesses in, 156; arterial, 520; gonorrhœa as a cause, 157
Pyelo-nephritis, 669
Pyo-pneumothorax, 497

Q.

Quinsy, 216

R.

Rabies, 158
Radial paralysis, 973
Railway spine, 1082; brain, 1082
Raynaud's disease, 1086
Reaction of degeneration, 806; partial, 899; significance of, 807
Rectum, cancer of, 328; mechanism of control of, 800; neuralgia of, 325
Reflexes, 801; ankle, 802; cutaneous, 801; deep-seated, 802; patellar, 802; tendon, 802; segments of cord presiding over, 804; their significance, 804
Relapsing fever, 51; crisis in, 53; complications of, 53; contagiousness of, 52; epidemic occurrence of, 51; incubation of, 52; inoculation in, 51; spirillum of, 52
Remittent fever, 67
Renal disease, relation of, to heart disease, 694; infarct the result of heart disease, 699
Rennet, action of, 241
Respiration and deglutition muscles, affections of, 959, 963, 964
Respiratory system, diseases of, 389
Retina, affections of, 925; functional disturbances of, 926; hemorrhage into, 925
Retinitis, 925
Rhachitis, 779
Rheumatic fever, 715
Rheumatism, 715; acute, 715; chronic, 725; gonorrhœal, 727; inflammatory, 715; muscular, 723; subacute, 720
Rheumatoid arthritis, 729
Rhinitis, 390; acute, 390; chronic, 391
Rhyncota, 1157
Rhythmical contractions, 799
Rickets, 779
Rose cold, 393

S.

Sacral plexus, lesions of, 975
Salivary glands, secondary, inflammation of, 211
Sand flea, 1150
Sarcoptes, or acarus scabiei, 1155
Scarlet fever, 95; acute nephritis in, 100; adenitis in, 100; anginose form, 99; arthritis in, 101; ataxic form, 99; complications and sequelæ in, 100; contagiousness of, 96; desquamation in, 99; eruption in, 97; hemorrhagic form, 99; incubation of, 97; invasion, 97; malignant, 99; nervous affections in, 101; otitis in, 101; prophylaxis against nephritis in, 103; simple form, 97; thoracic complications, 101; tongue in, 97; urine in, 99
Sciatica, 810
Sciatic nerve, lesions of, 975
Scleroderma, 1001
Sclérose en plaques, 1009
Sclerosis of brain, 1009
Scotoma, 935
Scribner's palsy, 1063
Scurvy, 786; infantile, 788
Secondary deviation of vision, 940
Sensory paths to the brain, 907
Septicæmia, 154; cryptogenetic, 199; and pyæmia, 154
Serratus palsy, 972
Seventh nerve, lesions of, 944
Shaking palsy, 1029
Sick headache, 1054
Simple continued fever, 199; tic, 1038
Sixth nerve, lesions of, 939
Small-pox, 116; complications, 119; confluent form, 119; contagiousness of, 116; discrete form, 119; eruption in, 118; hemorrhagic form, 119; incubation of, 117; initial rashes in, 117; prophylaxis against, 120
Small sciatic nerve, lesions of, 976
Spasm, constant or coördinate, 800; of muscles of mastication, 943; tonic and clonic, 799, 800
Spastic paraplegia in children, 1008; spinal paralysis, 866
Speech areas in cortex of brain, 909; derangements of irritative origin, 918; to test derangements of, 918
Spina bifida, 887
Spinal accessory nerve, lesions of, 965; cord, abscess of, 856; anæmia of, 851; anatomy of, 831; compression of, 883; congestion of, 851; embolism and thrombosis in, 851; grey degeneration of, 870; hemorrhage into substance of, 851; localization of function in, 835; pressure paralysis of, 883; secondary degeneration of, 849; secondary degeneration after traumatism, 849; secondary degeneration, clinical significance of, 850; sclerosis of, 869; softening of, 856; syphilis of, 1097; topical diagnosis, 846; tumors of, 888; unilateral lesions of, 847; membranes, hemorrhages into, 845; inflammation of, 842, 843; nerves, diseases of, 971; pachymeningitis, 842; paralysis, acute ascending, 864
Spleen, abscess of, 375; amyloid, 375; atrophy of, 376; diseases of, 375; echinococcus of, 376; hemorrhagic infarct of, 376; leucæmic, 595; neoplasms in, 376; rupture of, 375; wandering, 376
Splenic leucæmia, 602; pseudo-leucæmia, or splenic anæmia, 607
Splenitis, 375
Starch and sugar, digestion of, 241
Stenocardia, 565
Stomach, auscultation of, 233; cancer of, 267; diagnostic technique in diseases of, 230; dilatation of, 273; diseases of, 230; external examination of, 230; internal or chemical examination of, 234; palpation of, 231; percussion of, 232
Stomatitis, 207; aphthous, 208; catarrhal, 207; follicular, 208; gangrenous, 211; mercurial, 209; mycotic, 210; syphilitic, 209; ulcerative, 208
Strongyloides intestinalis, 1154
Strongylus duodenalis, 1150
St. Vitus's dance, 1032
Sugar and starch, digestion of, 241
Suggestion and hypnosis, 1074
Sun-stroke, 1131
Suppurative cholangitis, 340; encephalitis, 1022
Suprarenal capsule, diseases of, 712
Suprascapular nerve, lesions of, 973
Sydenham's chorea, 1032
Syphilis, 174; acquired, 175; amyloid degeneration in, 178; bacillus of, 174; congenital, 175; gummata in, 176; hereditary, 175; period of incubation of, 177; of the nervous system, 1097; primary sore of, 180; primary stage, 174; prophylaxis against, 180; secondary stage, 174; tertiary stage, 174; visceral, 178
Syringo-myelia, 881

T.

Tabes dorsalis, 870; mesenterica, 383
Tabetic crises, 874
Tables for conversion of metric into English system, 1173
Tachycardia, 560
Tactile sensibility, to test, 809
Tænia mediocanellata or saginata, 1138; flavopunctata, 1140; solium, 1138
Tapeworms, 1138; intestinal, 1138; visceral, 1144
Temperature, sense of, 810
Tenderness, 809
Tendon reflexes, 801
Tetanus, 162; bacillus of, 163; idiopathic, 163; period of incubation of, 164; intermittent, 1068; neonatorum, 163; toxic products of bacillus of, 163; traumatic, 163
Tetany, 1068
Theories of cardiac hypertrophy in renal disease, 694
Thermic fever, 1131
Third nerve, lesions of, 936
Thomsen's disease, 1107

Thorn-head worms, 1155
Thought, physical basis of, 910
Thread worms, 1146
Thrombosis of cerebral vessels, 996; of cerebral sinuses and veins, 1002
Thrush, 210
Thyroid gland, tumors of, 624; diseases of, 612; enlargement of, 616
Tic, coördinated, 1040; douloureux, 1058; with explosive utterances, 1039
Tinnitus aurium, 954
Tongue, coated, 205; inflammation of, 212; psoriasis of, 213
Tonsillitis, acute, 216; aphthous, 218; chronic, 219; follicular, 217
Tonsils, diseases of, 216
Torticollis, congenital, 966; fixed, 966; spasmodic, 967
Trachea, diseases of, 410
Tracheo-bronchitis, acute, 410
Traumatic neuroses, 1082
Trematodes or flukes, diseases caused by, 1136
Tremors or trembling motions, 799, 1032
Trichina spiralis, 1148
Trichiniasis, 1148
Tricocephalus dispar, 1154
Tricuspid incompetency regurgitation, 534; stenosis, 535
Trifacial nerve, lesions of, 942
Trigeminus, lesions of, 942
Trophic derangements, 812, 1085
Tubercle, calcareous infiltration of, 188; caseation of, 187; fibroid change in, 188; -granulum, 457; histogenesis of, 187; retroactive inflammation caused by, 188; softening of, 187; solitary, 188
Tubercular consumption, 456; physical signs, 462; broncho-pneumonic, 461; meningitis, 468
Tuberculosis, 182; acute miliary or general miliary, 189; afebrile form, 189, 465; bacillus of, 182; conditions influencing infection, 183; etiology, 183; expectoration in, 464; fever in, 465; Flick's studies in, 183; hemorrhage in, 466; histogenesis of, 187; histology of, 186; inhalation-, 456; method of staining bacillus of, 182; mode of invasion and spread of, 185; morbid anatomy of, 186; of bladder, 711; of brain, 468, 1015; of choroid coat, 190; of kidneys, 676; of larynx, 403; of lymph glands, 191; of membranes of brain, 468; of peritoneum, 383; of pleura, 460, 492; prophylaxis against, 482; pulmonary, 456; treatment of, 472; treatment by climate, 472; treatment, hygienic and dietetic, 475; treatment by medicine, 477; pneumo-therapy in, 479; serum therapy in, 479; treatment of special symptoms, 480
Tumors of the brain, 1015
Twelfth nerve, lesions of, 969
Typhoid fever, 17; abortive form, 32; albumin in, 32; anæmia in, 29; antipyretics in, 40; atypical, 31; bacillus of, 18; bed sores in, 42; blood changes in, 29; bone lesions in, 34; Brand bath treatment of, 37; cardiac complications, 26; chills in, 31; complications, 32; consumption in, 41; contagiousness of, 20; delirium in, 28; diabetes in, 51; diarrhœa in, 26; diazo-reaction of urine in, 29; disinfection of stools in, 45; Ehrlich's reaction in, 29; erythema in, 24; expectant symptomatic treatment of, 40; guaiacol in treatment of, 40; hemorrhage in, 26; hemorrhagic form, 32; herpes in, 24; incubation, 25; in children, 21, 36; indications for alcohol, 41; influence of age, 21; influence of seasons, 21; insanity in, 34; jaundice in, 24; meteorism in, 28, 42; mild forms of, 32; milk leg in, 32; mode of conveyance, 20; muscular tremor in, 28; nervous form of, 32; neuritis in, 33; noma in, 32; osseous system in, 34; parotitis in, 33; perforation in, 42; peritonitis in, 42; Peyer's patches in, 21; pneumonia and pleurisy in, 33; predisposing causes of, 20; prodromal symptoms of, 23; prognosis in children, 36; prophylaxis against, 43; pyuria in, 33; relapses in, 34; renal symptoms in, 32; respiratory symptoms in, 26; rose-colored spots in, 25; serum therapy in, 45; sequelæ of, 34; skin rashes in, 24; specific treatment of, 45; splenic enlargement in, 28; sudamina in, 24; suppurative processes in, 33; sweating in, 26; temperature in, 24; tender toes in, 34; tetany in, 34; thrombosis in, 32; tonsillar form, 32; treatment by diet and rest, 36; treatment, expectant symptomatic, 40; treatment of convalescence, 43; treatment of special symptoms of, 41; tubercular phthisis in, 34; tympanitic distention in, 42; typhoid spine in, 34; urine in, 29; walking form of, 23
Typhus fever, 47; contagiousness of, 48; eruption in, 49; incubation of, 48; temperature in, 49; urine in, 49

U.

Ulcer, gastric, 261; duodenal, 261
Ulceration of the bowel, 291
Ulnar nerve, lesions of, 974
Urinary organs, diseases of, 625

V.

Vaccination, operation of, 122; phenomena of, 123
Vaccine disease, 121
Vaccino-syphilis, 124
Vagus nerve, lesions of, 958
Valvular (cardiac) defects, relative frequency of, 537; lesions, associated or combined, 538
Varicella, 125
Varioloid, 119
Vaso-motor and trophic derangements, 1085; phenomena, 812

Vertigo, labyrinthine, 955
Vesicular emphysema, 425
Vision, modifications of, in nervous disease, 814
Volvulus, 310

W.

Weil's disease, 200
Whooping-cough, 126; complications and sequelæ, 127
Winckel's disease, 790
Wool-sorter's disease, 168
Word-deafness, 913
Word-image, 910
Writer's cramp, 1063
Wry neck, 966; fixed, 966; spasmodic, 967

X.

Xerostomia, 214

Y.

Yellow atrophy, 555
Yellow fever, 86; distribution of, 86; incubation, 87; organism of, 86; prophylaxis against, 89

6.G.432.
The practice of medicine : a te1896
Countway Library AJI3321
3 2044 045 158 318

6.G.432.
The practice of medicine : a te1896
Countway Library AJI3321
3 2044 045 158 318